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LOUIS H. BERMAN
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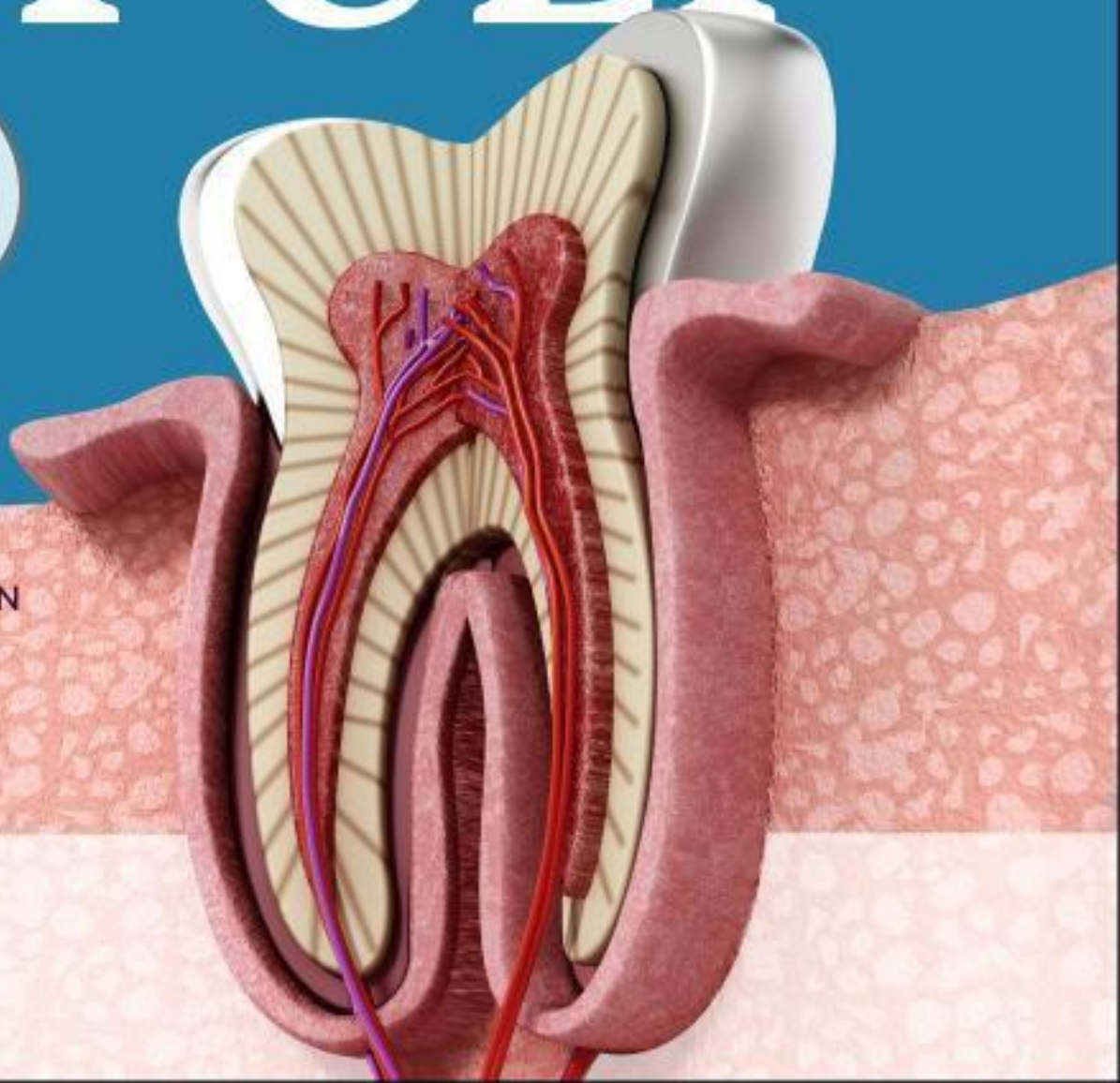
Cohen's PATHWAYS of the PULP

TWELFTH EDITION



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Cohen's Pathways *of the* Pulp

TWELFTH EDITION

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Table of Contents

Telegram: @dental_k

www.konkur.in

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COHEN'S PATHWAYS OF THE PULP, TWELFTH EDITION ISBN: 978-0-323-67303-7

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International Standard Book Number: 978-0-323-67303-7

Content Strategist: Joslyn Dumas
Senior Content Development Manager: Luke Held
Senior Content Development Specialist: Jennifer Wade
Publishing Services Manager: Julie Eddy
Book Production Specialist: Clay S. Broeker
Design Direction: Patrick Ferguson

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Printed in Canada

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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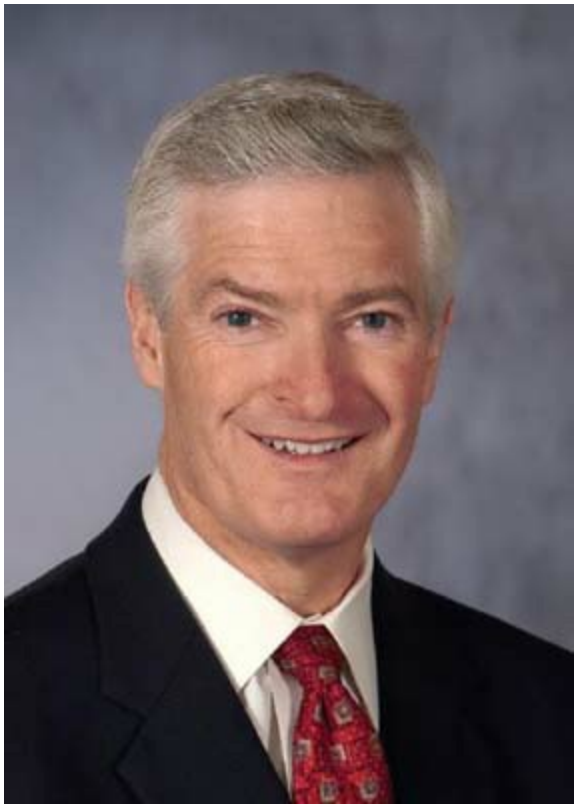
chapters on numerous topics in various endodontic textbooks. He is past president of the Maryland State Association of Endodontics and is a member of the Journal of Endodontics Scientific Advisory Board. A Diplomate of the American Board of Endodontics and Fellow of the American College of Dentistry, Dr. Berman has been in full-time private practice in Annapolis, Maryland since 1983.



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Dr. Hargreaves is Professor and Chair of the Department of Endodontics at the University of Texas Health Science Center at San Antonio. He is a Diplomate of the American Board of Endodontics and maintains a private practice limited to endodontics. He is an active researcher, lecturer, and teacher and serves as the Editor-in-Chief of the *Journal of Endodontics.* He is principal investigator on several nationally funded grants that combine his interests in pain, pharmacology, and regenerative endodontics. He has

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received several awards, including a National Institutes of Health MERIT Award for pain research, the AAE Louis I. Grossman Award for cumulative publication of research studies, and two IADR Distinguished Scientist Awards.

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He has served in leadership roles for various dental organizations, including Chair of the International Federation of Endodontic Associations' Research Committee; as a committee member of the American Association of Endodontists and European Society of Endodontology; and as a scientific

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reviewer for international endodontic and dental journals. He has also served as President of the Southern California Academy of Endodontists, Israel Endodontic Society, and International Association for Dental Research—Israel Division and as Chair of the Israel National Board of Diplomates in Endodontics.

Dr. Rotstein has published more than 150 scientific papers and research abstracts in the dental literature as well as chapters in international endodontic textbooks, including *Pathways of the Pulp, Ingle's Endodontics*, *Endodontics: Principles and Practice*, *Seltzer and Bender's Dental Pulp*, and *Harty's Endodontics in Clinical Practice*. He has lectured extensively in more than 25 countries throughout 5 continents.

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Dedication

Dr. John Ingle

The development of every edition of *Cohen's Pathways of the Pulp*, for all of its editors and contributors, is a journey into both the future and the past of endodontics. What we as clinicians know today and the care that knowledge enables us to provide to our patients are the result of the curiosity, dedication, and commitment of the teachers, researchers, and clinicians who have come before us. Dr. John Ingle, who contributed to the science, practice, and teaching of endodontics for more than 7 decades, is one of those extraordinary pioneers.

Dr. Ingle began his career as an educator at the University of Washington

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in Seattle, where he taught periodontics. While teaching, he became interested in the new field of endodontics and, perhaps drawing on the example of his pioneer great-grandfather, Daniel Boone, he entered a specialty program in endodontics at the University of Michigan to explore this new discipline, earning graduate degrees in endodontics and periodontics.

As an educator and clinician, Dr. Ingle knew the importance of evidence in establishing the efficacy of endodontic treatment as a new specialty. To meet this need, he evaluated the results of endodontic treatment in 3000 patients and presented his findings to the annual session of the American Association of Endodontists in 1953. The *Washington Study* established proof of outcome for endodontic treatment and remains a seminal work in the literature for our field.

New fields offer many new challenges to their pioneers, and Dr. Ingle soon turned his attention to the development of standardization of endodontic instruments. His work resulted in the metric measurements and the 0.2 taper of endodontics files that were established in 1957.

In 1965, Dr. Ingle brought together his experience as both a clinician and an educator in the publication of his foundational textbook *Ingle's Endodontics.* Now in its sixth edition, *Ingle's Endodontics* has provided an essential evidence-based reference to students and clinicians for more than 50 years.

While Dr. Ingle's many accomplishments are known and admired, the attributes of his character are equally revered. Dr. Ingle was modest about his many contributions to endodontics and was unfailingly generous in his support and encouragement of others. The compassion and empathy that motivated his work were experienced by all those he touched throughout his long and exemplary career and life.

It is with profound gratitude and appreciation that we dedicate this twelfth edition of *Cohen's Pathways of the Pulp* to Dr. John Ingle, a scholar, a leader, and a gentleman.

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New to this edition

The radiographic interpretation of odontogenic and nonodontogenic lesions is exactly that: an “interpretation.” This new twelfth edition boasts a completely new chapter entitled Lesions That Mimic Endodontic Pathosis that elucidates and differentiates lesions that may appear as endodontic origin. This is a perfect adjunct to the chapters on Diagnosis and Radiographic Interpretation.

The chapter on Managing Iatrogenic Events has been completely rewritten to include an expansive section on injury to the inferior alveolar nerve.

Damage to the inferior alveolar nerve secondary to endodontic treatment is an avoidable dilemma. There is now specific content elaborating on the avoidance and management of these types of injuries.

Root resorption and root fractures can be some of the most difficult defects to clinically manage. The Root Resorption chapter on these subjects has been completely updated and will prove beneficial to the clinician and academician.

This edition updates all of the previous chapters to reflect the changes in the literature since the last edition.

New chapter organization

Chapters have been reorganized and grouped into three parts: Part I: The Core Science of Endodontics, Part II: Advanced Science Topics, and Part III: Advanced Clinical Topics. The twelve chapters in Part 1 focus on the core clinical concepts for dental students, while the chapters in Parts II and III provide the information that advanced students and endodontic residents and clinicians need to know. In addition, three additional chapters are included in the online version.

The new organization better reflects the chronology of endodontic treatment.

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Digital content

New features included on the companion site include:

- Three chapters found exclusively online:
  - Chapter 26: Bleaching Procedures
  - Chapter 27: Endodontic Records and Legal Responsibilities
  - Chapter 28: Key Principles of Endodontic Practice Management
- Case Studies
- Review Articles
- Review Questions
- Videos

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Introduction

Louis H. Berman, Kenneth M. Hargreaves

The foundation of the specialty of endodontics is a gift from the generations of great endodontists and researchers before us. They guided us with the goals of treatment, the benefits of their advancements, and the frailties of their deficiencies. From volumes of research, we have collectively built a virtual library of knowledge that leads us to the evidence we need for mastering our clinical procedures and benefiting our patients. As we look into our future, we should be directed toward developing the necessary tools for maximizing our outcomes with consistency, longevity, and, above all, patient well-being.

Over the past several decades, we have gone from arsenic to sodium hypochlorite, from bird droppings to gutta-percha, from hand files to motor-driven files, from culturing to one-visit appointments, from 2D to 3D radiography, and from pulp removal to pulpal regeneration. Yet still, the clinical and academic controversies are pervasive.

With patients living longer and with the inescapable comparison of endodontics to endosseous implants, the demand for endodontic excellence has greatly increased. Surprisingly, we still base our diagnosis on a presumed and almost subjective pulpal status. Imagine a future in which endodontic diagnosis could be made more objective by noninvasively scanning the pulp tissue. Imagine algorithms built into all digital radiography for interpreting and extrapolating disease processes. CBCT has made a huge impact on endodontic diagnosis, but can we enhance these digital captures with a resolution that would approach microcomputed tomography, and with less radiation? Will these 3D scans guide us not just with diagnostic objectivity,

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but also with direct treatment facilitation to guide us during surgical and nonsurgical treatment? Truly, we are now on the cusp of gaining the knowledge and technology for accomplishing this. As for clinical visualization, will 3D visualization and monitor-based observation change the way we visualize and implement our procedures? Will our procedures still be done with the fine motor skills of the clinician, or with the augmented reality of digital microsurgical devices? In the years to come, will we be able to truly eliminate all of the canal microorganisms, biofilms, and pulpal tissue? Will we be facilitating our canal cleaning with less toxic and more directed irrigants? Once we are finally able to totally clean and disinfect the canals to a microscopic level, will we have an obturation material that finally satisfies ALL the material requirements that Dr. Louis Grossman enumerated at the inception of our specialty? Will this obturating material be newly regenerated vital pulp?

Clearly, our endodontic future lies in out-of-the-box thinking, with the next generation of transformations coming with collaborations not just from within the biological sciences, but also in conjunction with physicists, chemists, engineers, and a multitude of other great innovative minds. The predictability of endodontics must be incontestable, not just with better technology to guide us toward greater success, but also to better elucidate exactly when endodontic treatment cannot be successful. Our future needs to focus on predictability and consistency, which will only be achieved with disruptive technologies, rather than persisting with variations and modifications of our current convictions. As a specialty, we have advanced by leaps and bounds since our inception; but we are still in our infancy with a brilliant future ahead of us. Since 1976, and now with 12 editions, *Cohen's Pathways of the Pulp* has always been about the art and science of endodontics, with an emphasis on evidenced-based direction rather than anecdotal guidance. The dedicated contributing authors have generously given their time to meticulously describe what is considered the state of the art of our specialty. We are hopeful that future editions will guide us toward enhanced endodontic outcomes, with the never-ending pursuit of endodontic excellence.

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List of Tables

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Table 26.1 Overview of Intrinsic and Extrinsic Discolorations

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List of Illustrations

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Fig. 1.8 Swelling of the submental space associated with periradicular disease from the mandibular incisors.
Fig. 1.9 **A,** Extraoral drainage associated with periradicular disease from the mandibular right canine. Note the parulis on the right anterior side of the face. **B,** Initial scar associated with the extraoral drainage incision after the parulis was drained and root canal therapy performed on the canine. **C,** Three-month follow-up shows healing of the incision area. Note the slight inversion of the scar tissue.
Fig. 1.10 **A,** Extraoral sinus tract opening onto the skin in the central chin area. **B,** Radiograph showing large radiolucency associated with the mandibular incisors. **C,** A culture is obtained from the drainage of the extraoral sinus tract. **D,** The healed opening of the extraoral sinus tract 1 month after root canal therapy was completed. Note the slight skin concavity in the area of the healed sinus tract.
Fig. 1.11 Fluctuant swelling in the anterior palate associated with periradicular disease from the palatal root of the maxillary first premolar.
Fig. 1.12 Fluctuant swelling in the posterior palate associated with periradicular disease from the palatal root of the maxillary first molar.
Fig. 1.13 Fluctuant swelling in the mucobuccal fold associated with periradicular disease from the maxillary central incisor.
Fig. 1.14 **A,** To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. **B,** Radiograph of the area shows an old root canal in a maxillary second premolar and a questionable radiolucent area associated with the first premolar, with no clear indication of the etiology of the sinus tract. **C,** After tracing the sinus tract, the gutta-percha is seen to be directed to the source of pathosis, the apex of the maxillary first premolar.
Fig. 1.15 Percussion testing of a tooth, using the back end of a mirror handle.
Fig. 1.16 Mobility testing of a tooth, using the back ends of two mirror handles.
Fig. 1.17 **A,** Carbon dioxide tank with apparatus attached to form solid CO_2 stick/pencil. **B,** CO_2 gas being transformed into a solid stick/pencil. **C,** CO_2 stick/pencil extruded from end of a plastic carrier and ready for use.
Fig. 1.18 **A,** Refrigerant spray container. **B,** A large cotton pellet made of a cotton roll, or a ready-made size #2 (large) cotton pellet, can be used to apply

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the refrigerant spray to the tooth surface. The small #4 cotton pellet does not provide as much surface area as the #2 cotton pellet, and therefore should not be used to deliver the refrigerant to the tooth surface. **C,** A large cotton pellet sprayed with the refrigerant and ready to be applied to the tooth surface.

Fig. 1.19 Irreversible pulpitis associated with the mandibular right second molar. Patient has found that the only way to alleviate the pain is to place a jar filled with ice water against the right side of his face.

Fig. 1.20 **A,** Electric pulp tester with probe. The probe tip will be coated with a conducive medium, such as toothpaste, and placed in contact with the tooth surface. The patient will activate the unit by placing a finger on the metal shaft of the probe. **B,** View of the electric pulp tester control panel; the knob on the front right of the unit controls the rate at which the electric current is delivered to the tooth. The plastic panel on the left front displays the digital numerical reading obtained from the pulp test. The digital scale runs from 0 to 80.

Fig. 1.21 Nellcor OxiMax N-600x pulse oximeter.

Fig. 1.22 To determine which tooth, or tooth part, is sensitive to mastication, having the patient bite on a specially designed bite stick is often helpful.

Fig. 1.23 Sometimes there is no clear indication of why a tooth is symptomatic. This radiograph shows a mandibular second molar with a moderately deep restoration **(A);** the pulp tests nonvital. Without any transillumination, a fracture cannot be detected **(B).** However, by placing a high-intensity light source on the tooth surface, a root fracture can be observed on the buccal surface **(C)** and the distal-lingual surface **(D).**

Fig. 1.24 Radiograph showing what appears to be a mandibular lateral incisor associated with periapical lesion of a nonvital tooth. Although pulp necrosis can be suspected, the tooth tested vital. In this case, the appearance of apical bone loss is secondary to a cementoma.

Fig. 1.25 Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. **A,** When the source of radiation is directly perpendicular to overlapping objects, the image is captured without much separation of the objects. However, when the radiation source is at an angle to offset the overlapping objects, the image is captured with the objects being viewed as separated. **B,** The object that is closest to the film (or sensor) will move the least, with the object closest to the radiation source appearing farthest away.

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Fig. 1.26 Digital radiography has an advantage over conventional film in that the image can be enhanced and colorized—a useful tool for patient education.
Fig. 1.27 Cone-beam volumetric tomography, using the 3D Accuitomo 80.
Fig. 1.28 Cone-beam volumetric tomography has the ability to capture, store, and present radiographic images in various horizontal and vertical planes.
Fig. 1.29 Cone-beam volumetric tomography has the advantage of being able to detect pathosis in the bone or associated with the teeth without the obstruction of anatomic structures. The planes of vision may be axial, sagittal, or coronal.
Fig. 1.30 The radiation source in cone-beam volumetric tomography is conical. The receiving sensor captures the image as "voxels," or three-dimensional pixels of information, allowing digital interpretation.
Fig. 1.31 **A,** This standard two-dimensional radiographic image reveals recurrent caries under the mesial margin of the maxillary first molar. However, the patient localized pain to mastication on the maxillary second molar. **B,** Cone-beam volumetric tomography revealed an apical radiolucency associated with the maxillary second molar. The bone loss was obscured on the two-dimensional radiograph by the maxillary sinus, zygoma, and cortical bone.
Fig. 1.32 **A,** Periapical radiograph showing a large apical radiolucency associated with the mandibular second molar. Apical pathosis should be ruled out. **B,** Cone-beam volumetric tomography revealed salivary indentation of the mandible in the area apical and lingual to the mandibular second molar, consistent with a Stafne defect. **C,** Enlargement of coronal section in the area of the mandibular second molar and the Stafne defect located on the lingual aspect of the mandible.
Fig. 1.33 Poorly fitting intracoronal restorations can place stresses within the tooth that can cause a vertical root fracture. **A,** This radiograph of a mandibular second premolar (with a gold inlay) reveals extensive periapical and periradicular bone loss, especially on the distal aspect. **B,** The tooth pulp tested nonvital, and there was an associated 12-mm-deep, narrow, isolated periodontal pocket on the buccal aspect of the tooth. After the tooth was extracted, the distal aspect was examined. **C,** On magnification (×16) the distal aspect of the root revealed an oblique vertical root fracture. Similarly, the placement of an ill-fitting post may exert intraradicular stresses on a root that can cause a fracture to occur vertically. **D,** This radiograph depicts a

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symmetrical space between the obturation and the canal wall, suggesting a vertical root fracture. **E,** After the tooth is extracted, the root fracture can be easily observed.

Fig. 1.34 Physical trauma from sports-related injuries or seizure-induced trauma, if directed accordingly, may cause a vertical root fracture in a tooth. This fracture occurred in a 7-year-old child secondary to trauma from a grand mal seizure.

Fig. 1.35 Dentinal tubules are filled with fluid that, when stimulated, will cause sensation. Temperature changes, air, and osmotic changes can provoke the odontoblastic process to induce the stimulation of underlying A-delta fibers.

Fig. 2.1 **A,** A well-angulated periapical radiograph of the maxillary right first molar taken during a diagnostic appointment for endodontic evaluation of the maxillary right quadrant. At first glance, there is little radiographic evidence of significant or periradicular change. **B,** Contemporaneous CBCT image of same tooth gives an entirely different perspective; periapical changes are visible on all three roots in all three anatomic planes of section.

Fig. 2.2 **A** and **B,** High-resolution complementary metal oxide semiconductor (CMOS) sensors are available from many manufacturers. Note that Figure In **B,** wireless CMOS sensors transmit images to the chairside workstation by 2.4 GHz radiofrequency.

Fig. 2.3 **A,** Certain regions of interest (ROI) can be highlighted with a preset contrast tool that can be moved around the image. **B,** Preprogramed filters that enhance sharpness and contrast can be selected to optimize the image acquired.

Fig. 2.4 This case demonstrates the difficulty in assessing lesions in the mandibular posterior region when there is a dense cortex. **A,** This well-angulated periapical radiograph shows a normal periapical region associated with the mandibular left second molar, for which the patient has presented for evaluation and possible retreatment. **B,** The CBCT corrected sagittal, coronal, and axial reconstructed multiplanar views, (*left to right*, sagittal, coronal, and axial views) show previous endodontic treatment, with a 6 mm diameter radiolucency with a well-defined, mildly corticated border, centered over a point on the buccal aspect of the root, 2 mm coronal to the apex; these are features consistent with an apical rarefying osteitis. **C,** The ray sum images of the sagittal view, where the image is “thinned” by decreasing the number of

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adjacent voxels using postprocessing software, simulates a curvilinear projection, showing diminishing superimposition (*left to right,* image layer of 10 mm, 5 mm, 2 mm, and 0.076 mm).
Fig. 2.5 i-CAT unit.
Fig. 2.6 **A,** Planmeca ProMax 3D. **B,** J. Morita Veraviewepocs 3D.
Fig. 2.7 **A,** CS 9000 3D and CS 8100 extraoral imaging systems. **B,** Morita Accu-i-Tomo 170.
Fig. 2.8 **A,** *Left,* Drawing represents a *pixel* (picture element), the image capture and display element of any traditional digital image displayed on the computer. Shades of gray or color are displayed in these pixels to represent a 2D image. *Right,* Drawing represents a *voxel* (volume element). Voxels in CBCT are isometric and have the same dimension or length on all sides. They are very small (from 0.076 to 0.6 millimeters) and are the capture elements for cone beam imaging devices. Principles of conventional fan beam and cone beam computed tomography are presented in **B** and **C**, respectively.
Fig. 2.9 Multiplanar and 3D color reconstructed views of the mandibular quadrant taken on a CBCT machine with a volume size of 37 × 50 mm.
Fig. 2.10 Image of entire head (17 × 23 cm) from a large FOV unit.
Fig. 2.11 **A,** Panoramic 2D image exposed with the JMorita Veraviewepocs 3D for evaluation of the mandibular left central incisor. Other radiographic findings also were revealed, including the horizontal bony impaction of the mandibular right third molar and a possible lesion of endodontic origin associated with the endodontically treated maxillary left second molar. Data available from the scan allowed a 3D reconstruction to be made for areas of concern. **B,** Periapical radiograph of the maxillary left second molar on the same patient revealed a periapical area of low attenuation in the region of the periapex of the mesiobuccal root. In this case, also, changes could be evaluated in greater detail with CBCT imaging. **C,** CBCT of the maxillary left second molar revealed detailed periapical and periradicular changes in all three orthogonal planes of the section, specifically illustrating the lesion of endodontic origin associated with the mesiobuccal root. Examination of the width of the mesiobuccal root in both the axial and coronal views (buccolingually) showed that the mesiobuccal root possibly had two canals and that only a single canal was treated during the initial endodontic therapy.
Fig. 2.12 **A,** This reconstructed panoramic image from CBCT data

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approximates the view one would see with a conventional panoramic radiograph. It is somewhat difficult to see the lesion on the maxillary left first molar. **B,** This thin-slice pseudopanoramic image (0.01 mm) shows the lesion without most of the anatomic superimposition. These slices of the maxillary left first molar show the lesion in sagittal **(C)** and coronal **(D)** views, confirming the features seen in the pseudopanoramic view.

Fig. 2.13 CS 9300 3D Extra-Oral Imaging System.

Fig. 2.14 Veraviewepocs 3D R100.

Fig. 2.15 CS 8100-3D.

Fig. 2.16 **A,** Axial slice from data obtained with a 0.4 mm voxel size. **B,** Compare the trabecular pattern and outline of the mental foramina to the same location in an axial slice from data obtained with a 0.16 mm voxel size.

Fig 2.17 Lateral periodontal cyst. A 12-year-old male presented for orthodontic consultation with an elevated lesion in the maxillary right anterior region that was fluctuant under pressure; the patient was referred for endodontic evaluation and radiographic assessment. The pulp vitality was normal for the maxillary anterior dentition. A preoperative CBCT of the anterior maxilla was exposed (**A,** pseudopanoramic reformation; **B,** 3D surface reformation; **C,** cross-sectional reformation of the maxillary right lateral incisor) showing a unilocular/ovoid area of uniform low density that measured approximately 18.0 mm × 15.0 mm. **D,** Axial reformation showing a partially well-defined, noncorticated border mesial to the canine and a poorly defined border between the distal of the right lateral and central incisor. Surrounding tissues showed locally destroyed buccal and palatal cortical plates of the right anterior side of the maxilla with 6 mm of expansion of the buccal and palatal plates in the area between the canine and lateral incisor. Roots of the canine and lateral incisor are displaced mesially and distally, respectively. There was a 3 to 5 mm thickening of the sinus membrane of the overlying floor of the maxillary sinus. Seven months after surgical intervention, the patient was asymptomatic and a follow-up CBCT study was acquired, showing normal healing. **E,** 3D surface. **F,** Cross-sectional. **G,** Axial reformation.

Fig. 2.18 A 62-year-old female was referred for evaluation and possible treatment for persistent dentoalveolar pain (PDAP) in the maxillary left region. The patient could alleviate this condition by placing a moist cotton roll in the adjacent vestibule to prevent the buccal mucosa from contacting

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the alveolus supporting the maxillary teeth in this area. This condition began after a history of local anesthetic administration, persisted for 1 year, and led to three successive new crowns and endodontic treatment on the "offending tooth," in an effort to remedy the condition without improvement. The response to endodontic tests, TMJ, and myofascial evaluations were normal. Application of topical xylocaine resulted in cessation of pain for 15 minutes. **A,** A PA radiograph showed a root-treated, maxillary left first molar with no apparent radiographic lesion. **B,** A limited FOV CBCT of the maxillary left posterior was acquired. The corrected sagittal view showed an approximately 4 mm, well-defined, ovoid, mildly corticated area of low attenuation (radiolucent) centered over the apex of the mesiobuccal root and extending to the junction of the middle and apical third of the mesiobuccal root *(yellow arrow)*. There was mild mucositis *(green arrow)*. **C,** There was a previously untreated mesioaccessory canal *(yellow arrow)* and mild mucositis. A diagnosis of neuropathic pain and a chronic apical periodontitis was made. Daily application of topical ketamine, gabapentin, and clonidine was prescribed. Endodontic revision of the maxillary left first molar was performed 3 months after the patient was stabilized with the topical medications.

Fig. 2.19 This patient was referred for evaluation and possible treatment after emergency pulp extirpation performed by others. **A,** This PA radiograph shows that the location of the physiologic terminus *(yellow arrow)* and radiographic apex *(blue arrow)* did not appear to coincide within the normal range. **B,** Contemporaneous CBCT sagittal reformation shows the anomalous location of the physiologic terminus *(yellow arrow)* and radiographic apex *(blue arrow)*. **C,** Presence of a mesioaccessory canal with isthmus *(blue arrow)* and **(D)** an oval-shaped canal *(yellow arrow)* is apparent.

Fig. 2.20 **A,** Dens en dente of the mandibular left second bicuspid; coronal slice. **B,** Coronal view. **C,** Panoramic reconstruction from CBCT. **D,** Sagittal view.

Fig. 2.21 **A,** Mandibular left second molar referred for endodontic evaluation and possible treatment. This 2D radiograph reveals significant pulp stones and canal calcification developing not only in the coronal aspect of the root canal system, but also extending down the visible distal canal. The apical third of the canal system appears unusual and dilacerated. Cone-beam computed tomography (CBCT) would be beneficial in visualizing the root

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canal anatomy, to create the ideal endodontic access. **B,** Single slice of the CBCT image for the same tooth. Information about the direction of the root canal anatomy is provided in all three planes of the section: axial, coronal, and sagittal. Interestingly, the axial slice shows that the mesial lingual root actually traverses buccally as it approaches its terminal extent. This is valuable information for the clinician before the entire root canal system is cleaned and shaped; it may also establish a higher degree of treatment predictability.

Fig. 2.22 This 38-year-old female patient presented for evaluation and treatment of a symptomatic mandibular right second molar, which had been endodontically treated more than 10 years previously. This tooth was sensitive to percussion and biting forces; periodontal findings were normal. Microscopic examination of the exposed dentin was negative for a vertical fracture. **A,** The PA radiograph showed the previous endodontic treatment; a post present in the distal canal; and an approximately 5 mm diameter, unilocular, well-defined area of uniform low density centered at the periapex of the distal root—features consistent with an apical periodontitis. Contemporaneous **(B)** CBCT sagittal and **(C)** axial reformations revealed a previously untreated distobuccal canal *(yellow arrow)*.

Fig. 2.23 Root fracture in an endodontically obturated maxillary right central incisor. **A,** Axial view with artifacts from a highly attenuating (opaque) obturant. **B,** View without artifacts from obturant. **C,** Oblique parasagittal view. **D,** Paracoronal view.

Fig. 2.24 This 64-year-old male patient presented with sensitivity when biting on the mandibular left second molar. The history included endodontic revision more than 6 months earlier and subsequent transient paresthesia and dysesthesia along the distribution of the inferior alveolar nerve, which persisted for 1 week after the retreatment. Epicritic and protopathic sensibility was normal. The results of periodontal probing, staining and microscopic examination of the exposed root surface at the sulcus were normal. **A,** Initial PA radiograph showed the approximate location of excess radiopaque material, a feature consistent with extruded root canal sealer *(yellow arrow)*. **B,** Contemporaneous CBCT corrected sagittal reformation shows approximate length of extruded material, measuring 3.4 mm *(yellow arrow)*. **C and D,** Corrected cross-sectional reformation shows the foreign material interior to the inferior alveolar canal *(yellow arrow)*, an area of low

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attenuation extending from the apex superiorly to an area near the alveolar crest *(blue arrow)*, and erosion of the lingual endosseous surface. **E,** The same feature was shown in the corrected axial view *(yellow arrow)*, bisected by a dark line extending from the obturant to the surface of the root—features suggestive of a vertical fracture. Examination of the extracted tooth revealed a vertical fracture at the lingual aspect of the distal root.

Fig. 2.25 Unexpected torsional and flexural failure of endodontic instruments can occur during instrumentation. **A,** PA radiograph shows a separated file *(yellow arrow)* located at the midroot of the mandibular left lateral incisor in a patient referred for revision treatment. **B,** To aid development of a retreatment strategy, CBCT was used to localize the instrument *(yellow arrow)* in the lingual canal, with identification of the buccal canal *(green arrow)* to facilitate bypassing the instrument and **(C),** subsequent removal. **D,** Obturation was completed without complication.

Fig. 2.26 **A** to **W,** Anatomic landmarks on CBCT images acquired using CS 9000, CS 9300, and iCAT units.

Fig. 2.27 After a traumatic injury to the maxillary right and left central incisors, crown fractures were noted. The PA radiograph showed an extrusive luxation injury in the maxillary left central incisor **(A).** The alveolar fracture (**B,** *yellow arrow*) and true extent of the displacement (**C,** *yellow arrow*) became evident with CBCT.

Fig. 2.28 **A,** Horizontal root fractures resulting from trauma were evident in this 22-year-old male patient, who was referred with a contemporaneous film–based PA radiograph **(B)** for evaluation and possible treatment 9 months after trauma to his maxillary lateral and central incisors. Since the trauma, the teeth had been stabilized with a ribbon-type splint on the palatal surface; they were of normal color and responded normally to all sensibility tests. There was slight mobility of the traumatized teeth. **C,** The true nature of the root fractures are shown in the 3D segmented reformation and the corrected cross-sectional views of the **(D)** maxillary right lateral and **(E)** central incisors, the **(F)** maxillary left central and **(G)** lateral incisors, and the 3D surface-rendered reformation **(H).** Temporal reevaluation at 7 years showed minimal changes when compared to the initial presentation *(left to right in each group)*. Task-specific exposure parameters allowed each successive CBCT image volume to be exposed with lower mA, resulting in a 20% radiation dose reduction.

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Fig. 2.29 **A,** External cervical resorption was evaluated in this patient after PA radiographic image, and **(B)** visual examination showed pathognomonic signs of this lesion *(yellow arrow)*. A CBCT image was exposed to determine the true extent of resorption and restorability. **C,** The lesion showed a perforative defect at the CEJ on the facial *(yellow arrow)* and **(D)** palatal aspects *(yellow arrow)*. **D,** The presence of an intact layer of mineralized dentin, the "pericanalar resorption-resistant sheet" *(blue arrow)*, is a hallmark of this condition.

Fig. 2.30 The problem of superimposition of unrelated structures onto the features of interest is reduced when tomographic slices are used instead of images in which an entire volume of data is compressed into a planar image. **A,** This PA radiographic image of the maxillary left second molar shows no radiographic indicators of pathosis. A contemporaneously exposed CBCT image (**B,** corrected sagittal view) shows a 4.3 × 1.9 mm, well-defined unilocular, noncorticated area of low attenuation centered over the apex of the mesiobuccal root, consistent with a periradicular periodontitis *(yellow arrow)*. There is a moderate mucositis in the region of the maxillary sinus adjacent to this tooth *(blue arrow)*. Experimental semiautomated segmentation of this image using active contour methods (ITK-SNAP) allowed for measurement of the true volume of the lesion **(C)** and for future temporal comparisons based on volumetric measurements **(D).** This lesion measured 85,112 voxels and 38.1044 cubic mm.

Fig. 2.31 Radiographic interval change is shown by comparing two volumetric datasets exposed at different times. **A,** Initial CBCT corrected sagittal reformation of maxillary right second molar, distobuccal root and **(B)** corrected sagittal reformation of same root at 13-month checkup. **C** and **D,** ITK-SNAP segmented sagittal reformation of same root. Alignment and overlap of the 3D models: tooth-blue, initial lesion is red, lesion at 13-month check-up is yellow, respectively. **E,** Voxel counting, volumetric measurement (mm) and mean intensity of the periapical lesion. **F,** Models were exported to the .stl format and then viewed with MeshLab software. **G,** 3D Mesh Metric Qualitative (color-coded map) and quantitative (point value) assessments of the lesions before (translucent), and after (colored). The color map shows changes with green color in the mesial region (0.28 mm) and changes with dark red color in the distal region (1.30 mm).

Fig. 2.32 Guided access flow chart. **A,** 2D rendering from the CBCT of a

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block of 2 molars. **B,** Volumetric rendering of the teeth. **C,** Lateral view of the teeth with virtual files in position. **D,** Printed guide. **E,** Representative occlusal view of an accessed tooth.

Fig. 2.33 Guided endodontic surgery. **A,** 3D printed guide. **B,** Guide placed in position prior to endodontic surgery. **C,** Surgical access with printed guide in position.

Fig. 3.1 A well-defined radiolucency near the apex of the maxillary first premolar and canine. Although the premolar did not respond to pulp testing, the location of the lesion and clinical presentation were suspicious and a biopsy revealed an odontogenic keratocyst.

Fig. 3.2 **A,** Deep caries in the mandibular second premolar, but the patient was asymptomatic with no history of pain. **B,** Tooth #29 responded normally to both cold and electric pulp testing, but nonsurgical root canal treatment was completed for restorative reasons.

Fig. 3.3 A limited field CBCT is a useful adjunct when the findings on a periapical image are not conclusive. **A,** A subtle radiolucent area is present at the mesial root apex of the mandibular first molar. **B,** Sagittal, **(C)** axial and **(D)** coronal CBCT images reveal a more prominent loss of density at this root apex.

Fig. 3.4 Periapical inflammatory disease presents with a wide spectrum of radiographic changes. **A,** Preoperative periapical image of a mandibular first molar with a necrotic pulp and asymptomatic apical periodontitis. There is a widened PDL space and subtle radiolucency at the mesial root apex. **B,** Nonsurgical root canal treatment was completed. **C,** Evidence of bone healing at 6-month recall. **D,** Preoperative periapical image of a mandibular first molar with a necrotic pulp and asymptomatic apical periodontitis. This lesion is considerably larger than in image **A** and involves the mesial and distal root apices with coronal extension along the distal aspect. **E,** Nonsurgical root canal treatment was completed and a lateral canal is present in the apical half of the root. **F,** Evidence of bone healing at 6-month recall.

Fig. 3.5 Although most odontogenic keratocysts occur as multilocular radiolucencies at the angle of the mandible, they can be located at any position in either jaw. A cropped panoramic radiograph reveals a corticated odontogenic keratocyst in the mandibular right premolar region.

Fig. 3.6 **A,** A cropped panoramic radiograph shows a well-defined unilocular odontogenic keratocyst at the right angle of the mandible and distal root of

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second molar. The lesion was surgically removed and good bone healing was evident at a 3-year recall with no indication of recurrence **(B)**.

Fig. 3.7 **A,** A panoramic radiograph of a 10-year-old female with a mixed dentition. **B,** Serial panoramic images were obtained for orthodontic reasons and a well-defined expansile lesion developed in the mandibular right premolar region over 9 months. This type of radiolucency should not be confused with an inflammatory etiology due to its location around the crown of an unerupted tooth. This odontogenic keratocyst was treated by unroofing the cyst without removal of the entire lesion. **C,** Panoramic image with no evidence of recurrence at 2 years. **D,** Periapical image with no evidence of recurrence at 6 years.

Fig. 3.8 **A,** 3D volumetric rendering of an ameloblastoma between roots of mandibular canine and mandibular lateral incisor with destruction of the buccal cortical plate. **B,** Sagittal CBCT image reveals a low-density lesion with numerous septae displacing the roots of teeth #22 and #23. **C,** Axial CBCT image exhibiting a perforation of the buccal cortical plate and buccal-lingual expansion that is common with ameloblastomas. This lesion would be characterized as a multilocular radiolucency on a periapical image.

Fig. 3.9 **A,** Central giant cell lesion extending from left mandibular premolar region across the midline in a 17-year-old female. This radiolucency cannot be differentiated from other multilocular lesions without a biopsy (see Box 3.1). **B,** The left mandibular premolars and canine had nonsurgical endodontic treatment prior to removal of the central giant cell lesion and the mandibular incisors had nonsurgical endodontic treatment after removal of the lesion, when it was determined intraoperatively the blood supply to these teeth had been compromised. **C,** Eighteen-month recall with evidence of increased bone density, but incomplete healing.

Fig. 3.10 Axial view of CT showing the extent of the central giant cell lesion of the same patient from Fig. 3.9. This plane reveals a multilocular growth pattern with thinning of both the buccal and lingual cortical plates.

Fig. 3.11 **A,** Cropped panoramic image of condensing osteitis exhibiting a widened PDL space with a radiodense rim at the apex of a carious tooth #29. **B,** Sagittal CBCT image of maxillary premolar with previous endodontic treatment. A homogeneous high-density lesion is present with circumferential loss of density at the root apex suggesting an inflammatory etiology.

Fig. 3.12 **A,** Periapical image of #14 demonstrating cervical resorption and

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superimposition of the maxillary sinus over the palatal root apex. There is no way to determine the presence of inflammatory changes in the sinus based on this image. **B,** Sagittal CBCT image obtained because of resorption in first molar. Teeth in the field of view were asymptomatic and responded normally to pulp testing. There is no indication of mucosal thickening in the sinus. **C,** Periapical image of necrotic #3 with well-defined radiolucencies at the apices. **D,** Sagittal CBCT image confirms areas of low density at the root apices and reveals resorption of the maxillary sinus floor with localized mucosal thickening. **E,** Coronal CBCT image illustrates collateral edema into the sinus with a perforation of the sinus floor near the palatal root apex.

Fig. 3.13 **A,** Periapical image of #2. This tooth is nonresponsive to thermal testing and painful to percussion. Radiographically there is a slightly increased density in the maxillary sinus near the palatal root apex, but superimposition of normal anatomical structures limits the diagnostic accuracy of the image. **B,** Sagittal CBCT image reveals a homogeneous dome-shaped elevation into the maxillary sinus.

Fig. 3.14 Axial CBCT image of mandibular arch demonstrates irregular medullary bone destruction and cortical defects from tooth #25 to tooth #31. There is new bone formation along the inferior facial cortex consistent with osteomyelitis and proliferative periostitis.

Fig. 3.15 Idiopathic osteosclerosis. **A,** A cropped panoramic image with a well-defined alteration in the trabecular pattern apical to teeth #20 to 22 in a 15-year-old female. Peripherally there is slight cortication with no evidence of root resorption. **B,** Sagittal CBCT image showing a homogeneous area of hyperdensity near the canine and premolar root apices. The lesion has an irregular outline, but there is no indication of root resorption.

Fig. 3.16 **A,** Periapical image of a compound odontoma in a 35-year-old female. Multiple radiopacities resembling teeth are located interproximal to the maxillary central and lateral incisors. **B**, CBCT image demonstrating multiple high-density toothlike structures in association with a corticated low-density zone resembling dental follicle.

Fig. 3.17 Complex odontoma. Sagittal CBCT image with a high-density globular mass near the crown of a maxillary first molar. The mass exhibits the density of dentin.

Fig. 3.18 Panoramic radiograph demonstrating Paget disease of bone. A multifocal presentation involving both right and left mandibular molar areas.

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The coarse trabecular pattern and mixed radiolucency is common to Paget disease. There is significant root resorption, but the typical hypercementosis is absent.

Fig. 3.19 **A,** A radiodense ridge superimposed over the coronal portion of the roots extending from the implant in the #19 position to the mesial of tooth #30. This represents a prominent lingual exostosis and should not impact endodontic diagnosis but could interfere with visualization of the canals radiographically. This panoramic image also demonstrates well-defined uniform radiopacities inferior to tooth #21 and tooth #27 consistent with idiopathic osteosclerosis. **B,** Axial CBCT image of a maxillary torus characterized by a well-defined high-density area in the midline.

Fig. 3.20 **A,** Multiple periapical radiolucencies suggest the possibility of a noninflammatory etiology. However, teeth #12 and #13 did not respond to cold testing and both teeth were tender to percussion and palpation. The diagnosis for tooth #12 and tooth #13 was pulpal necrosis and symptomatic apical periodontitis. Nonsurgical root canal treatment was completed in both teeth. **B,** A 6-month recall demonstrated bone healing at the root ends of both teeth.

Fig. 3.21 Multiple well-defined radiolucencies in the anterior mandible. The patient reported no history of trauma and was asymptomatic. These teeth had no significant dental history and responded to both cold and electric pulp testing. Based on this clinical presentation, a diagnosis of periapical cemento-osseous dysplasia was made and no treatment was indicated.

Fig. 3.22 **A,** A 55-year-old female presented with pain, a sinus tract facial to #24, and a history of biopsy in the area several years prior. Teeth #23, #24, and #25 did not respond to cold or electric pulp testing. Radiographically the mixed radiolucent and radiopaque lesions were consistent with periapical cemento-osseous dysplasia, but the sinus tract and pulp testing indicated pulpal necrosis in teeth #23, #24, and #25. Pulpal necrosis was likely due to a disruption of the pulpal blood supply at the time of the biopsy. **B,** Nonsurgical root canal treatment was completed and there was no indication of vital pulp tissue in the canals. The previous biopsy report was obtained prior to root canal treatment, and the diagnosis of periapical cemento-osseous dysplasia was confirmed. **C,** At the 6-month recall the sinus tract had healed and the patient was asymptomatic. Because the radiographic lesions were not the result of a bacterial etiology, there was no expectation to see a significant

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change in the bone at the 6-month recall.

Fig. 3.23 Florid cemento-osseous dysplasia. A mixed radiolucent and radiopaque presentation with multiquadrant involvement. These teeth should respond normally to pulp testing unless there is a history of previous endodontic treatment. If a radiolucent area is present near the root apex of a previous root canal, it may be impossible to determine if the bone loss is due to a bacterial etiology or a manifestation of florid cemento-osseous dysplasia.

Fig. 3.24 Hyperparathyroidism. **A,** Sagittal CBCT image with multiple low-density lesions and changes in mandibular bone density. **B,** Axial CBCT image showing a thinning of the cortical plates in association with the low-density lesions.

Fig. 3.25 Langerhans cell histiocytosis. **A,** Reformatted panoramic image with destructive radiolucent area in mandibular molar region demonstrating "floating" teeth in the #17 and #18 area. **B,** Sagittal CBCT image confirming the aggressive nature of the lesion. **C,** Axial CBCT image revealing destruction of both buccal and lingual cortical plates.

Fig. 3.26 **A,** 50-year-old male with a complaint of increasing pain and swelling from the maxillary left canine region over the last year. Examination revealed a palatal swelling and radiographically there was an ill-defined radiolucent lesion near teeth #11 and #12. **B,** A periapical image with a mixed radiolucent and radiopaque lesion is present, but the margins are more well-defined than on the panoramic image. A biopsy was done to rule out a salivary gland neoplasm due to the palatal swelling and destructive nature of the radiolucency. The biopsy revealed both acute and chronic inflammation with sequestrum formation and bacterial colonies consistent with a diagnosis of osteomyelitis.

Fig. 3.27 **A,** Nonsurgical root canal treatment completed in tooth #30 due to pulpal necrosis. **B,** A 1-year recall due to increasing pain in the area. Radiographs revealed an ill-defined radiolucency associated with teeth #28, #29, and #30. These teeth exhibited abnormal mobility and there was significant soft-tissue inflammation, gingival bleeding, and exposed bone. The destructive nature of the bone loss and clinical presentation suggested malignancy, but a biopsy revealed sheets of chronic inflammatory cells and necrotic bone consistent with osteomyelitis. **C,** A panoramic image reveals complete osseous healing at 4-year recall.

Fig. 3.28 Medication-related osteonecrosis of the jaw. A cropped panoramic

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radiograph shows a sclerotic rim of bone over the mesial buccal root of tooth #3 and the root apex of tooth #4. There is a well-defined radiolucent area over the midroot of tooth #4 suggesting localized periodontal disease. Purulent discharge and exposed bone were present from teeth #2 to #5.

Fig. 3.29 A 29-year-old female with a history of numbness and swelling. These teeth responded normally to pulp testing, but there was a firm and tender buccal swelling from tooth #2 to #6. A diffuse radiolucency was present in the area of the buccal swelling with well-defined radiolucencies at the root apices. Teeth #3 and #4 had limited restorative histories and #2 and #5 were unrestored. Given this clinical presentation a noninflammatory etiology was suspected and biopsy revealed a lymphoma.

Fig. 3.30 A periapical image demonstrating multiple areas of radiolucency in a 42-year-old female with a chief complaint of achiness and loose teeth in the maxillary right quadrant. These teeth responded to both cold and electric pulp testing. A CBCT revealed an irregular and ill-defined loss of density that perforated the buccal and palatal cortical plates and the maxillary sinus. There was resorption of several root ends. The overall presentation suggested malignancy, and biopsy revealed metastatic breast cancer.

Fig. 3.31 **A,** Volumetric rendering reveals multiple osseous defects throughout the mandible consistent with multiple myeloma. **B,** Sagittal CBCT image showing a well-defined and destructive low-density lesion. **C,** Axial CBCT image exhibiting destruction of the trabecular bone and marked thinning of both buccal and lingual cortical plates.

Fig. 3.32 **A,** Well-defined radiolucency at the root apex of tooth #8. The previous root canal filling is short of the apex and exhibits numerous voids. A tissue sample obtained during apical surgery revealed a periapical granuloma. **B,** Well-defined radiolucency at the root apex of tooth #10. Apical surgery was completed, and the biopsy confirmed a periapical cyst. There is no way to differentiate periapical cysts from periapical granulomas using conventional imaging.

Fig. 3.33 CBCT images of the periapical granuloma from Fig. 3.32, *A*. It is not possible to differentiate periapical cysts from periapical granulomas using CBCT, and biopsy remains the best method to definitively make this distinction. **A,** Sagittal CBCT image of periapical granuloma. **B,** Axial CBCT image of periapical granuloma. **C,** Coronal CBCT image of periapical granuloma.

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Fig. 3.34 **A,** Periapical image of tooth #30 with a previous root canal and a well-defined radiolucency at the mesial root apex. **B,** Angled periapical image of the same tooth, further demonstrating the extent of the lesion. **C,** Sagittal CBCT demonstrating a loss of density at the mesial root apex of tooth #30. This well-defined lesion was sampled during an apical surgery, and the biopsy showed a periapical cyst.

Fig. 3.35 **A,** Biopsy revealed a periapical cyst at the apex of tooth #7. There is a loss of trabeculation at the apex blending into normal bone resulting in an ill-defined radiolucency. **B,** Well-defined periapical granuloma at the root apex of tooth #27 was confirmed with a biopsy when bone loss and symptoms persisted following nonsurgical root canal treatment. Size and radiographic definition of periapical cysts and periapical granulomas are highly variable.

Fig. 3.36 Lateral radicular cyst. **A,** Preoperative image with gutta-percha tracing a draining sinus tract. Patient has a history of biopsy in the area that was diagnosed as a dentigerous cyst. Tooth #20 responds to both cold and electric pulp testing. Options included root canal treatment, extraction, or a second biopsy. The sinus tract suggests a necrotic pulp and the potential for a false-negative pulp test. Also, the diagnosis of dentigerous cyst is not accurate because the tooth has erupted. Nonsurgical root canal treatment was completed. **B,** Several lateral canals can be seen opposite the radiolucent areas and the sinus tract resolved within 1 week. **C,** Healing of bone is evident at 18-month recall.

Fig. 3.37 Lateral periodontal cyst. **A,** Periapical image of a well-defined radiolucency near the root apex of mandibular premolar and canine. The periapical image suggests a multilocular presentation that would not be consistent with periapical inflammatory disease from a bacterial etiology. **B,** Sagittal CBCT image revealing the loss of density between the canine and premolar teeth with a normal PDL space at the apex of tooth #21.

Fig. 3.38 **A,** Pulp testing indicated irreversible pulpitis symptoms from tooth #20 and a well-defined radiolucent area was identified between teeth #20 and #21. **B,** Nonsurgical root canal treatment was completed to address the thermal symptoms, and referral was made for a biopsy that identified an odontogenic keratocyst. Lateral radicular cysts, unicystic ameloblastomas, odontogenic keratocysts, and lateral periodontal cysts can exhibit identical radiographic appearances in the mandibular canine and premolar areas.

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Fig. 3.39 Focal cemento-osseous dysplasia. **A,** Sagittal CBCT image demonstrating a mixed-density lesion with a low-density periphery between the mandibular second premolar and first molar. A second high-density lesion is present near the distal root apex of the first molar. **B,** Axial CBCT image exhibiting an expanded and thinned cortex from a mixed-density central globular mass with a hypodense periphery.
Fig. 3.40 **A,** Periapical image with a well-defined unilocular radiolucency at the root apex of tooth #8. Asymptomatic and no history of trauma. Tooth #8 responded to both cold and electric pulp testing. A biopsy revealed a nasopalatine duct cyst. **B,** The more classic presentation of nasopalatine duct cyst with a rounded radiolucency in the midline.
Fig. 3.41 Stafne defect. **A,** Sagittal CBCT image showing a well-defined loss of density inferior to the mandibular canal. **B,** Axial CBCT image demonstrating a lingual concavity. **C,** Coronal CBCT image also exhibiting a lingual concavity inferior to the mandibular canal with no involvement of the root ends.
Fig. 3.42 Anterior Stafne defect. **A,** Periapical image exhibiting a well-defined radiolucency near, but not at, the root apex of tooth #23. The lack of restorative history and location of the lesion suggest a noninflammatory etiology and these teeth respond to pulp testing. **B,** Reformatted panoramic image showing a loss of density near the apex of tooth #23. **C,** Coronal CBCT images reveal a concavity in the lingual cortex of bone with no involvement of the root ends.
Fig. 4.1 Pantomogram of a patient who has undergone several endodontic procedures without resolution of her chief complaint.
Fig. 4.2 A graphic depiction of the trigeminal nerve entering the brainstem. The primary afferent neuron synapses with a second-order neuron in the trigeminal nucleus. The second-order neuron carries pain information to the thalamus, from which it is sent to the cerebral cortex for interpretation.
Fig. 4.3 Illustration of pain that is referred from an area innervated by one nerve *(C2)* to an area innervated by a different nerve *(V2)*. Note that this phenomenon occurs secondary to the convergence of different neurons onto the same second-order neuron in the trigeminal nucleus. The sensory cortex perceives two locations of pain. One area is the trapezius region that represents the source of pain. The second area of perceived pain is felt in the temporomandibular joint area, which is only a site of pain, not a source of

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pain. This pain is heterotopic (referred).

Fig. 4.4 Illustration of the laminated pattern of innervation from orofacial structures into the trigeminal nucleus. These laminated patterns commonly reflect the patterns of referred pains felt in the orofacial structures.

Fig. 4.5 Herpes zoster involving the maxillary division of the left trigeminal nerve of the palate of a 45-year-old male. He complained of a deep, diffuse dull ache of his maxillary left quadrant for 1 week before this vesicular outbreak.

Fig. 4.6 Extrusion of the filling material from the distal canal of tooth #30 of a 36-year-old female. Her complaint was of extreme pain after completion of the root canal treatment followed by sharp, burning continuous pain that could be made worse by a light touch of the tooth.

Fig. 4.7 Diagnostic criteria for persistent dentoalveolar pain disorder *(PDAP)*. *CT,* Computed tomography; *IASP,* International Association for the Study of Pain; *MRI,* magnetic resonance imaging.

Fig. 4.8 Example of a form to evaluate odontogenic pain.

Fig. 4.9 Example of a form to evaluate pain history. *VAS,* Verbal analog scale.

Fig. 4.10 **A**, Local anesthetic at the site of pain fails to reduce the pain. **B**, Local anesthetic at the source of pain reduces the pain at the source as well as at the site.

Fig. 4.11 Periapical radiograph showing prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3.

Fig. 5.1 Sample medical consultation letter.

Fig. 5.2 **A,** Periapical view of tooth #29 after endodontic treatment by a general dental clinician. The diagnosis was irreversible pulpitis. **B,** Patient was referred to an endodontist 4 months later to evaluate radiolucencies of teeth #29 and #30. Symptoms indicated irreversible pulpitis of tooth #30, with concurrent lower right lip and chin paresthesia. Past medical history revealed breast cancer in remission. **C,** Nonsurgical endodontics was performed on tooth #30. Immediate referral was made to an oncologist/oral surgeon for biopsy to rule out nonodontogenic origin of symptoms. **D,** Surgical posttreatment radiograph of teeth #29 and #30. The biopsy report confirmed metastatic breast cancer.

Fig. 5.3 Incision and drainage should be performed on this fluctuant swelling *(arrow)* in conjunction with canal instrumentation.

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Fig. 5.4 Two years after endodontic therapy of tooth #8, the patient returned with pain and swelling. A clinician mistakenly began endodontic access on tooth #7, without confirming the apparent radiographic diagnosis by sensibility testing. Tooth #7 was vital, and tooth #8 was successfully retreated after removal of the post.

Fig. 5.5 Many years after endodontic treatment of tooth #19, the patient returned with a chief complaint of pain and an inability to chew with the tooth. Despite the radiographic appearance of excellent endodontic treatment, the tooth was retreated and the patient's pain disappeared. Note the unusual distal root anatomy, which was not apparent during the initial procedure. **A,** Initial radiograph. **B,** Completion of initial endodontic therapy. **C,** Retreatment.

Fig. 5.6 Initial radiograph was misleading and implicated tooth #23 and tooth #24. Pulp testing indicated a vital pulp in tooth #24, and it was not treated. Retreatment of #23 resulted in healing of the periradicular lesion.

Fig. 5.7 Nonsurgical retreatment of tooth #30. An additional root was located and treated. **A,** Note inadequate endodontic treatment and large periapical lesion. **B,** Bitewing radiograph. **C,** Retreatment after post removal. **D,** Eighteen-month recall radiograph indicates periapical healing.

Fig. 5.8 Despite several exacerbations during endodontic retreatment, this case responded rapidly after the completion of therapy. Radiographic evaluation indicates a good periapical response after only 4 months. **A,** Radiograph taken 2 years after initial root canal therapy. **B,** Retreatment of the mesial root. **C,** Radiograph taken 4 months following retreatment.

Fig. 5.9 Four years after endodontic therapy, the patient complained of pain and swelling associated with tooth #6. The initial impression was that apical surgery was indicated. However, further radiographs revealed the true cause of the endodontic failure. The initial endodontic access through the crown or caries damaged the coronal seal and recurrent decay followed.

Fig. 5.10 **A,** Inflamed, edematous interproximal tissue *(arrow)* caused by acute endodontic pathosis. **B,** Soft-tissue healing *(arrow)* 3 days after initiation of endodontic treatment. **C,** Periradicular pathosis. **D,** Completed endodontic therapy. **E,** Periradicular healing at 1-year recall.

Fig. 5.11 A large, bony defect associated with tooth #20 healed after endodontic therapy. The tooth was nonvital, and no significant periodontal probing depth indicated pulpal disease.

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Fig. 5.12 Tooth #30 has a poor prognosis. Periodontal probing reached the apex of the distal root. Extraction is indicated and should be done as soon as possible to prevent further damage to the mesial bone associated with tooth #31. Implant site preservation is another consideration in treatment planning for this case.

Fig. 5.13 Resorptive defects can be successfully treated. Early intervention, before there is perforation of the root, increases the chance of success.

Fig. 5.14 **A,** Invasive cervical resorption of tooth #8 was detected on a periapical radiograph. **B,** A radiograph taken 9 years prior to the current examination. **C,** CBCT provided an accurate view of the location and the extent of the lesion. **D,** During surgery, following root canal therapy, the resorptive defect was excavated. **E,** The surgical site after restoration with Geristore. **F,** A 2-year follow-up CBCT shows that the lesion has not continued to extend.

Fig. 5.15 The presence of curved roots and multiple canals is a complicating factor.

Fig. 6.1 A cold refrigerant may be used to test for pulpal anesthesia before the start of a clinical procedure.

Fig. 6.2 An electric pulp tester also may be used to test for pulpal anesthesia before a clinical procedure is started.

Fig. 6.3 The central core theory. The axons in the mantle bundle supply the molar teeth, and those in the core bundle supply the anterior teeth. The extraneural local anesthetic solution diffuses from the mantle to the core.

Fig. 6.4 Incidence of first mandibular molar anesthesia as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

Fig. 6.5 Incidence of first mandibular molar anesthesia: comparison of 0.5% bupivacaine with 1:200,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 6 hours. The bupivacaine solution showed a longer duration of anesthesia than the lidocaine solution.

Fig. 6.6 Incidence of first mandibular molar anesthesia: comparison of 1.8 and 3.6 mL of 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant

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difference between the two volumes was noted.
Fig. 6.7 Injection site for the mylohyoid nerve block.
Fig. 6.8 Incidence of first mandibular molar anesthesia: comparison of the combination mylohyoid infiltration plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two techniques was noted.
Fig. 6.9 Incidence of anesthesia for intraosseous and infiltration injections. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The intraosseous injection showed a quicker onset and a shorter duration of anesthesia.
Fig. 6.10 The Stabident perforator, a solid 27-gauge wire with a beveled end that is placed in a slow-speed handpiece.
Fig. 6.11 The anesthetic solution is delivered to the cancellous bone through a needle placed into the hole made by the perforator.
Fig. 6.12 The X-Tip anesthetic delivery system consists of an X-Tip *(top)* that separates into two parts: the drill (a special hollow needle) and the guide sleeve component *(bottom).*
Fig. 6.13 The anesthetic solution is injected through the X-Tip guide sleeve.
Fig. 6.14 Septodont Evolution needle used for the Tuttle Numb Now intraosseous injection technique.
Fig. 6.15 Septodont needle sleeve, which allows safe bending of the needle for the Tuttle Numb Now intraosseous injection technique.
Fig. 6.16 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection of 2% lidocaine with 1:100,000 epinephrine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique was significantly better at all postinjection times.
Fig. 6.17 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection with 3% mepivacaine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum

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setting (percentage of 80 readings) across time for 60 minutes. The combination technique proved to be significantly better for approximately 30 minutes.

Fig. 6.18 Mean plasma concentrations of lidocaine for the intraosseous and infiltration injection techniques. No statistical differences were seen between the two techniques at any time period.

Fig. 6.19 Incidence of first maxillary molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The 3% mepivacaine showed a shorter duration of anesthesia than the lidocaine solution.

Fig. 6.20 Incidence of maxillary lateral incisor pulpal anesthesia using an initial infiltration and a repeated infiltration 30 minutes later (both infiltrations used 1.8 mL of 2% lidocaine with 1:100,000 epinephrine). Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings). The repeated infiltration injection significantly prolonged the duration of pulpal anesthesia.

Fig. 6.21 A flexible analgesic strategy.

Fig. 6.22 Comparison of ibuprofen *(IBU)* 600 mg plus acetaminophen *(APAP)* 1000 mg with IBU alone or to placebo treatment in postendodontic pain patients.

Fig. 7.1 **A,** The color of the pulp chamber floor *(Fl)* is always darker than the walls. The orifices are usually located at the junction of the walls and the floor *(FWJ)*. **B,** The same is true for the maxillary first premolar. **C,** Electron photomicrograph of the pulp chamber floor of a mandibular first molar (×20). **D,** Electron photomicrograph of the furcation surface of a mandibular first molar (×30). *D,* Distal canal; *M,* mesial canals; *arrows,* accessory foramina. (**A** and **B**, From Krasner P, Rankow HJ: Anatomy of the pulp chamber floor, *J Endod* 30:5–16, 2004.)

Fig. 7.2 The dental operating microscope (DOM) has vastly improved locating the position of the coronal canal anatomy.

Fig. 7.3 Major anatomic components of the pulp cavity.

Fig. 7.4 **A,** Formation of reparative dentin and reduction of the pulp chamber space. **B,** Calcific narrowing and closing of a root canal as it leaves the pulp chamber.

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Fig. 7.5 **A,** μCT reconstruction image of a maxillary molar showing the location of accessory canal. **B,** Morphological variations of accessory canal.
Fig. 7.6 **A,** Apical delta in maxillary canine and maxillary second premolar. **B,** Morphological variations of apical delta.
Fig. 7.7 Mandibular first molar showing a furcation canal *(FC, arrows)*. *F,* Furcation; *PC,* pulp chamber floor; *RC,* root canal.
Fig. 7.8 Accessory canals occur in three distinct patterns in the mandibular first molars. **A,** In 13% a single furcation canal extends from the pulp chamber to the intraradicular region. **B,** In 23% a lateral canal extends from the coronal third of a major root canal to the furcation region (80% extend from the distal root canal). **C,** About 10% have both lateral and furcation canals.
Fig. 7.9 **A,** Furcation lesion. **B,** Healing occurred after treatment.
Fig. 7.10 Diagrammatic representation of canal configurations. **A,** One canal at apex based on the work of Vertucci. **B,** Two canals at apex based on the work of Vertucci. **C,** Three canals at apex based on the work of Vertucci. **D,** Supplemental canal configurations based on the work of Gulabivala and colleagues on a Burmese population.
Fig. 7.11 Common variant of a C-shaped canal anatomy found in Native Americans and Asian populations.
Fig. 7.12 μCT scans of mandibular premolars reflect a wide variation in canal morphology, moving from coronal to apical sections. The red color indicates a non–C-shaped canal space; green indicates a semilunar canal shape; and yellow indicates a continuous C-shaped configuration.
Fig. 7.13 An oval orifice must be explored with apically curved small instruments. When trying to locate the buccal canal, the clinician should place the file tip in the orifice with the tip curved to the buccal side. To explore for the palatal canal, a curved file tip is placed toward the palate. *B,* Buccal; *P,* palatal.
Fig. 7.14 **A,** In a mandibular second molar with two canals, both orifices are in the mesiodistal midline. **B,** If two orifices are not directly in the mesiodistal midline, a search should be made for another canal on the opposite side in the area of "X." *D,* Distal; *M,* mesial.
Fig. 7.15 Schematic representation of isthmus classifications described by Kim and colleagues. Type I is an incomplete isthmus; it is a faint communication between two canals. Type II is characterized by two canals

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with a definite connection between them (complete isthmus). Type III is a very short, complete isthmus between two canals. Type IV is a complete or incomplete isthmus between three or more canals. Type V is marked by two or three canal openings without visible connections.

Fig. 7.16 A, Mesial view of a mandibular premolar with a type V canal configuration. The lingual canal separates from the main canal at nearly a right angle. **B,** This anatomy requires widening of access in a lingual direction to achieve straight-line access to the lingual canal. This should be done with a DOM.

Fig. 7.17 A, Morphology of the root apex. From its orifice the canal tapers to the apical constriction, or minor apical diameter, which generally is considered the narrowest part of the canal. From this point the canal widens as it exits the root at the apical foramen, or major apical diameter. The space between the minor and major apical diameters is funnel shaped. **B,** Clinical view of major apical foramen. **C,** Histologic view of the apical constriction *(arrow)* and apical foramen *(yellow line)*. *P,* Pulp. (**B** and **C,** From Gutmann JL, Lovdahl PE: *Problem solving in endodontics,* ed 5, St. Louis, 2011, Elsevier.)

Fig. 7.18 Root apex following root canal filling *(RCF)* short of the actual root length. Histologic evidence shows that hard tissue *(black arrows)* has formed from the cells of the periodontal ligament *(PDL)* adjacent to the root filling material. Cementum formation on the internal aspect of the apical foramen is indicated by the yellow arrows. These findings accentuate the variable nature of the apical tissues.

Fig. 7.19 A, Straight-line access to a canal. The instrument should not be deflected until it reaches the initial canal curvature. In some cases coronal tooth structure must be sacrificed to obtain direct access to the pulp chamber. **B,** Diagrammatic representation of centrality and concentricity of symmetry and location of canal orifices. *D,* Distal; *M,* mesial.

Fig. 7.20 An incisal access cavity on mandibular anterior teeth may allow for improved straight-line access and canal débridement.

Fig. 7.21 A, Maxillary molar requiring root canal procedure. **B,** Removal of the amalgam reveals a vertical fracture on the palatal margin. **C,** Complete cleaning and shaping of the canals. Fracture lines are still visible, but no probings are present.

Fig. 7.22 A, A photograph of a mandibular molar showing minimally

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invasive access cavity outline. **B,** A sagittal view of a μCT reconstruction showing minimally invasive access cavity pathway. **C,** A photograph of a mandibular molar showing traditional access cavity outline. **D,** A sagittal view of a μCT reconstruction showing traditional access cavity pathway.

Fig. 7.23 **A,** A schematic diagram of the traditional access cavity outline *(A, black dotted line)* and minimally invasive access cavity outline *(B, red dotted line)* in a mandibular molar. **B,** A photograph of a mandibular molar showing mesial access and orifice. **C,** A photograph of a mandibular molar showing distal access and orifice.

Fig. 7.24 Merged μCT images of a mandibular first molar demonstrating the root canals pretreatment *(green)* and posttreatment *(red)*. **A,** Buccal view. **B,** Distal view. **C,** Lingual view. **D,** Mesial view.

Fig. 7.25 Access burs. **A,** #2, #4, and #6 round carbide burs. **B,** Safety-tip tapered diamond bur *(left)*; safety-tip tapered carbide bur *(right)*. **C,** Transmetal bur. **D,** Round-end cutting tapered diamond bur. **E,** #57 fissure carbide bur. **F,** #2 and #4 round diamond burs. **G,** Mueller bur *(left)*; LN bur or Extendo Bur *(right)*.

Fig. 7.26 **A,** In anterior teeth, the starting location for the access cavity is the center of the anatomic crown on the lingual surface *(X)*. **B,** Preliminary outline form for anterior teeth. The shape should mimic the expected final outline form, and the size should be half to three fourths the size of the final outline form. **C,** The angle of penetration for the preliminary outline form is perpendicular to the lingual surface. **D,** The angle of penetration for initial entry into the pulp chamber is nearly parallel to the long axis of the root. **E,** Completion of removal of the pulp chamber roof; a round carbide bur is used to engage the pulp horn, cutting on a lingual withdrawal stroke.

Fig. 7.27 Lingual shoulder of the anterior tooth, extending from the cingulum to 2 mm apical to the orifice.

Fig. 7.28 Placing an incisal bevel on the lingual surface of a maxillary anterior tooth can lead to fracture of the permanent restoration during occlusal function.

Fig. 7.29 **A,** The lingual ledge of dentin remains, deflecting the file toward the labial wall. As a result, portions of the lingual canal wall will not be shaped and cleaned. **B,** Removal of the lingual ledge results in straight-line access.

Fig. 7.30 **A,** Starting location for access to the maxillary premolar *(X)*. **B,**

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Initial outline form *(dark area)* and projected final outline form *(dashed line)*. *B*, Buccal; *L*, lingual.

Fig. 7.31 The crown of a mandibular premolar is tilted lingually relative to the root. *B*, Buccal; *L*, lingual.

Fig. 7.32 **A,** Mandibular first premolar and access starting location *(X)* (occlusal view). **B,** Mandibular first premolar and starting location (proximal view). **C,** Mandibular second premolar and access starting location *(X)* (occlusal view). **D,** Mandibular second premolar and starting location (proximal view). *B*, Buccal; *DL*, distolingual; *L*, lingual; *ML*, mesiolingual.

Fig. 7.33 **A,** Mesial and distal boundary of a maxillary molar with the access starting location *(X)*. **B,** Mesial and distal boundary of a mandibular molar showing the access starting location *(X)*. *D*, Distal; *M*, mesial; *MB*, mesiobuccal; *ML*, mesiolingual.

Fig. 7.34 **A,** Periapical radiograph of the teeth in the maxillary right quadrant. **B,** Bitewing radiograph of the same teeth provides a clearer delineation of the pulpal morphology.

Fig. 7.35 An endodontic explorer is used to search for canal orifices.

Fig. 7.36 Safety-tip carbide bur is used to shape the axial wall in one plane from the orifice to the cavosurface margin. *B*, Buccal; *P*, palatal.

Fig. 7.37 A fiberoptic light can be applied to the cervical aspect of the crown to help obtain maximal visibility with magnification. Transillumination often reveals landmarks otherwise invisible to the unaided eye.

Fig. 7.38 Mandibular molar with what appears to be almost complete calcification of the pulp chamber and root canals. However, pathosis is present, which indicates the presence of bacteria and some necrotic tissue in the apical portion of the roots.

Fig. 7.39 Allowing sodium hypochlorite (NaOCl) to remain in the pulp chamber may help locate a calcified root canal orifice. Tiny bubbles may appear in the solution, indicating the position of the orifice. This is best observed through magnification.

Fig. 7.40 Mandibular first molar with a class I restoration, calcified canals, and periradicular radiolucencies. Presumably a pulp exposure has occurred, resulting in calcification and ultimate necrosis of the pulp tissue.

Fig. 7.41 Excavation of a restoration and base material. The cavity preparation is extended toward the assumed location of the pulp chamber, keeping in mind that pulp chambers are located in the center of the tooth at

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the level of the cementoenamel junction (CEJ).
Fig. 7.42 Use a long-shank #2 or #4 round bur to remove dentin and attempt to locate calcified canals.
Fig. 7.43 An endodontic explorer is used to probe the pulp floor. A straight ultrasonic tip may be used to remove dentin. Angled radiographs must be taken to monitor progress.
Fig. 7.44 At the first indication of a canal space, the smallest instrument (i.e., a #.06 or #.08 C or C+ file or micro openers) should be introduced into the canal. Gentle passive movement, both apical and rotational, often produces some penetration. A slight pull, signaling resistance, usually is an indication that the canal has been located. This should be confirmed by radiographs.
Fig. 7.45 A small K-file negotiates the canal to its terminus. An apex locator or radiograph is used to confirm the file's position.
Fig. 7.46 **A,** Access cavity on crowded mandibular anterior teeth. The access preparation is cut through the buccal surface on the canine. The lateral incisor has also been accessed through the buccal surface; root canal procedures were performed, and the access cavity was permanently restored with composite. **B,** Obturation.
Fig. 7.47 Maxillary central incisor. Average time of eruption—7 to 8 years; average age of calcification—10 years; average length—22.5 mm. Root curvature (most common to least common): straight, labial, distal.
Fig. 7.48 μCT scans of maxillary central incisors. **A,** Common anatomic presentation. **B,** Central incisor with a lateral canal, which is common. **C,** Rare multiple-canal variation. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.3 online at the Expert Consult site for rotational views of these teeth.)
Fig. 7.49 Access cavity for a maxillary central incisor as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×8.4 magnification.
Fig. 7.50 Maxillary lateral incisor. Average time of eruption—8 to 9 years; average age of calcification—11 years; average length—22 mm. Root curvature (most common to least common): distal, straight.
Fig. 7.51 μCT scans of maxillary lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with a large lateral canal, which is common. **C,** Lateral incisor with an apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional

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anatomy at the coronal, middle, and apical levels. (See Video 7.4 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.52 Maxillary canine. Average time of eruption—10 to 12 years; average age of calcification—13 to 15 years; average length—26.5 mm. Root curvature (most common to least common): distal, straight, labial.

Fig. 7.53 μCT scans for the maxillary canine. **A,** Common anatomic presentation. **B,** Canine with two roots. **C,** Canine with significant deviations of the canal system in the apical third. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.5 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.54 Maxillary first premolar. Development and anatomic data: average time of eruption—10 to 11 years; average age of calcification—12 to 13 years; average length—20.6 mm. Root curvature (most common to least common): buccal root—lingual, straight, buccal; palatal root—straight, buccal, distal; single root—straight, distal, buccal.

Fig. 7.55 μCT scans of maxillary first premolars. **A,** Common anatomic presentation of this tooth showing two roots. **B,** Premolar with only one canal. **C,** Premolar with three roots. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.6 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.56 Maxillary second premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 14 years; average length—21.5 mm. Root curvature (most common to least common): distal, bayonet, buccal, straight.

Fig. 7.57 μCT scans of maxillary second premolars. **A,** Common anatomic presentation showing one canal. **B,** Second premolar with two canals and an apical delta. **C,** Second premolar with three roots/canals that divide at the junction of the middle and apical third of the main root. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.7 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.58 Maxillary first molar. Average time of eruption—6 to 7 years; average age of calcification—9 to 10 years; average length—20.8 mm. Root curvature (most common to least common): mesiobuccal root—distal,

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straight; distobuccal root—straight, mesial, distal; palatal root—buccal, straight.

Fig. 7.59 μCT scans of maxillary first molars. **A,** Common anatomic presentation showing accessory/lateral canals. **B,** First molar with four canals, with mesiobuccal and mesiopalatal sharing an anastomosis in the midroot. **C,** Maxillary molar with four pulp horns, five canals, and significant anastomoses between the canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.9 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.60 Maxillary second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 16 years; average length—20 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—straight, buccal.

Fig. 7.61 μCT scans of maxillary second molars; four possible variations. **A,** Uncommon anatomic presentation of this tooth with one canal. **B,** Second molar with two canals. **C,** Second molar with three canals. **D,** Second molar with four distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.10 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.62 Maxillary third molar. Average time of eruption—17 to 22 years; average age of calcification—18 to 25 years; average length—17 mm.

Fig. 7.63 μCT scans of maxillary third molars show a range of anatomic variations. **A,** Single-canal tooth. **B,** Two-rooted third molar. **C,** Two-rooted, three-canal third molar with significant root curvatures. **D,** Three-rooted, four-canal third molar. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.11 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.64 Mandibular central/lateral incisors. Average time of eruption—6 to 8 years; average age of calcification—9 to 10 years; average length—20.7 mm. Root curvature (most common to least common): straight, distal, labial.

Fig. 7.65 μCT scans of mandibular central incisors. **A,** Common anatomic presentation. **B,** Central incisor with two canals. **C,** Central incisor with an

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apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.12 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.66 μCT scans of mandibular lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with broad, thin buccolingual anatomy. **C,** Lateral incisor in which the canal splits into two but returns to form one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.13 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.67 Mandibular canine. Average time of eruption—9 to 10 years; average age of calcification—13 years; average length—25.6 mm. Root curvature (most common to least common): straight, distal, labial.

Fig. 7.68 μCT scans of mandibular canines. **A,** Common anatomic presentation. **B,** Canine with an extra apical canal. **C,** Canine that splits into two but returns to one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.14 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.69 Mandibular first premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 13 years; average length—21.6 mm. Root curvature (most common to least common): straight, distal, buccal.

Fig. 7.70 μCT scans of mandibular first premolars. **A,** Common anatomic presentation. **B,** First premolar with significant canal deviations in the middle to apical third before returning to a single large canal apically and a small deviating canal to the proximal. **C,** First premolar with a branching main canal lingually and multiple accessory canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.15 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.71 Mandibular second premolar. Average time of eruption—11 to 12 years; average age of calcification—13 to 14 years; average length—22.3 mm. Root curvature (most common to least common): straight, distal, buccal.

Fig. 7.72 μCT scans of mandibular second premolars. **A,** Common anatomic presentation. **B,** Second premolar with significant canal deviations in the

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middle to apical third. **C,** Second premolar with fused root that exhibits two distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.16 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.73 Mandibular first molar. Average time of eruption—6 years; average age of calcification—9 to 10 years; average length—21 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal.

Fig. 7.74 μCT scans of mandibular first molars. **A,** Common anatomic presentation. **B,** First molar with three main canals and a deviant fourth canal/fourth root. **C,** First molar with wide connections or anastomoses between the mesial canals, demonstrating multiple canal exits. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.17 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.75 **A,** A photograph (×8) of a mandibular molar showing fins between MB and ML canals**. B,** A photograph (×8) of a mandibular molar showing three mesial canals. *MB,* Mesiobuccal; *ML,* mesiolingual.

Fig. 7.76 **A,** Radix entomolaris. Notice lingual position of its orifice in relation to the two canals in the distal root. **B,** Radiograph with obvious radix entomolaris on mandibular first molar that curves to the buccal. The reader is referred to a recent publication that details extensively this type of anatomy (see following credit for part **B**). (**A,** Courtesy Dr. William J. Aippersbach, Venice, FL. **B,** From Abella F, Patel S, Durán-Sindreu F, Mercadé M, Roig M: Mandibular first molars with disto-lingual roots: review and clinical management, *Int Endod J* 45:963–978, 2012.)

Fig. 7.77 Mandibular second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 15 years; average length—19.8 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal, mesial, buccal; single root—straight, distal, bayonet, lingual.

Fig. 7.78 μCT scans of mandibular second molars. **A,** Two-canal second molar with fused roots. **B,** Second molar with three initial canals ending in one canal apically in both roots. **C,** Second molar with four distinct canals.

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All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.18 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.79 Mandibular third molar. Average time of eruption—17 to 21 years; average age of calcification—18 to 25 years; average length—18.5 mm.

Fig. 7.80 μCT scans of mandibular third molars represent multiple variations for this tooth. **A**, Single C-shaped type canal. **B,** Complex anatomy with significant canal curvatures apically. **C,** Three canals that curve in multiple directions. **D,** Flattened, ribbon-shaped canals with significant apical curvatures. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.19 online at the Expert Consult site for rotational views of these teeth.)

Fig. 7.81 **A,** Diagram of a C-shaped canal anatomy: one continuous canal from pulp chamber floor to apex. **B,** One large canal moving from the top of the C-shaped canal *(TCS)* to the apical-most or bottom of C-shaped canal *(BCS)*, encompassing most of the canal space. *CEJ*, Cementoenamel junction.

Fig. 7.82 C-shaped canal anatomy. **A,** Mandibular second molar. **B,** Maxillary first molar.

Fig. 7.83 Types of pulpal floor. *M*, Mesial side; *D*, distal side.

Fig. 7.84 Three-dimensional classification of C-shaped canal configuration. **A,** Merging type. **B,** Symmetrical type. **C,** Asymmetrical type. **D,** Additional C-shaped canal variations.

Fig. 8.1 Effect of routine root canal treatment of a mandibular molar. **A,** Pretreatment radiograph of tooth #19 shows radiolucent lesions adjacent to both mesial and distal root apices. **B,** Working length radiograph shows two separate root canals in the mesial root and two merging canals in the distal root. **C,** Posttreatment radiograph after shaping of root canal systems with nickel-titanium rotary files and obturation with thermoplasticized gutta-percha. **D,** Six-month recall radiograph after restoration of tooth #19 with an adhesively inserted full ceramic crown; some periradicular bone fill can be seen. **E,** One-year recall radiograph displays evidence of additional periradicular healing. **F,** Five-year recall radiograph; tooth not only is periapically sound but also clinically asymptomatic and fully functional.

Fig. 8.2 Root canal treatment in a case of apical and interradicular pathosis.

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A, Pretreatment radiograph of tooth #19 shows an interradicular lesion. **B–C,** Posttreatment radiographs after root canal preparation and obturation. Note the lateral canal in the coronal third of the root canal. **D–E,** Two-month recall radiograph suggests rapid healing.

Fig. 8.3 Root canal therapy as part of a comprehensive treatment plan. The patient, who was recovering from intravenous drug addiction, requested restorative dental treatment. Because of extensive decay, several teeth had to be extracted, and nine teeth were treated endodontically. Root canal treatment was aided by nickel-titanium rotary instruments, and obturation was done with lateral compaction of gutta-percha and AH26 as the sealer. Microsurgical retrograde therapy was performed on tooth #8, and the distobuccal root of #14 had to be resected. Metal-free adhesively luted restorations were placed, and missing mandibular teeth were replaced by implants. **A,** Pretreatment intraoral status, showing oral neglect. **B,** Posttreatment intraoral status at 4-year follow-up, showing fully functional, metal-free, tooth-colored reconstructions. **C,** Panoramic radiograph at 4-year recall shows sound periradicular tissues in relation to endodontically treated teeth.

Fig. 8.4 Example of a desired shape, with the original root canal fully incorporated into the prepared outline. **A–B,** Micro–computed tomography reconstructions in clinical and mesiodistal views of a maxillary molar prepared with a NiTi rotary system. The *green area* indicates the pretreatment shape, and the *red area* indicates the posttreatment shape. Areas of *mixed red and green* indicate no change (i.e., no removal of radicular dentin). **C–E**, Cross sections of the coronal, middle, and apical thirds; the pretreatment cross sections *(green)* are encircled by the posttreatment outlines *(red)* in most areas.

Fig. 8.5 Example of excessive thinning of dental structure during root canal treatment. **A–B**, Micro–computed tomography reconstructions show pretreatment and posttreatment root canal geometry of a maxillary molar. **C–E**, Cross sections of the coronal, middle, and apical thirds with pretreatment canal cross sections. Note the transportation and thinning, in particular, in the main mesiobuccal canal.

Fig. 8.6 Presence of microorganisms inside the main root canal and dentinal tubules. **A,** Scanning electron micrograph of a root canal surface shows a confluent layer of rod-shaped microbes (×3000). **B,** Scanning electron

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micrograph of a fractured root with a thick smear layer and fungi in the main root canal and dentinal tubules.

Fig. 8.7 Irrigation needles inserted into prepared root canals. **A–B**, A 27-gauge needle barely reaches the middle third. **C–D**, A 30-gauge, side-venting needle reaches the apical third in adequately enlarged canals.

Fig. 8.8 Spectrum of strategies for accomplishing the primary aim of root canal treatment: elimination of infection. **A,** Schematic diagram of minimally invasive therapy using the noninstrumentation technique (NIT). **B,** Example of teeth cleaned in vitro using NIT. Note the clean intracanal surface, which is free of adhering tissue remnants. **C–D**, Examples of teeth cleaned in vivo and later extracted to investigate the clinical effects of NIT. Note the relatively clean, tissue-free canal space in **C** and the significant tissue revealed by rhodamine B staining in **D. E–F**, Course of maximally invasive therapy; apically involved tooth #30 was extracted, effectively removing the source of periradicular inflammation.

Fig. 8.9 Panel of 36 anatomic preparations of maxillary molars from the classic work by Professor Walter Hess of Zurich. Note the overall variability of root canal systems and the decrease of canal dimensions with age.

Fig. 8.10 Sinus tract as a sign of a chronic apical abscess and effect of routine root canal treatment. **A,** Intraoral photograph of left maxillary region with draining sinus tract *(arrow)* periapical to tooth #14. **B,** Pretreatment radiograph with gutta-percha point positioned in the sinus tract, pointing toward the distobuccal root of #14. **C,** Finished root canal fillings after 2 weeks of calcium hydroxide dressing. **D,** Intraoral photograph of the same region as in **A,** showing that the sinus tract had closed by the time obturation was performed.

Fig. 8.11 Relationship of radicular anatomy and endodontic disease as shown by filled accessory canals. **A,** Working length radiograph of tooth #13 shows lesions mesially and distally but not apically. **B,** Posttreatment radiograph shows the accessory anatomy. **C,** Six-month recall radiograph before placement of the restoration. **D,** Two-year recall radiograph after resection of the mesiobuccal root of tooth #14 and placement of a fixed partial denture. Excess sealer appears to have been resorbed, forming a distal residual lesion. **E,** Four-year recall radiograph shows almost complete bone fill. **F,** Seven-year recall radiograph; tooth #14 is radiologically sound and clinically within normal limits.

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Fig. 8.12 Comparison of the flute geometry and tip configuration of a hand file *(insert)* and a NiTi rotary instrument. **A,** K-file with sharp cutting edges *(arrow)* and Batt tip *(arrowhead)*. **B,** GT rotary file with rounded, noncutting tip *(arrowhead)*, smooth transition, and guiding radial lands *(arrow)*.

Fig. 8.13 Design characteristics of nickel-titanium rotary instruments. **A,** Lateral view showing the details of the helical angle, pitch *(p)*, and the presence of guiding areas, or radial lands *(rl)* (scanning electron micrograph, ×25). **B,** Ground-working part of the instrument in **A,** showing U-shaped excavations and the dimension of the instrument core *(c)*.

Fig. 8.14 The cutting angle of an endodontic file can be negative (**A**), neutral (**B**), or positive (**C**).

Fig. 8.15 Schematic drawing of an ISO-normed hand instrument size #35. Instrument tip sizing, taper, and handle colors are regulated by the ISO/ANSI/ADA norm.

Fig. 8.16 Increase in tip diameter in absolute figures and in relation to the smaller file size. Note the particularly large increase from size #10 to size #15.

Fig. 8.17 Scanning electron micrographs of endodontic hand files fabricated by twisting (K-file size #40, **A**) and grinding (Hedström file #50, **B**).

Fig. 8.18 Stress-strain behavior of nickel-titanium alloy. **A,** Schematic diagram of linear extension of a NiTi wire. **B,** Torque to failure test of a size #60, .04 taper ProFile NiTi instrument. Note the biphasic deformation, indicated by arrows in **A–B. C,** Comparison of stainless steel and nickel-titanium crystal lattices under load. Hookian elasticity accounts for the elastic behavior *(E)* of steel, whereas transformation from martensite to austenite and back occurs during the pseudoelastic *(PE)* behavior of NiTi alloy.

Fig. 8.19 Pseudoelastic behavior of nickel titanium is based on the two main crystal configurations, martensite and austenite, which depend on temperature (**A**) and applied strain (**B**). Formation of the respective configuration initiates at the start temperatures, M_s and A_s.

Fig. 8.20 Physical factors (torque, axial force, and insertion depth) that affect root canal instrumentation documented with a torque-testing platform. **A,** ProFile size #45, .04 taper used in a mildly curved canal of a single-rooted tooth, step-back after apical preparation to size #40. **B,** FlexMaster size #35, .06 taper used in a curved distobuccal canal of a maxillary first molar, crown-

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down during the initial phase of canal preparation.
Fig. 8.21 Deformation of endodontic instruments manufactured from nickel-titanium alloy. **A** and **B,** Intact and plastically deformed ProFile instruments (*arrow* indicates areas of permanent deformation). **C,** ProFile instrument placed on a mirror to illustrate elastic behavior.
Fig. 8.22 Result of an overenthusiastic attempt at root canal treatment of a maxillary second molar with large stainless steel files. Multiple strip perforations occurred, so consequently the tooth had to be extracted.
Fig. 8.23 Scanning electron micrograph of a barbed broach.
Fig. 8.24 Various Gates-Glidden (GG) burs made of stainless steel (**A**) and scanning electron micrograph (**B**, working tip).
Fig. 8.25 Removal of a fractured NiTi instrument from a mesiolingual canal of a mandibular molar. **A,** Fragment located in the middle third of the root. **B,** Clinical aspect of the fragment after enlargement of the coronal third of the root canal with modified Gates-Glidden drills, visualized with an operating microscope (×25). **C,** Radiograph taken after removal of the fragment; four hand files have been inserted into the canals. **D,** Final radiograph shows slight widening of the coronal third of the mesiolingual canal and fully sealed canal systems. A full crown was placed immediately after obturation. **E,** Recall radiograph 5 years after obturation shows sound periradicular tissues. **F,** Removed fragment and separated file (gradation of ruler is 0.5 mm).
Fig. 8.26 The SAF instrument. The instrument is made as a hollow, thin NiTi lattice cylinder that is compressed when inserted into the root canal and adapts to the canal's cross section. It is attached to a vibrating handpiece. Continuous irrigation is applied through a special hub on the side of its shank.
Fig. 8.27 Scanning electron micrographs (SEM) of current nickel-titanium rotary instruments, seen laterally (*left panel*, ×50) and in cross sections (*right panel*, ×100). Note the radial landed cross section of the *GTX* instrument (*red arrow*).
Fig. 8.28 Design features of a ProFile instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.
Fig. 8.29 Example of an ultrasonic unit (Neutron P5 piezoelectric).
Fig. 8.30 Examples of motors used with rotary nickel-titanium endodontic instruments. **A,** First-generation motor without torque control. **B,** Fully

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electronically controlled second-generation motor with sensitive torque limiter. **C,** Frequently used simple torque-controlled motor. **D,** Newer-generation motor with built-in apex locator and torque control.

Fig. 8.31 Diagram comparing fracture loads at D_3 *(upper section of graph)* to torques occurring during preparation of root canals *(lower section of graph)*. Filled columns represent the largest file in each set, and open columns show the scores of the most fragile file (see text and Box 8.3 for details).

Fig. 8.32 Sequence of instruments used for optimal preparation of an access cavity (e.g., in an incisor). A parallel-sided diamond or steel bur is used to remove overlying enamel in a 90-degree angle toward the enamel surface *(1)*. The bur is then tilted vertically to allow straight-line access to the root canal *(arrow)*. A bur with a noncutting tip (e.g., Endo-Z bur or ball-tipped diamond bur) is then used to refine access *(2)*. Overhangs or pulp horns filled with soft tissue are finally cleared with a round bur used in a brushing or pulling motion *(3)*.

Fig. 8.33 Clinical views of an access cavity in a mandibular molar as seen through an operating microscope (×20). **A,** Modification with an ultrasonically activated tip (**B**).

Fig. 8.34 Diagram of coronal enlargement in a more complicated maxillary posterior tooth. This maxillary molar presents several difficulties, including a narrow mesiobuccal canal that exits the pulp cavity at an angle. A possible approach in a case involving difficult entry into the root canal system is to use a small orifice shaper *(OS1)* after ensuring a coronal glide path with a K-file. Use of a sequence of orifice shapers *(OS3 to OS1)* then allows penetration into the middle third of the root canal. Wider canals can accept a second sequence of orifice shapers. Copious irrigation and securing a glide path with a size #10 K-file are prerequisites for use of NiTi rotary instruments.

Fig. 8.35 Various precurved, stainless steel hand files for path finding and gauging. Compare the curves in the instruments to the ones in a plastic training block (gradation of ruler is 0.5 mm).

Fig. 8.36 Root ZX apex locator with lip clip and file holder.

Fig. 8.37 Microcomputed tomographic scan of anatomy of the apical 5 mm of a mesiobuccal root (8 µm resolution). **A–B**, Three-dimensional reconstruction of outer contour and root canal systems. **C-L,** Cross sections

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0.5 mm apart.

Fig. 8.38 Presence of dentin dust as a possible source of microbial irritation. Tooth #18 underwent root canal therapy. The clinician noted an apical blockage but was unable to bypass it. Unfortunately, intense pain persisted and at the patient's request, the tooth was extracted a week later. **A,** Mesial root of tooth #18; mesial dentin has been removed. **B,** Magnified view (×125) of rectangle in **A** shows an apical block (gradation of ruler is 0.5 mm).

Fig. 8.39 Schematic diagrams showing the most common preparation errors. **A,** Apical zip. **B,** Ledge. **C,** Apical zip with perforation. **D,** Ledge with perforation.

Fig. 8.40 Diagram of handle movements during balanced force hand preparation. STEP 1: After pressureless insertion of a Flex-R or NiTiFlex K-file, the instrument is rotated clockwise 90 degrees, using only light apical pressure. STEP 2: The instrument is rotated counterclockwise 180 to 270 degrees; sufficient apical pressure is used to keep the file at the same insertion depth during this step. Dentin shavings are removed with a characteristic clicking sound. STEP 3: This step is similar to step 1 and advances the instrument more apically. STEP 4: After two or three cycles, the file is loaded with dentin shavings and is removed from the canal with a prolonged clockwise rotation.

Fig. 8.41 Instrumentation of root canals with the single length technique, in this example using the ProTaper instruments. After irrigation and scouting (*1* and *2*), the coronal thirds are enlarged with shaping files *S1* and *S2*. Hand files then are used to determine the WL and to secure a glide path. Apical preparation is completed with S1 and S2. Finishing files are used to the desired apical width.

Fig. 8.42 Stepwise enlargement of mesial root canal systems in an extracted mandibular molar demonstrated with micro–computed tomography (μCT) reconstructions. The buccal canal *(left)* was prepared with a LightSpeed (LS) instrument, and the lingual canal *(right)* was shaped with a ProTaper (PT) instrument. **A,** Pretreatment view from the mesial aspect. Note the additional middle canal branching from the lingual canal into the coronal third. **B,** Initial preparation and opening of the orifices, aided by ultrasonically powered instruments. **C,** First step of root canal preparation, up to LightSpeed size #20 and ProTaper shaping file S1. **D,** Further enlargement to LS size #30 and PT shaping file S2. **E,** Apical preparation to LS size #40 and PT finishing file F1.

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F, Additional enlargement to LS size #50 and PT finishing file F2. **G,** Superimposed μCT reconstructions comparing the initial canal geometry *(in green)* with the shape reached after use of the instruments shown in **F**. **H,** Final shape after step-back with LS instruments and PT finishing file F3. **I,** Superimposed μCT reconstructions comparing initial geometry and final shape. Note the slight ledge in the buccal canal after LS preparation and some straightening in the lingual canal after PT preparation.

Fig. 8.43 Remaining potentially infected tissue in fins and isthmus configuration after preparation with rotary instruments. **A,** Cross section through a mesial root of a mandibular molar, middle to coronal third of the root. Both canals have been shaped; the left one is transported mesially (×10). **B,** Magnified view of rectangle in **A**. Note the presence of soft tissue in the isthmus area (×63).

Fig. 8.44 Effect of a hybrid technique on root canal anatomy studied in a Bramante model. **A1–A4,** Both mesial canals of an extracted mandibular molar have been instrumented. Canal cross sections are shown before instrumentation **(B1–D1). B2–D2**, Cross sections after preenlargement with a ProTaper F3 file *(left canal)* and a size #45, .02 taper instrument *(right canal)*. The final apical sizes were LightSpeed (LS) #50 and size #50, .02 taper in the left and the right canal, respectively.

Fig. 8.45 Irrigation and movement of irrigants depends on canal shape. Sequential enlargement of a canal in clear plastic block was performed with a sequence of ProFile instruments in accordance with the manufacturer's recommendations. Alternating irrigation with blue and red fluid was done after each preparation step. Note the apical presence of irrigant after sufficient shape has been provided. Note the distribution of fluid immediately after irrigation with a 30-gauge needle.

Fig. 8.46 A, Various types of needles for root canal irrigation. Shown are examples with open end and closed end, side vented. These are manufactured from plastic and stainless steel. **B,** SEM image of 30 gauge safety needle.

Fig. 8.47 Schematic diagram of the mechanism of action of NaOCl with the main interactions and properties highlighted. This diagram shows the chemical reaction of NaOCl when in contact with organic matter. The solution produces glycerol and soap (saponification reaction) and acts as a fat solvent degrading fatty acids. Moreover, hypochlorous acid is formed with a reduction of the pH and formation of chloramines (chloramination reaction).

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Damage to the cell membrane, denaturalization of proteins, and DNA damage may be observed.

Fig. 8.48 Effect of heating on the ability of 0.5% sodium hypochlorite (NaOCl) to dissolve pulp tissue: NaOCl heated to 113°F (45°C) dissolved pulp tissue as well as the positive control (5.25% NaOCl) did. When the NaOCl was heated to 140°F (60°C), almost complete dissolution of tissue resulted.

Fig. 8.49 Device for heating syringes filled with irrigation solution (e.g., sodium hypochlorite) before use.

Fig. 8.50 Toxic effect of sodium hypochlorite on periradicular tissues. After root canal treatment of tooth #3, the patient reported pain. **A,** On a return visit, an abscess was diagnosed and incised. **B,** Osteonecrosis was evident after 3 weeks.

Fig. 8.51 Schematic drawing of the chlorhexidine molecule.

Fig. 8.52 Red precipitate forming after contact between NaOCl and chlorhexidine. **A,** When 2% chlorhexidine (CHX) is mixed with different concentration of NaOCl, a change of color and precipitate occurs. The higher the concentration of NaOCl, the larger is the precipitate formation. **B,** Detail of the interaction between 2% CHX and 5% NaOCl.

Fig. 8.53 Penetration of irrigants into dentinal tubules after root canal preparation with different dentin pretreatments. *Left column,* Irrigation with tap water and then with blue dye. *Right column,* Smear layer is removed with 17% EDTA, applied in high volume and with a 30-gauge needle, followed by irrigation with blue dye. Note the comparable diffusion of dye in the apical sections, whereas dye penetrated deeper into the dentin in the two coronal sections.

Fig. 8.54 Example of canals with minimal smear layer. **A,** Middle third after irrigation with 17% ethylenediaminetetraacetic acid (EDTA) and 2.5% sodium hypochlorite (NaOCl). **B,** Apical third with some particulate debris.

Fig. 8.55 Scanning electron microscope (SEM) image of root canal dentin exposed to 2 minutes of EDTA.

Fig. 8.56 Container with BioPure MTAD.

Fig. 8.57 QMiX 2in1. Combination of CXH, EDTA, and detergent. QMix irrigating solution is a single solution used as a final rinse after bleach for one-step smear layer removal and disinfection

Fig. 8.58 **A,** Application of calcium hydroxide paste in the canal with a

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lentulo spiral. **B,** Calciject is a calcium hydroxide prefilled, easy-to-use, single-dose syringe system. Centrix NeedleTube cartridges can be used for direct syringe injection into the root canal.

Fig. 8.59 The EndoActivator, a sonic frequency system.

Fig. 8.60 The EndoVac system. **A,** Magnification of the closed-ended microcannula (**A**). **B,** Clinical view of the EndoVac system combined with the Safety-Irrigator.

Fig. 8.61 The Safety-Irrigator.

Fig. 8.62 The GentleWave system (Sonendo, Irvine, CA, USA). The system uses multisonic energy to develop a broad section of waves within the irrigation solution to clean inside the roots canal system. It has two main components: a hand piece and a console (shown in figure).

Fig. 8.63 Testing platform for analysis of various factors during simulated canal preparation with rotary endodontic instruments. Labeled components are a force transducer *(A)*, a torque sensor *(B)*, a direct-drive motor *(C)*, and an automated feed device *(D)*. For specific tests, a cyclic fatigue phantom or a brass mount compliant with ISO No. 3630-1 *(inserts)* may be attached.

Fig. 8.64 Instruments with increased taper that can be used by hand. **A,** ProTaper instruments with special handles attached to rotary instrument shanks. **B,** GT hand instruments.

Fig. 8.65 Evidence of coronal hard-tissue deposition. **A,** Periapical radiograph of tooth #19 shows evidence of reduced coronal and radicular pulp space. **B,** Intraoral photograph, taken through an operating microscope (×25), of access cavity of the tooth shown in **A**; note the calcific metamorphosis.

Fig. 8.66 Clinical cases treated according to the principles detailed in this chapter. **A,** Pretreatment radiograph of tooth #30 with a periradicular lesion. **B,** Postobturation radiograph. **C,** Two-year follow-up radiograph shows osseous healing. **D,** Immediate postobturation radiograph of tooth #29 shows both a periapical and a lateral osseous lesion. **E–F,** One-year and 3-year follow-up radiographs show progressing osseous healing. Note the imperfect obturation of tooth #30.

Fig. 8.67 Complicated clinical cases treated with hybrid techniques. **A,** Pretreatment radiograph of tooth #16 indicates laceration and significant curvature of all roots. **B,** Posttreatment radiograph shows multiple planes of curvature. **C,** Pretreatment radiograph of tooth #19, which was diagnosed

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with irreversible pulpitis. **D,** Angulated posttreatment radiograph shows three canals in the mesiobuccal root canal system, all of which were prepared to apical size #50.

Fig. 9.1 Examples of inadequate obturation. **A,** Maxillary right canine with adequate length but lacking density and no coronal seal. Central incisor is filled to adequate length, but obturation exhibits voids. **B,** Maxillary central incisors. Maxillary right central incisor exhibits a lack of density and taper. Maxillary left central incisor has voids and unfilled canal space. **C,** Mandibular left first molar with adequate obturation; provisional restoration shows poor adaptation on the distal because of the failure to remove caries.

Fig. 9.2 **A,** Posttreatment radiograph of a mandibular left lateral incisor with ostensibly adequate obturation. **B,** Angled view reveals voids.

Fig. 9.3 Histologic section of a root apex, demonstrating anatomy of the classic foramen and constriction.

Fig. 9.4 Histologic sections demonstrating the foramen exiting short of the root apex.

Fig. 9.5 Scanning electron microscopy of a tooth exhibiting a necrotic pulp and apical pathosis and resorption.

Fig. 9.6 Scanning electron microscopy of the apex of an extracted tooth that was removed because of pulp necrosis. Note the multiple accessory foramina and resorption.

Fig. 9.7 Histologic section of a mesial root of a mandibular molar with a lateral canal present and associated lesion. Will the lesion resolve after the removal of the main canal contents, or will the lesion persist because of necrotic pulpal remnants in the lateral canal? The question remains unanswered.

Fig. 9.8 Posttreatment radiograph of a mandibular right first molar with a lateral canal associated with the distal root.

Fig. 9.9 Scanning electron microscopy of a prepared canal wall. The tubules are covered with a smear layer of organic and inorganic material.

Fig. 9.10 Scanning electron microscopy of the canal wall after removal of the smear layer with 17% EDTA and 5.25% sodium hypochlorite.

Fig. 9.11 A periapical radiograph of a mandibular left second premolar and first molar, demonstrating Sargenti paste NS RCT. In addition to the toxic material, the technique often accompanies inadequate C&S procedures.

Fig. 9.12 **A,** Posttreatment radiograph of a maxillary right first molar

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demonstrating adequate length, density, and taper. **B,** Posttreatment radiograph of a mandibular right first molar with an adequate obturation.
Fig. 9.13 **A,** Extrusion of sealer evident on the posttreatment radiograph of a maxillary first molar. The separated lentulo spiral in the mesiobuccal root indicates a possible method of sealer placement. **B,** Maxillary occlusal film demonstrates that the sealer is located in the maxillary sinus. Correction by nonsurgical techniques is not possible. **C,** Maxillary right first molar with extrusion of the sealer and GP.
Fig. 9.14 Activ GP (Brasseler USA, Savannah, GA) glass ionomer–coated GP points and sealer.
Fig. 9.15 AH Plus sealer is a resin formulation.
Fig. 9.16 GuttaFlow trituration capsule and injection syringe (Coltène/Whaledent, Cuyahoga Falls, OH).
Fig. 9.17 BioRoot RCS (Septodont, Saint-Maur-des- Fossés, France).
Fig. 9.18 iRoot SP (Innovative BioCeramix Inc., Vancouver, Canada; *aka* EndoSequence BC sealer; Brasseler USA, Savannah, GA).
Fig. 9.19 MTA Plus (NuSmile, Houston, TX).
Fig. 9.20 Patient treated with Sargenti paste in her mandibular left second premolar and first molar. **A,** Pretreatment radiograph exhibits an osteolytic response associated with the premolar and a proliferative response associated with the molar. **B,** Posttreatment radiograph of the teeth. **C,** One-year follow-up radiograph exhibiting osseous regeneration apical to the second premolar.
Fig. 9.21 Lentulo spiral used for sealer placement during obturation.
Fig. 9.22 Silver cones have been advocated for ease of placement and length control. **A,** Radiograph of a maxillary right central incisor obturated with a silver cone. **B,** Tissue discoloration indicating corrosion and leakage. **C,** Lingual view indicates coronal leakage. **D,** Corroded silver cone removed from the tooth. **E,** Posttreatment radiograph of the tooth.
Fig. 9.23 Nonstandard GP cones: extra fine, fine fine, fine, medium fine, fine medium, medium, large, and extra-large.
Fig. 9.24 **A,** Standard GP cone sizes #15 to #40. **B,** Standard cones #.06, taper sizes #15 to #40. **C,** Standard cones ProTaper F1, F2, and F3.
Fig. 9.25 Size #30 standard GP points exhibiting #.02, #.04, and #.06 tapers.
Fig. 9.26 Apical root resorption often results in an open apex requiring fabrication of a custom cone. **A,** Pretreatment radiograph of a maxillary left central incisor with pulp necrosis and chronic apical periodontitis. Apical root

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resorption is present. **B,** In fabricating a custom master cone, a GP point is fit several millimeters short before softening in solvent and tamping into place. **C,** Softening the apical 2 to 3 mm in chloroform that has been placed in a tuberculin syringe. **D,** The completed custom cone represents an impression of the apical portion of the RCS. **E,** The posttreatment radiograph with post space prepared. **F,** A 1-year follow-up radiograph demonstrating osseous regeneration.

Fig. 9.27 For large RCS, several GP points can be heated and rolled together, using a spatula or two glass slabs.

Fig. 9.28 Left first mandibular molar. **A,** Pretreatment radiograph. **B,** Working length radiograph. **C,** Coronal access opening, demonstrating the prepared mesiobuccal canal. **D,** Standardized master cones with coronal reference marked. **E,** Standard master cones fit to length as they exhibit minimal taper and permit deeper penetration of the spreader. **F,** Master cone radiograph.

Fig. 9.29 Lateral compaction. **A,** Finger spreader in place. **B,** Fine-medium accessory cone placed in the space created by the spreader. **C,** Finger spreader placed in preparation, creating space for additional accessory cones. **D,** Additional cones are placed until the spreader does not penetrate past the coronal one third of the canal. The cones are then removed at the orifice with heat, and the coronal mass is vertically compacted with a plugger. **E,** Interim radiograph may be exposed to assess the quality of obturation. **F,** Posttreatment radiograph demonstrating adequate length, density, and taper. The gutta-percha is removed to the level of the orifice, and a coronal seal has been established with an adequate provisional restoration.

Fig. 9.30 Vertical root fractures can occur with excessive compaction forces. **A,** Follow-up radiograph of a mandibular left first molar. A deep isolated periodontal probing defect was associated with the buccal aspect of the mesiobuccal root. **B,** Flap reflection revealed a vertical root fracture.

Fig. 9.31 Various pluggers are manufactured for compacting warm GP. **A,** ISO standard spreader. **B,** ISO standard plugger. **C,** Obtura S Kondensers. **D,** Obtura S-Kondensers.

Fig. 9.32 Warm vertical compaction of GP employs heat and various condensers. **A,** Nonstandard cones are selected and fit short of the prepared length because they more closely replicate the prepared canal. **B,** Heated pluggers or spreaders are used to apply heat to the master cone and remove

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the excess coronal material. **C,** A room temperature plugger is used to compact the heated GP. **D,** Apical compaction is complete. **E,** A GP segment is placed in the canal, and heat is applied. **F,** The heated segment is compacted. **G,** The process is repeated for the coronal portion of the canal by placing and heating a segment of GP. **H,** A plugger is again used to compact the heated material. **I,** Completed obturation.

Fig. 9.33 Touch 'n Heat unit.

Fig. 9.34 Continuous wave obturation uses the System B unit. **A,** The System B unit. **B,** System B plugger with a nonstandard cone of similar taper. **C,** System B pluggers.

Fig. 9.35 System B plugger fit.

Fig. 9.36 System B activation and compaction.

Fig. 9.37 Backfill by the Obtura II thermoplastic injection technique.

Fig. 9.38 Thermoplastic techniques are often used in cases with significant canal irregularities. **A,** A pretreatment radiograph of a maxillary central incisor exhibiting internal resorption. **B,** Posttreatment radiograph demonstrates a dense obturation of the resorptive defect with GP.

Fig. 9.39 Obtura III unit with silver tips, GP plugs, and cleaning solution (Obtura Spartan, Earth City, MO).

Fig. 9.40 The Calamus thermoplastic unit (DENTSPLY Tulsa Dental Specialties, Tulsa, OK) for heating and injecting GP.

Fig. 9.41 The Elements obturation unit (Kerr Endo, Orange, CA) for injecting and compacting GP. Note the System B heat source.

Fig. 9.42 The battery-powered HotShot unit (Discus Dental, Culver City, CA) for heating and injecting GP.

Fig. 9.43 The Ultrafil 3D system consists of an injection syringe, GP cannulas, and heating unit (Coltène/Whaledent, Cuyahoga Falls, OH).

Fig. 9.44 Thermafil carrier and size verifier (DENTSPLY Sirona, Tulsa, OK).

Fig. 9.45 GT obturator and instrument (DENTSPLY Sirona, Tulsa, OK).

Fig. 9.46 The Thermafil oven with carrier in place (DENTSPLY Tulsa Dental Specialties, Tulsa, OK).

Fig. 9.47 Thermafil carrier placed in a distal canal.

Fig. 9.48 Apical obturation of accessory canals by the Thermafil technique.

Fig. 9.49 SuccessFil is an additional carrier system (Coltène/Whaledent, Cuyahoga Falls, OH).

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Fig. 9.50 Simplifill carrier and LightSpeed file (LightSpeed Technology, San Antonio, TX/Discus Dental, Culver City, CA).
Fig. 9.51 Simplifill fitted to 1 to 3 mm from the prepared length.
Fig. 9.52 Mineral trioxide aggregate is available as ProRoot MTA. This material is advocated for use in apexification, repair of root perforations, repair of root resorption, root-end fillings, and pulp capping.
Fig. 9.53 Immediate obturation employs a barrier technique to prevent extrusion when the apex is open. This case involves a maxillary left central incisor with pulp necrosis caused by trauma. **A,** Pretreatment radiograph demonstrates a large canal with an open apex. **B,** Working length is established and the RCS is prepared. **C,** Mineral trioxide plug is placed. **D,** The canal is obturated with GP.
Fig. 10.1 Some of the armamentarium needed to perform retreatment at the highest level.
Fig. 10.2 Clinical presentations of posttreatment disease. **A,** Canals that are poorly cleaned, shaped, and obturated. **B,** Mesial canal with apical transport, ledge, and zip perforation. **C,** Strip perforation of the mesial root. **D,** Missed MB2 canal in an upper molar. **E,** Suspected coronal leakage of bacteria and a separated file.
Fig. 10.3 The causes of posttreatment disease. *1,* Intraradicular microorganisms. *2,* Extraradicular infection. *3,* Foreign body reaction. *4,* True cysts.
Fig. 10.4 **A,** Apparently good nonsurgical retreatment with large persistent lesion. **B,** Surgical exposure of apical lesion in situ. **C,** Large lesion removed in toto. **D,** Histopathologic section confirming cystic nature of the lesion. **E,** Four-year postoperative film showing apical scar formation due to the large size of the lesion. The teeth were asymptomatic and in function.
Fig. 10.5 This patient was misdiagnosed for years and underwent unnecessary endodontic therapy. The actual cause of the patient's complaint was nondental pain.
Fig. 10.6 **A,** Buccal aspect of a premolar with posttreatment disease. **B,** Higher magnification reveals a vertical fracture *(arrow).*
Fig. 10.7 **A,** Posttreatment disease. Previous endodontic therapy performed 3 years previously. **B,** Distal angle radiograph reveals asymmetry indicating the presence of an untreated mesiobuccal canal. **C,** Immediate postobturation film showing treated MB canal. **D,** Fourteen-month postoperative view. The

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patient was asymptomatic.

Fig. 10.8 **A,** Preoperative radiograph of symptomatic tooth #3. **B,** Sagittal slice of tooth #3 showing periodontal ligament thickening and associated sinus mucosal thickening. **C,** Axial slice showing untreated MB2 canal *(arrow)*. **D,** Treated case.

Fig. 10.9 **A,** Preoperative radiograph showing suspected small area of resorption associated with an endodontically treated tooth filled with a silver cone. **B,** Axial slice showing how beam hardening artifact from the metallic root filling obscures the image. **C,** Sagittal view showing very large palatally oriented external root resorption. The prognosis for retention of this tooth was poor and the patient elected to extract it.

Fig. 10.10 **A,** Radiograph indicating presence of asymptomatic persistent apical periodontitis 7 years after initial treatment. The patient elected no treatment at that time. **B,** Six-year follow-up. Lesion has enlarged and the tooth has become symptomatic.

Fig. 10.11 **A,** Classic peri-implantitis. The implant needed removal. **B,** Another peri-implantitis. Note the endodontically treated root tip apical to the implant that may have contributed to the persistent disease. Perhaps apicoectomy should have been performed.

Fig. 10.12 **A,** Preoperative film showing deep caries approaching the pulp. The patient's holistic dentist advised extraction and replacement rather than endodontic therapy to retain the tooth. **B,** Fixed partial denture fabrication procedures resulted in irreversible pulpitis on both abutments requiring endodontic therapy.

Fig. 10.13 **A,** Deep caries approaching the furcation and the biologic width. Necessary crown-lengthening surgery would open the furcation to bacterial invasion and persistent periodontal disease. **B,** Distal root vertical fracture resulting in a split root. **C,** Severe caries and post perforation. Inadequate root structure remaining to restore. **D,** Multiple distal root perforations so weaken the root as to make it nonrestorable. Note: Cases *A*, *B*, and *D* could have resective endodontic surgery such as hemisection, but long-term prognosis is poorer than for extraction and replacement.[35,142]

Fig. 10.14 **A,** Posttreatment disease following apical surgery. Off-center positioning of the root filling indicates the presence of a second, untreated canal. **B,** One-year following nonsurgical retreatment showing complete

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healing.

Fig. 10.15 **A,** J-shaped radiolucency possibly indicating root fracture. **B,** Exploratory surgery confirms presence of vertical root fracture.

Fig. 10.16 **A,** Limited visibility and access with crown present. **B,** Enhanced visibility and access with crown off. Note: isolation achieved by using a Silker-Glickman clamp and sealing putty.

Fig. 10.17 **A,** KY Pliers (GC America, Alsip, IL) and supplied emery powder. **B,** Roydent Bridge Remover (Roydent, Rochester Hills, MI). **C,** CoronaFlex Kit (KaVo, Lake Zurich, IL). **D,** *Top,* Crown-A-Matic (Peerless International, South Easton, MA). *Bottom,* Morrell Crown Remover (Henry Schein, Port Washington, NY) with interchangeable tips. **E,** Tooth inadvertently extracted using a crown/bridge remover. Endodontic therapy was performed in hand and the tooth was replanted; a procedure known as unintentional replantation. **F,** Kline Crown Remover (Brasseler, Savannah, GA).

Fig. 10.18 **A,** Richwil Crown and Bridge Remover (Almore, Portland, OR). **B,** Using hot water to soften the material. **C,** The remover is placed on the restoration to be removed and the patient bites into the material. **D,** Image showing the removed crown adhering to the material.

Fig. 10.19 **A,** Relative radiopacities of post materials: left to right—stainless steel, fiber post, titanium post, gutta-percha. **B,** Diagrammatic representation of post types: *1,* custom cast, *2,* tapered, *3,* parallel, *4,* active, *5,* passive/metal, *6,* passive/nonmetal.

Fig. 10.20 **A,** Broken post (incisal view before excavation). **B,** Root has been so thinned and weakened by excavation procedures that restorability is questionable.

Fig. 10.21 **A,** Radiograph of fractured post. **B,** Fractured post, labial view. **C,** Ultrasonic troughing. **D,** Post removed by ultrasonic alone. **E,** Check film confirming complete post removal.

Fig. 10.22 **A,** Radiograph of fractured post. **B,** Roto-Pro Kit. **C,** Roto-Pro Bur. **D,** Post removed by vibration of the instrument alone.

Fig. 10.23 Gonon post removal technique. **A,** Fractured post in a lower incisor. **B,** Tooth isolated with a rubber dam. **C,** Gonon Kit. **D,** Ultrasonic exposure of the post. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Extraction device tapping a thread onto the post. Note the three bumpers needed to protect the tooth from

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the vice. **H,** Vice applied. Turning the screw on the vice opens the jaws creating the extraction force. **I,** Post removed.

Fig. 10.24 Thomas screw post removal technique. **A,** Broken screw post. **B,** Head of post being contoured to a roughly cylindrical shape. **C** and **D,** Thomas Post Removal Kit. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Application of counterclockwise rotational force using the wrench. **H,** Post removed.

Fig. 10.25 **A,** Perforated post requiring removal. **B** and **C,** Ruddle post removal kit. **D,** Post removed and perforation repaired.

Fig. 10.26 **A,** Eggler Post Remover. **B,** Post has been contoured with a highspeed bur. **C,** Eggler Post Remover grasping the post. **D,** Elevating the post.

Fig. 10.27 Methylene blue dye applied to cosmetic materials in the canal orifice. The dye may outline or penetrate the materials/fiber posts but will allow more conservative removal of the obstruction preserving dentin. If no dye penetration occurs at all, the post may be made of ceramic or zirconia requiring a different removal strategy. **A,** Composite outlined. **B,** Composite penetrated. **C,** Fiber post outlined. **D,** Fiber post penetrated.

Fig. 10.28 GyroTip technique. **A,** Broken fiber post in an extracted tooth. **B,** Radiograph of test tooth with post in place. **C,** Creating a pilot hole. **D,** GyroTip instrument. **E,** GyroTip cutting through the fiber post. Note alignment with long axis of post. **F** and **G,** Post removed. **H,** Clinical case showing fiber post perforation into furcation area. **I,** Post removed with the GyroTip. **J,** One-year follow-up of MTA repair.

Fig. 10.29 Tissue damage from heat generated by ultrasonic application to a post during removal. The ultrasonic tip was applied to the post for no more than 5 minutes at high power with the assistant applying a constant water spray. **A,** Preoperative radiograph. **B** and **C,** These images were taken 1 month after the retreatment. Note sloughing bone visible on *C*. The tooth was lost 1 month later.

Fig. 10.30 Accumulation of materials removed from retreated teeth in a 3-month period.

Fig. 10.31 **A,** Touch ‘n Heat instrument. **B,** Gutta-percha adhering to the Touch ‘n Heat tip as it cools.

Fig. 10.32 **A,** Chloroform. **B,** Eucalyptol. **C,** Halothane. **D,** Rectified Turpentine. **E,** Xylenes.

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Fig. 10.33 C+ files. These rigid instruments remove gutta-percha more efficiently than more flexible types of K-files.
Fig. 10.34 Removal of overextended gutta-percha. **A,** Preoperative radiograph showing overextended filling material. **B,** A small Hedstrom file pierces the overextended material and retrieves it. **C,** Eighteen-month reevaluation. The tooth is asymptomatic.
Fig. 10.35 Nickel-titanium rotary profile thermoplasticizing and removing gutta-percha. Optimum rotary speed is 1500 rpm.
Fig. 10.36 Rotary gutta-percha removal instruments. **A,** ProTaper Universal Package. **B,** ProTaper Universal retreatment file has a cutting tip for enhanced penetration of the root filling materials.
Fig. 10.37 Canal conforming files for enhanced cleaning of irregular and ovoid canals. **A,** CT slice on axial plane showing the ovoid nature of canal systems. **B,** TruShape package. **C,** XP System package. **D,** TruShape instrument *(top)* and XP Shaper Instrument *(bottom)*. **E,** XP Shaper instrument removed more gutta percha after the canal had already been retreated, cleaned, and shaped with traditional tapered instruments.
Fig. 10.38 Epiphany Obturation System using Resilon material.
Fig. 10.39 Endosolv-E *(left)* and Endosolv-R *(right)*.
Fig. 10.40 Comparison of radiographic appearances of three different obturating materials. **A,** Gutta-percha. **B,** Stainless steel Thermafil carrier (note subtle fluting effect in the fill). **C,** Plastic Thermafil carrier.
Fig. 10.41 **A,** Steiglitz forceps in 45- and 90-degree head angles. **B,** Tips of the Steiglitz forceps ground to a thinner contour to create the “modified” instrument. This allows deeper penetration into the tooth to enhance removal of obstructions.
Fig. 10.42 Metal carrier retreatment. **A,** Preoperative radiograph. **B,** Metal carriers exposed by careful excavation of gutta percha. **C,** Use of the Touch ‘n Heat instrument to heat the carriers and soften the gutta-percha. This allowed removal of one of the carriers using modified Steiglitz forceps. The other could not be removed using heat or solvents. **D,** Ultrasonic troughing around the carrier to facilitate grasping it with forceps. **E** and **F,** Carriers removed and confirmed with a radiograph. **G,** Metal carriers showing gutta-percha still adhering to them. **H,** Final obturation of the tooth.
Fig. 10.43 Plastic carrier retreatment. **A,** Preoperative radiograph. At this stage, the nature of the root filling is unknown. **B,** Plastic carriers visible in

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the access as two black spots in the gutta percha mass. **C,** Gutta percha in the chamber is carefully removed from the carriers. **D,** Carrier is exposed. **E,** Chloroform solvent is placed into the chamber and a small file is worked alongside the carriers to remove the gutta percha. **F** and **G,** A Hedstrom file is gently screwed into the canal alongside the carrier and withdraws it on removal. **H,** A hemostat removes the other carrier. **I,** Plastic carriers removed. **J,** Final obturation with Resilon and Epiphany sealer.

Fig. 10.44 Plastic Thermafil carrier adhering to a System B heat plugger.

Fig. 10.45 Two different solid core carriers in an endodontic access preparation upon initial exposure. GuttaCore is on the left and a plastic carrier Thermafil Plus is on the right. Note the lighter grey color of the new modified gutta-percha core compared to the plastic carrier.

Fig. 10.46 Example of poor length control with paste root fillings. **A,** Overextended paste filling into the inferior alveolar canal. **B,** Overextended paste filling into the mental foramen. **C,** Overextended paste filling extending through a perforation in an upper central incisor. **D,** Clinical appearance of the case in *C*. Note the material extending out through a sinus tract.

Fig. 10.47 **A,** Preoperative radiograph of a hard paste root filling exhibiting a short fill, inadequate seal, and periapical radiolucency. Note the proximity of the inferior alveolar canal. **B,** Ultrasonic files like this were used to break up the hard paste in the apical third of the canal allowing removal. **C,** Seventeen-month postoperative follow-up. The patient is asymptomatic and has no paresthesia.

Fig. 10.48 **A,** Endocal 10 (formerly known as Biocalyx). **B,** Split root in a case filled with Endocal 10.

Fig. 10.49 **A,** Persistent disease in a silver point filled tooth. **B,** Silver point removed. Note the radiopaque material in the apical portion of the canal system. This represents corrosion products remaining in the canal and a possible separated apical segment of the cone. **C,** Removed silver point showing black corrosion products adhering to the apical one half. **D,** Crown-down instrumentation prevents extrusion of most of the corrosion products into the periradicular tissues.

Fig. 10.50 **A,** Preoperative view of a silver point case with persistent disease. Note that the periapical radiolucency extends coronally on the distal aspect of the root end indicating the presence of an unfilled lateral canal. **B,** Postobturation radiograph showing the cleaned and filled distal canal branch.

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Fig. 10.51 Removal of a highly retentive silver point using a needle driver to squeeze the tips of the Steiglitz forceps. This applies increased gripping force to aid in removal.

Fig. 10.52 Application of indirect ultrasonic energy to a silver point by placing the ultrasonic tip against forceps that are holding the silver point.

Fig. 10.53 **A,** Diagram illustrating the braiding of Hedstrom files around a silver point. By twisting the braided files, a gripping force is applied which aids in removal of the obstruction. **B,** Small files being braided around a silver point. **C,** Pulling coronally with the braided files removes the silver point.

Fig. 10.54 Twist-off silver point case. **A,** Preoperative radiograph showing apical periodontitis and a split silver cone ("twist-off") obturation technique. **B,** The cone was initially bypassed but could not be loosened. **C,** The braided Hedstrom file technique was attempted but was unsuccessful. **D,** A Brasseler Endo Extractor tube is cemented to the cone with cyanoacrylate cement. This instrument is no longer on the market. **E,** The silver point is removed. **F,** Immediate postobturation radiograph. **G,** One-year follow-up showing apical healing.

Fig. 10.55 **A,** Meisinger Trephine Burs. **B,** Masserann kit. **C,** Roydent Extractor System. **D,** Separated Instrument Retrieval System (SIR). **E,** Instrument Removal System *(IRS)*.

Fig. 10.56 Case illustrating healing despite inability to remove a separated silver point. **A,** Preoperative radiograph showing persistent disease in an upper premolar and molar. **B,** Working film showing the separated cone that could not be retrieved despite extensive clinical efforts. **C,** Final obturation after two appointment procedure using a calcium hydroxide interappointment medicament. **D,** Four-year follow-up showing apical healing.

Fig. 10.57 If silver point retreatment is unsuccessful, then apical surgery may be needed. Note the silver point visible on the resected root end. If the point cannot be pulled out in a retrograde direction, then ultrasonic root-end preparation may be complicated by its presence, and root-end preparation using rotary burs may be necessary.

Fig. 10.58 **A,** Preoperative radiograph of a tooth with symptomatic posttreatment disease. **B,** Although not readily apparent on the preoperative film, there is a separated nickel-titanium instrument in the distal canal. **C,** Check film showing that ultrasonics has removed the separated file. **D,**

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Thirteen-month recall film. The patient was asymptomatic.
Fig. 10.59 Complicated canal anatomy can increase stress on rotary instruments leading to separation such as in this S-Shaped canal.
Fig. 10.60 The surgical operating microscope is not only invaluable in helping to remove separated instruments; it is in fact a necessary tool for these procedures.
Fig. 10.61 **A,** Separated instrument in the mesiobuccal canal of a molar. **B,** Unmodified Gates-Glidden drill. **C,** Modified instrument. The tip has been ground off to the maximum diameter of the cutting head. **D,** Staging platform created in the straight coronal section of the canal. Note the enhanced visibility and the triangular cross section of this rotary instrument.
Fig. 10.62 **A,** Cotton pellets protecting the orifices of the other canals when ultrasonic obstruction removal is needed. **B,** Ultrasonic tip separated during excavation around separated instrument. If the adjacent canals were not protected, further unnecessary complication could result.
Fig. 10.63 **A,** Preoperative radiograph showing a separated file in the palatal canal, potential coronal leakage, and apical periodontitis. **B,** Check film showing the separated instrument after gutta-percha removal. **C,** Photograph showing the separated instrument in the palatal canal and a paper point in the buccal canal to protect it. **D,** Separated nickel-titanium file removed. Note that it is in two pieces; a result typical when applying ultrasonic energy to nickel-titanium. **E,** Check film showing that the file has been completely removed. **F,** Final canal obturation.
Fig. 10.64 Tube and Hedstrom file removal technique. The tube is slipped over the obstruction and a Hedstrom file is gently screwed into the space between the tube and the obstruction. Pulling the tube and Hedstrom file together can withdraw the obstruction.
Fig. 10.65 **A,** Diagram illustrating the wire loop and tube method of obstruction removal. The wire loop is carefully placed around the obstruction, tightened, and then removed. **B,** Larger diameter tubes and smaller diameter 0.11 mm ligature wire enhances the efficiency of this technique.
Fig. 10.66 **A,** Separated file wedged into an upper incisor. **B,** Brasseler Endo Extractor tubes (no longer available). **C,** Cyanoacrylate cement and de-bonding agent. **D,** Separated file pulled out by the bonded tube. **E,** Final obturation. Note the excessive amount of tooth structure removal by the

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trephine bur that was needed to bond the tube.
Fig. 10.67 Close-up view of the Roydent Extractor tip. The tip is placed over the separated instrument and tightened to grasp the obstruction.
Fig. 10.68 **A,** Preoperative radiograph showing two separated instruments in one tooth. **B,** IRS instrument with a removed file. **C,** Note that large size files are difficult to push through the cutout window. **D,** Postoperative radiograph.
Fig. 10.69 The Terauchi File Retrieval Kit. This kit was designed to distill down all the techniques mentioned in the text into a simple to use system for separated file removal.
Fig. 10.70 Separated file removal using the GentleWave System. **A,** A small fragment of file is lodged at the apical extent of the canal. **B,** After one irrigation cycle of the GentleWave device, a check radiograph shows that the separated instrument has been removed. **C,** Postobturation image.
Fig. 10.71 **A,** Diagrammatic representation of a canal block. Fibrotic and/or calcified pulp and debris that is potentially infected remains in the apical segment of the canal when the canal is instrumented short of the apical constriction. **B,** Preoperative radiograph showing obturation short of ideal length. The patient was symptomatic and the canals were blocked. **C,** Three months posttreatment. Treatment took a total of 3.5 hours over three appointments due to the time-consuming nature of bypassing blocked canals.
Fig. 10.72 **A,** Diagrammatic representation of a ledged canal. Potentially infected debris remaining in the apical segment can result in posttreatment disease. **B,** Attempting to bypass the ledge with a small file having a 45-degree bend in the tip. Note that the opening to the apical canal segment is on the inside of the canal curvature and coronal to the level of the ledge.
Fig. 10.73 **A,** Preoperative radiograph showing a distal canal ledge with a small amount of sealer that has entered the apical segment. The ledge prevented proper cleaning and sealing of the canal system resulting in posttreatment disease. **B,** The ledge has been bypassed. The attempt is made to blend the ledge into the contour of the prepared canal wall. **C,** Final obturation showing the filled ledge and apical segment. **D,** Thirteen-month recall showing healing. The patient was then directed to have a definitive coronal restoration placed.
Fig. 10.74 **A,** Endobender Pliers (Kerr Corporation) used to overbend a nickel-titanium hand GT file. **B,** GT hand file can hold a bend to allow it to bypass ledges.

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Fig. 10.75 **A,** Preoperative radiograph showing mesial canal blockages and potential distal root ledging with accompanying posttreatment disease. **B,** Final film showing mesial blockages bypassed but inability to negotiate beyond the distal ledge. The patient elected to pursue no further treatment at this time. **C,** One-year recall. Despite not achieving all the aims of conventional endodontic therapy, periradicular healing is apparent. The patient is asymptomatic and will now begin the final restoration with the knowledge that apical surgery may be needed in the future.

Fig. 10.76 Frequently missed canals that may result in posttreatment disease. **A,** Missed second mesiobuccal canal (MB2) in an upper molar. **B,** Final obturation showing cleaned, shaped, and filled MB2 canal. **C,** Missed lingual canal on a lower incisor results in posttreatment disease. **D,** Immediate postoperative film showing management of the missed canal.

Fig. 10.77 Furcal post perforation resulting in persistent infection and furcal bone loss.

Fig. 10.78 **A,** Mesially angulated preoperative radiograph showing a palatally oriented post perforation in an upper incisor. **B,** An 8 mm narrow-based probing defect on the mesiopalatal corner of the tooth. **C,** Following coronal disassembly, the true canal can be seen lying in a facial direction relative to the palatal post preparation. **D,** The perforation was repaired with an external matrix of Colla-Cote and MTA. Subsequently, in conjunction with a periodontist, periodontal flap surgery was used to remove periodontal disease etiology from the long-standing pocket, and guided tissue regeneration procedures were instituted. **E,** Three-year reevaluation. The tooth is asymptomatic and mesiopalatal probing depth is 4 mm.

Fig. 10.79 Bioceramics are the materials of choice for perforation repair. **A,** ProRoot MTA (Dentsply Endodontics) is a medical grade of Portland cement that has had the arsenic removed so that it can be used in the human body. **B,** Brasseler Endosequence Root Repair Material (Brasseler USA). This material has better handling characteristics than MTA and minimizes discoloration of the tooth. **C**, Biodentine (Septodont) is another bioceramic that minimizes tooth discoloration.

Fig. 10.80 Geristore Kit. This and other resin-ionomers have been advocated for cervical perforation repair due to good biocompatibility and the shorter more controlled setting times that make them useful for transgingival root fillings.

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Fig. 10.81 **A,** Large furcal perforation created during an attempt at endodontic access. **B,** Colla-Cote (Integra Life Sciences, Plainsboro, NJ) to be used as an external matrix material to recreate the external root contour. **C,** Canals have been found, preliminarily instrumented, and the external matrix has been placed. **D,** Canals are protected from being blocked using large endodontic files cut off above the orifice. MTA has been placed into the defect. **E,** Radiograph showing the initial repair with MTA recreating the vault of the furcation. **F,** On the second appointment, the blocking files are removed with difficulty since the MTA has flowed into the flutes and set. At this time, endodontic therapy is completed as normal. **G,** Postobturation radiograph. Note the radiolucency in the furcal vault, which represents the Colla-Cote external matrix material. **H,** Nineteen-month reevaluation. The patient is asymptomatic and there is evidence of healing in the furcal vault area.

Fig. 10.82 Mid-root level perforation repair. **A,** Preoperative film showing mesial strip perforation with bone loss. **B,** Nonsurgical internal repair with inability to negotiate canals to the apical terminus and MTA overextension into the furca. **C,** Apical and perforation repair surgery performed. **D,** One-year follow-up showing complete healing.

Fig. 10.83 **A,** This patient was in extreme pain following initial endodontic instrumentation by her dentist. Mid-root level perforation found on access. **B,** The original canal was found and protected with an endodontic file. MTA was vibrated using ultrasonic energy applied to the file and it flowed into the defect. Then the file is moved in a push-pull manner to dislodge it from the MTA prior to closure. Note that the defect was intentionally enlarged to allow for more predictable application of the MTA. **C,** On the second appointment, the file is withdrawn easily since it was detached from the MTA repair material. Now the endodontic therapy is concluded as normal. The patient has been asymptomatic since the end of the first appointment. **D,** Twenty-seven–month follow-up showing complete healing.

Fig. 10.84 **A,** Preoperative radiograph showing mid-root level post perforation and associated periradicular periodontitis. **B,** The crown was removed and ultrasonic energy was applied to the post. **C,** Using the trephine bur from the Ruddle Post Removal kit to mill the end of the post. **D,** The screw post is removed using the wrench from the Ruddle kit. **E,** After removal of the post and gutta-percha, a plastic solid core carrier is found in

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the canal and removed using the techniques described in this chapter. **F,** Postoperative radiograph showing MTA perforation repair, canal seal with gutta-percha, and post and core fabrication. **G,** Thirteen-month recall showing healing around the perforation repair site.

Fig. 10.85 **A** and **B,** Preoperative images showing posttreatment disease in the upper left central incisor and a large custom cast post. The patient elected to leave the post and perform surgery rather than risk damage to his new crown. **C,** Submarginal rectangular flap design. **D,** Three-week follow-up showing excellent soft-tissue healing. **E,** Eighteen-month follow-up showing excellent healing of the periradicular tissues.

Fig. 10.86 **A,** This lower second molar became symptomatic many years after nonsurgical treatment. Nonsurgical retreatment had a guarded prognosis due to the large multiroot cast custom post and core. Surgery was precluded by the poor access and proximity of the inferior alveolar canal. **B,** Ultrasonic root end preparation is made in the extracted tooth. **C,** A white MTA retrograde filling was placed. Note the C-shape. **D,** Immediate post-implantation radiograph. **E,** Seven-month reevaluation showing apical healing. The patient was asymptomatic.

Fig. 11.1 Previously treated maxillary lateral incisor with persistent periradicular disease. Nonsurgical retreatment is possible but would involve disassembly of an otherwise adequate coronal restoration. Periradicular surgery is a reasonable option.

Fig. 11.2 **A,** Previously treated maxillary first molar with persistent periradicular disease and obvious transportation of the main mesiobuccal (MB-1) canal. Nonsurgical retreatment is unlikely to correct this iatrogenic condition, and surgery is the preferred choice. **B,** Immediate postsurgery radiograph. The root-end preparation and fill was extended from the MB-1 canal in a palatal direction to include the isthmus area and the second (MB-2) canal. **C,** One-year follow-up examination: the tooth is asymptomatic, and periradicular healing is apparent radiographically. Although surgery was the first-choice treatment in this case and the outcome was favorable, a good argument could be made for nonsurgical retreatment before surgery to ensure disinfection of the canal and to attempt to locate a MB-2 canal.

Fig. 11.3 **A,** Indication for surgery (biopsy): large radiolucent lesion in the area of the maxillary left central and lateral incisors was detected on routine radiographic examination. All anterior teeth responded within normal limits

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to pulp vitality testing. **B,** After administration of a local anesthetic but before surgery, the lesion was aspirated with a large-gauge needle to rule out a vascular lesion. **C,** Buccal and palatal flaps were reflected. The lesion was directly accessible from the palate. An excisional biopsy was performed, and the sample was submitted for evaluation. **D,** Light microscopic section of the biopsy specimen; the lesion was diagnosed as a nasopalatine duct cyst (×400).

Fig. 11.4 Exploratory surgery was indicated to rule out or confirm a root fracture. Magnification and staining with methylene blue dye confirmed the presence of multiple root fractures. The tooth was subsequently extracted.

Fig. 11.5 Lateral view of the skull showing the anterior nasal spine and the proximity of the maxillary anterior root apices to the floor of the nose *(red arrow)*, as well as the typical location of the mental foramen *(black arrow)*.

Fig. 11.6 Preoperative evaluation of a mandibular left second premolar included a panoramic radiograph to help locate the mental foramen *(white arrow)*, which was not visible on a standard periapical radiograph.

Fig. 11.7 Palatal view showing the position of the greater palatine foramen *(arrows)*. The approximate location of the anterior palatine artery is marked in *red*.

Fig. 11.8 **A,** Radiograph demonstrating periradicular radiolucency associated with previously treated tooth #9. **B–C,** After full-thickness flap reflection, the nasopalatine neurovascular bundle was identified (**B**), protected, and isolated (*arrow* in **C**) during curettage of cystic lesion.

Fig. 11.9 **A,** Periapical radiograph with gutta-percha tracing the sinus tract to tooth #5. Periodontal probings were within normal limits. This tooth was initially referred for exploratory surgery to rule out root fracture. CBCT confirmed root fracture and the treatment plan was changed to extraction with possible bone graft and guided tissue regeneration for ridge augmentation prior to implant placement. **B,** Axial CBCT view demonstrating extent of lesion. **C,** Coronal CBCT view demonstrating root fracture *(red arrow)* and perforation of buccal cortical plate in area of root fracture but intact bone in the cervical area.

Fig. 11.10 **A,** Preoperative periapical radiograph showing large radiolucency associated with teeth #4, 5, and 6. **B,** CBCT reconstruction *(lateral view)*. Rotation of the 3D volume on a computer monitor demonstrated that intact bone was present around the apex of tooth #6 and the lesion was associated

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only with #4 and 5 (this could not be detected with multiple angled periapical radiographs). **C,** Axial CBCT view demonstrating facial-palatal extent of the lesion. **D,** Coronal CBCT view showing displacement of the sinus membrane (a finding that suggests a sinus membrane perforation will be detected during surgery). **E,** Clinical image after surgical access removal of granulation tissue, and root-end resection (note sinus perforation [*arrow*]). **F,** Placement of bone graft and Capset. **G,** Immediate postoperative radiograph. **H,** Two-year follow-up radiograph demonstrating good healing.

Fig. 11.11 **A,** Periapical radiograph demonstrating persistent periradicular pathosis following nonsurgical retreatment of tooth #19. The extension of the lesion in the vicinity of the mandibular canal and possible involvement of tooth #20 was uncertain based on interpretation of periapical and panoramic radiographs. Tooth #20 responded WNL to pulp vitality test. **B,** Coronal CBCT demonstrates that the periradicular lesion does not extend apical or lingual to the mandibular canal. **C,** Axial CBCT view demonstrating perforation of the buccal cortical plate and extension into, but not through, the furcation of #19 and presence of intact bone around #20.

Fig. 11.12 **A,** Basic tray setup for initial surgical access. Surgical instruments shown are distributed by Hu-Friedy ([HF] Chicago), CK Dental Specialities ([CKDS] Orange, CA), EIE ([EIE]), and G. Hartzell & Son ([GHS]). *Left to right* (left section of tray): Small round micromirror (CKDS); medium oval micromirror (CKDS); handle for microscalpel (CKDS); scissors (S18 [HF]); surgical suction tip (GHS). *Top to bottom* (main section of tray): Carr #1 retractor (EIE); Carr #2 retractor (EIE); TRH-1 retractor (HF); periosteal elevator (HF); Ruddle R elevator (EIE); Ruddle L elevator (EIE); Jacquette curette (SJ 34/35 [HF]); spoon curette (CL 84 [HF]); scaler (7/8 [HF]); surgical forceps (TP 5061 [HF]); mouth mirror (HF); periodontal probe (HF). **B,** Instrument tray for root-end filling and suturing. *Left to right* (left section of tray): Two Castroviejo needle holders (Roydent Dental Products, Rochester Hills, MI); Castroviejo scissors (S31 [HF]); micro–tissue forceps (TP 5042 [HF]). *Top to bottom* (main section of tray): Cement spatula (HF); Feinstein super plugger (F1L); microexplorer (CX-1 [EIE]); endoexplorer (DG-16 [EIE]); right Super-EBA Placing & Plugging instrument (MRFR [HF]); left Super-EBA Placing & Plugging instrument (MRFL [HF]); small anterior microburnisher and plugger (HF); small left microburnisher and plugger (HF); small right microburnisher and plugger (HF); medium anterior

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microburnisher and plugger (HF); medium left microburnisher and plugger (HF); medium right microburnisher and plugger (HF); large anterior microburnisher and plugger (HF); large left microburnisher and plugger (HF); large right microburnisher and plugger (HF).

Fig. 11.13 Comparison of microsurgical scalpel *(top)* to #15C surgical blade. Microsurgical scalpels are particularly useful for the intrasulcular incision and for delicate dissection of the interproximal papillae.

Fig. 11.14 **A,** Microcondensers in assorted shapes and sizes for root-end filling. **B,** The microcondenser should be selected to fit the root-end preparation.

Fig. 11.15 Comparison of standard #5 mouth mirror *(top)* to diamond-coated micromirrors (CK Dental Specialties).

Fig. 11.16 Micromirror used to inspect resected mesial root of a mandibular first molar.

Fig. 11.17 Retractors used in periradicular surgery. *Top to bottom*, EHR-1, ER-2, and ER-1 (equivalent to Carr #2 and #1 retractors) (CK Dental Specialties).

Fig. 11.18 Retractors positioned to expose the surgical site and protect adjacent soft tissues from injury. Care must be taken to rest the retractors only on bone, not on the reflected soft-tissue flap or on the neurovascular bundle as it exits the mental foramen.

Fig. 11.19 Teflon sleeve and plugger specially designed for placement of mineral trioxide aggregate (DENTSPLY Tulsa Dental Specialties).

Fig. 11.20 Messing gun–type syringe (CK Dental Specialties) can be used for placement of various root-end filling materials.

Fig. 11.21 Another delivery system designed specifically for mineral trioxide aggregate placement (Roydent). Kit includes a variety of tips for use in different areas of the mouth and a single-use Teflon plunger.

Fig. 11.22 Hard plastic block with notches of varying shapes and sizes (G. Hartzell & Son). MTA is mixed on a glass slab to the consistency of wet sand and then packed into a notch. The applicator instrument is used to transfer the preformed plug of MTA from the block to the root end.

Fig. 11.23 Placement of a root-end filling.

Fig. 11.24 Surgeon, assistant, and patient positioned for initiation of surgery. The patient should be given tinted goggles or some other form of eye protection before the procedure begins.

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Fig. 11.25 Intrasulcular incision with two vertical releasing incisions (rectangular flap).
Fig. 11.26 Papillary-based incision with one vertical releasing incision.
Fig. 11.27 Intrasulcular incision with one vertical releasing incision (triangular flap).
Fig. 11.28 Reflection of the triangular flap to expose the root-end area.
Fig. 11.29 Submarginal (Ochsenbein-Luebke) flap.
Fig. 11.30 Clinical case involving a submarginal incision and flap. **A,** Periodontal probing of the entire area was performed before this type of flap was selected and the incision started. A submarginal incision often is used in aesthetic anterior areas of the mouth where postoperative gingival recession might expose the crown margins. **B,** The incision should be at least 2 mm apical to the depth of the sulcus. **C,** With the flap reflected, osteotomy and root-end resection were performed. Methylene blue dye was placed to mark the outline of the root end and to help identify cracks or fractures before root-end cavity preparation and filling. **D,** The flap was repositioned and sutured with 5-0 Tevdek.
Fig. 11.31 Elevator placed in the vertical incision for the first step in undermining flap reflection.
Fig. 11.32 Continuation of reflection of full-thickness flap.
Fig. 11.33 Surgical handpiece with 45-degree angle head and rear air exhaust (Impact Air 45).
Fig. 11.34 **A,** Absorbable collagen (CollaCote) is a convenient, biocompatible packing material for localized hemostasis. **B,** Cotton pellets impregnated with racemic epinephrine (Racellet) also may be used for localized hemostasis.
Fig. 11.35 Cleared section of a typical single-canal root that was injected with dye to demonstrate apical accessory canals. Most of the apical ramifications can be eliminated with a 3-mm resection.
Fig. 11.36 Cleared section of a mesiobuccal (MB) root in a maxillary first molar. Root resection at the recommended 3-mm level exposes isthmus tissue connecting the MB-1 and MB-2 canals.
Fig. 11.37 Periradicular surgery circa 1990. **A,** The root end was prepared with a 45-degree bevel and rotary bur microhandpiece. Amalgam was a commonly used root-end filling material at this time. **B,** Immediate postoperative radiograph of a mandibular second premolar with amalgam

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root-end filling. Although many teeth treated this way healed successfully, newer materials and techniques described in this chapter are currently recommended.

Fig. 11.38 Perpendicular or near perpendicular root-end resection *(green line)* can be achieved with the use of microsurgical instruments and enhanced magnification and illumination.

Fig. 11.39 Diagram of a perpendicular root-end preparation and 3-mm deep cavity preparation along the long axis of the root.

Fig. 11.40 Error in root-end cavity preparation: ultrasonic preparation did not follow the long axis of the mesial root and therefore did not allow for proper sealing of this root. Healing is unlikely.

Fig. 11.41 Ultrasonic tip in use. Preparation along the long axis of the root is possible using tips designed for each area of the mouth. In this case, the tip is properly positioned for root-end cavity preparation of a maxillary first premolar but is dangerously close to the lip because of improper retraction. Heat generated by an ultrasonic tip can cause a thermal burn, which may result in scar tissue formation.

Fig. 11.42 Scanning electron microscope image of a root end prepared in vitro with an ultrasonic device at high-power setting. A distinct fracture line can be seen *(red arrow).* Root-end preparation with ultrasonic devices should be done at low power and with water coolant.

Fig. 11.43 Ultrasonic tips (Obtura Spartan) are available in a wide variety of configurations for use in different areas of the mouth. Most new tips have a special coating (zirconium nitride or diamond) that improves cutting efficiency.

Fig. 11.44 Ultrasonic tip with diamond coating and irrigation port (DENTSPLY Tulsa Dental Specialties).

Fig. 11.45 **A,** Immediate postoperative radiograph of a bonded root-end filling in a maxillary first premolar. **B,** Follow-up radiograph at 20 months showing good periradicular healing.

Fig. 11.46 **A,** Stropko syringe used to dry a root-end preparation before placement of the root-end filling material. **B,** Clinical use of an MTA delivery system (Dentsply Tulsa Dental). The device is loaded with MTA and placed over the root-end preparation. **C,** Pressing the plugger into the sleeve delivers the filling material to the root-end cavity preparation. The filling material then is compacted with microcondensers, and additional filling material is

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placed as needed.

Fig. 11.47 **A,** Preoperative radiograph (mesial angle) of a left mandibular first molar. Periradicular disease and symptoms had persisted after nonsurgical retreatment by an endodontic resident. The mesial canals were completely obstructed at the midroot level. The preoperative evaluation included three periapical radiographs, two different horizontal angulations, and one vertically positioned radiograph. **B,** Straight view preoperative radiograph. **C,** Vertical periapical view preoperative radiograph. **D,** Osteotomy and root resection perpendicular to the long axis of the root were performed; a partial bony dehiscence over the mesial root can be seen. **E,** Racellets were packed into the bony crypt to establish hemostasis. **F,** The Racellets were removed (one usually is left in the deepest part of the crypt during root-end preparation and filling), and the root end was beveled perpendicular to the long axis of the root. Methylene blue dye can be useful for identifying the root outline and locating any cracks. Ultrasonic root-end preparation was completed to a depth of 3 mm, connecting the MB and ML canals. **G,** Mineral trioxide aggregate root-end filling was placed and inspected. The bony crypt was then gently curetted to initiate bleeding and to remove any remnants of hemostatic materials. The flap was repositioned, and a radiograph was taken. **H,** An immediate postoperative radiograph confirmed the depth and density of the root-end filling and the absence of any foreign objects. Note that calcium sulfate and bone grafting material was placed in the osseous defect and over the root because of the large buccal dehiscence, although this is not routinely required in these cases.

Fig. 11.48 **A,** Preoperative radiograph of a maxillary left central incisor showing evidence of previous surgery but no apparent root-end filling. Because of the short root length and inadequate band of keratinized gingiva, an intrasulcular incision and full-thickness triangular mucoperiosteal flap design were selected. **B,** The root was minimally resected, and the root-end cavity was prepared ultrasonically. **C,** Mineral trioxide aggregate was placed and condensed into the root-end cavity preparation. **D,** The root-end filling was inspected before the flap was repositioned and sutured. **E,** Immediate postoperative radiograph. **F,** The 6-month follow-up radiograph showed good initial periradicular healing.

Fig. 11.49 **A,** A maxillary right first molar that was sensitive to percussion and tender to palpation over the MB root. Periodontal probing revealed a

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deep, narrow bony defect on the facial aspect of the MB root. The presence of a vertical root fracture was confirmed visually using magnification and methylene blue dye. The maxillary right second premolar had been recently extracted because of a vertical root fracture. **B,** A bonded amalgam core buildup was placed in the DB and P canals, and Geristore was placed in the MB canal system. **C,** The MB root was resected, and methylene blue dye was used to help define the extent of the fracture. **D,** At the 3-year follow-up visit, a new crown had been fabricated for the molar, and the premolar had been replaced with an implant.

Fig. 11.50 Example of postoperative instructions. Written instructions provide an essential reference for the patient, because verbal instructions often are difficult to remember after surgery. The instructions may be modified as needed; it is important to provide instructions that the patient can understand. For example, the readability of these instructions is at approximately the eighth-grade level using the Flesch-Kincaid Grade Level scale.

Fig. 11.51 **A,** Preoperative periapical radiograph of tooth #19. **B,** Clinical image demonstrating periodontal defect along the facial aspect of the mesial root. **C,** Immediate postsurgical radiograph. The mesial root end was prepared with ultrasonics and filled with MTA. A mixture of DFDBA and Capse was placed for guided tissue regeneration. **D,** One-year follow-up radiograph demonstrating good healing. Periodontal probings were WNL.

Fig. 11.52 (Case 1): **A,** Periapical radiograph of tooth #3. Retreatment RCT was attempted, but MB1 and MB2 were blocked. **B,** Sagittal view demonstrating the extent of the periapical lesion. **C,** Clinical view of periodontal probing to the apex. **D,** Axial view of the apical one third demonstrating the close proximity of the periapical defect to the MB root of tooth #2 (#2 responded WNL to pulp vitality testing). **E** and **F,** Coronal views of the MB and DB/P roots, respectively.

Fig. 11.53 (Case 1, continued): **A,** A 3D reconstruction of the tooth #3 area demonstrating the furcation periodontal defect. **B,** After flap reflection, clinical view demonstrating the periodontal defect. **C,** A 3D reconstruction with the buccal plate cropped to visualize the extent of the periradicular defect. **D** and **E,** DB and P root resection, root-end preparation, root-end filling with mineral trioxide aggregate. **F,** Periradicular defect grafted with Puros allograft (Zimmer Dental, Carlsbad, CA) and CopiOs pericardium

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membrane (Zimmer Dental).

Fig. 11.54 (Case 1, continued): **A,** Axial view of immediate postoperative CBCT scan, A-1 coronal view of MB root, A-2 sagittal view and A-3 coronal view of DB and palatal roots. **B,** A 6-month recall, axial view, B-1 coronal view of MB root, B-2 sagittal view, and B-3 coronal view of DB and palatal roots. **C,** A 1-year recall, axial view, C-1 coronal view of MB root, C-2 sagittal view, and C-3 coronal view of DB and palatal roots.

Fig. 11.55 (Case 1, 1-year recall): **A** and **B,** A 3D reconstruction of 1-year recall. **C,** Clinical picture showing resolution of the periodontal defect.

Fig. 11.56 (Case 2, tooth #14): **A** and **B,** Periapical radiographs of tooth #14, mesial and distal angles, respectively. **C,** Coronal view of the MB root demonstrating a missed MB2 canal *(arrow).* **D,** Axial view showing a previous DB root amputation site *(arrow)* that was not obvious in the periapical radiograph. **E,** A 3D reconstruction demonstrating a crestal defect. **F,** Sagittal view demonstrating the crestal defect *(red arrow)* communicating with the periapical lesion *(white arrow),* elevation of the floor of the maxillary sinus but no evidence of sinus perforation.

Fig. 11.57 (Case 2, continued): **A,** Clinical view after flap reflection demonstrating both crestal and periapical lesions. **B,** Communication between both defects. **C,** Both defects grafted with EnCore Combination Allograft (Osteogenics Biomedical, Lubbock, TX). **D,** CopiOs pericardium membrane (Zimmer Dental, Carlsbad, CA). **E,** Immediate postoperative radiograph.

Fig. 11.58 (Case 3): **A,** Periapical radiograph of maxillary anterior region demonstrating a periapical lesion associated with tooth #9. **B,** CBCT reconstruction demonstrating the intact buccal cortical plate. **C,** Palatal view of the 3D reconstruction demonstrating perforation of the palatal plate. **D,** A 3D reconstruction showing the nasopalatine bundle.

Fig. 11.59 (Case 3, continued): **A,** Palatal view CBCT reconstruction showing the exit of the nasopalatine neurovascular bundle from the incisive canal. **B,** Sagittal view demonstrating the periapical radiolucency involving teeth #9, 10, and 11. **C,** Coronal view the exit of the nasopalatine bundle from incisive canal. **D,** A 3D reconstruction demonstrating the periapical lesion extension to tooth #11. **E,** Axial view demonstrating the extension of the lesion, palatal plate perforation, and relation of the nasopalatine bundle to the periapical lesion.

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Fig. 11.60 (Case 3, continued): **A** and **B,** Clinical pictures before and after flap reflection showing the intact buccal plate. **C,** Periapical defect after degranulation showing the apices of teeth #9 and 10 prior to resection. **D,** Palatal bone perforation with palatal mucosa evident *(circle).* **E** and **F,** Clinical pictures of the nasopalatine bundle intact after degranulation.
Fig. 11.61 (Case 3, continued): **A** and **B,** Lateral wall of the maxillary sinus distal to tooth #11. **C,** CopiOs membrane placed palatally to cover the palatal mucosa. **D,** CopiOs membrane covering the Puros allografting material. **E,** Through-and-through defect immediate postoperative radiograph.
Fig. 11.62 **A,** Preoperative angled radiograph of maxillary right first and second molars. Both teeth had been treated previously, and the patient has reported a history of pain in the area for the past 5 years. The treatment plan included nonsurgical retreatment followed by periradicular surgery with bone grafting and guided tissue regeneration (GTR). **B,** Preoperative straight-on radiograph of the maxillary right first and second molars. **C,** Immediate postoperative radiograph showing root-end resections and fillings. The root ends were prepared with ultrasonics, conditioned with 17% ethylenediaminetetraacetic acid (EDTA), filled with Diaket, and smoothed with a superfine diamond finishing bur. The crypt was packed with BioOss xenograft material, and a Guidor resorbable membrane was placed. **D,** Immediate postoperative radiograph (straight on view). **E,** A 4-year follow-up radiograph. The patient was asymptomatic, and all objective findings were within normal limits. The teeth were restored with porcelain fused to metal crowns.
Fig. 11.63 **A,** Preoperative radiograph of a mandibular left first molar. Gutta-percha was inserted into the buccal sulcus and traced to the apex of the distal root. Nonsurgical root canal treatment had been performed 12 months earlier. **B,** Root-end resection and MTA root-end fillings (M and D roots). **C,** Immediate postoperative radiograph. BioOss xenograft material was placed. **D,** The 19-month follow-up radiograph showed good periradicular healing.
Fig. 11.64 Intentional replantation. **A,** Preoperative radiograph of a mandibular left second molar. The tooth was persistently sensitive to percussion and biting after nonsurgical retreatment. **B,** Radiograph of the tooth immediately after extraction, root-end preparation and filling, and replantation. **C,** At the 1-year follow-up visit, the tooth was asymptomatic and showed good periradicular healing.

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Fig. 11.65 Postoperative ecchymosis can be alarming to the patient but resolves spontaneously within 7 to 14 days.

Fig. 12.1 Schematic drawing demonstrating the different stages of root development and the subsequent reduction of the apical papilla and Hertwig root sheath**.**

Fig. 12.2 Schematic drawing illustrating potential sources of postnatal stem cells in the oral environment. Cell types include tooth germ progenitor cells *(TGPCs)*, dental follicle stem cells *(DFSCs)*, salivary gland stem cells *(SGSCs)*, stem cells of the apical papilla *(SCAPs)*, dental pulp stem cells *(DPSCs)*, inflamed periapical progenitor cells *(iPAPCs)*, stem cells from human exfoliated deciduous teeth *(SHEDs)*, periodontal ligament stem cells *(PDLSCs)*, bone marrow stem cells *(BMSCs)*, and, as illustrated in the inset, oral epithelial stem cells *(OESCs)*, gingiva-derived mesenchymal stem cells *(GMSCs)*, and periosteal stem cells *(PSCs)*.

Fig. 12.3 Dissection of an immature permanent tooth indicating the extent of the apical papilla (**A–C**). Note that this structure is likely lacerated during the evoked bleeding step of revascularization cases and thus cells from this structure, including mesenchymal stem cells of the apical papilla (SCAPs), are likely to be delivered into the root canal space. *Arrow* in **C** denotes junction of apical papilla and dental pulp.

Fig. 12.4 Evoked-bleeding step in endodontic regenerative procedures in immature teeth with open apices leads to significant increase in expression of undifferentiated mesenchymal stem cell markers in the root canal space. Systemic blood, saline irrigation, and intracanal blood samples were collected during second visit of regenerative procedures. Real-time RT-PCR was performed by using RNA isolated from each sample as template, with validated specific primers for target genes and 18S ribosomal RNA endogenous control. Expression of mesenchymal stem cell markers CD73 and CD105 was upregulated after the evoked-bleeding step in regenerative procedures. Data were normalized to the housekeeping gene 18S levels and presented as mean ± standard deviation fold increase in relation to systemic blood levels for each gene and analyzed with one-way analysis of variance with Bonferroni post hoc test ($n = 8$; $*P < .05$; $**P < .01$; n.s., not statistically significant).

Fig. 12.5 Mesenchymal stem cells were delivered into root canal spaces during regenerative procedures in immature teeth with open apices. Cells

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collected from intracanal blood samples after the evoked-bleeding step or from systemic blood were stained with antibodies against CD105, CD73, or Stro-1 and evaluated with a laser-scanning confocal microscope. (**A**) Cells in intracanal blood samples collected after evoked-bleeding step showed expression of mesenchymal stem cell marker and CD105 (*green* in **A**), CD73 (*green* in **B**), and STRO-1 (*green* in **C**), whereas nuclei appear blue as stained with TO-PRO-3.

Fig. 12.6 Schematic illustrating the potential actions of irrigants and medicaments in the release and/or exposure of bioactive molecules sequestered in dentin *(1)* and their influences on regenerative events, including chemotaxis of apical undifferentiated cells *(A)*, odontoblast-like cell differentiation *(B)*, mineralization, angiogenesis, and neurogenesis *(2)*.

Fig. 12.7 SCAP repopulation in decellularized dental pulp scaffold. (**A**) Cell viability in repopularized human dental pulp (rHDP) at 2 weeks after cell seeding. The data are presented as mean number of viable cells and standard deviation values. ***$P < .001$ with one-way ANOVA analysis followed by post hoc Bonferroni test compared with control (b) Masson trichrome staining (a) and fluorescent staining with 4',6-diamidino-2-phenylindole (DAPI) and phalloidin at 2 weeks after cell seeding. *Arrowhead* in left side indicates repopulated cells which have spindle-like shape, those in the right side indicate odontoblastic processes (**B**). There was no statistically significance between other experimental groups. (**C**) Immunofluorescence cell staining at 8 weeks after cell seeding for dentin sialoprotein (DSP) and alkaline phosphatase, liver/bone/kidney (ALPL). Positive signals *(green)* were mainly found in the cells adjacent to dentin. (**D**) Western blot analysis at 8 weeks after cell seeding revealed elevation of ALPL expression in rHDP group. Scale bars: 50 μm in **B** and 20 μm in **C** and **D.** *C*, Control; *P1*, protocol 1; *P2*, protocol 2; *P3*, protocol 3; *p*, dental pulp; *d*, dentin; *R*, repopulation.

Fig. 12.8 Schematic drawing illustrating bioengineering using a cell homing approach. An immature premolar with a necrotic pulp and apical lesion **(A)** is disinfected **(B),** followed by placement of a biodegradable scaffold *(S)* containing growth factors and chemotactic factors *(G)* to allow progressive proliferation and migration of apical stem cells into the canal space **(C)** leading to the population of the canal space with stem cells concomitantly with vascular supply and tissue organization **(D).**

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Fig. 12.9 Regeneration of pulp tissue after autologous transplantation of mobilized dental pulp stem cells (MDPSCs) with G-CSF in pulpectomized teeth of young dogs on day 14. (**A** and **B**) Regenerated pulp tissue. (**B**) Odontoblastic cells *(black arrows)* lining to newly formed osteodentin/tubular dentin *(TD)* along with the dentin. (**C**) Immunohistochemical staining of BS-1 lectin. (**D**) Immunohistochemical staining of PGP9.5.

Fig. 12.10 An 11-year-old female was referred for evaluation of tooth #19 diagnosed with pulp necrosis and chronic apical abscess traced with a gutta-percha cone as seen in the periapical (PA) radiograph (**A**). A small-volume CBCT revealed resorption in mesial and distal roots (**B**). The canals were disinfected and medicated with Vitapex (**C**). Four weeks later, the patient was asymptomatic and tooth #19 and the sinus tract healed. The tooth was reaccessed, irrigated with 1.5% NaOCl and 17% EDTA, and bleeding evoked in all canals precurved 25 K. MTA was placed over the blood clot followed by glass ionomer and composite restoration (**D**). After 24 months, the patient was asymptomatic, the tooth responded to electric pulp tester (EPT) demonstrated complete healing as seen in PA radiograph (**E**) and small-volume CBCT (**F**).

Fig. 12.11 TAP remains in dentin, whereas most $Ca(OH)_2$ is eliminated after endodontic irrigation. Radiolabeled TAP or $Ca(OH)_2$ was placed within canals of standardized root segments and incubated for 28 days at 37°C. The canals were flushed with standardized volumes of EDTA and saline using either positive pressure with a side-vented needle (PP) or positive pressure with ultrasonic activation of irrigants (PUI). There was no difference in labeled TAP removal among groups with only approximately 20% of the medicament being removed by the irrigation protocols (**A**). In contrast, >80% of $Ca(OH)_2$ was removed, with more efficient removal observed in canals irrigated with PUI (**B**). Data are presented as the mean percentage of total radiolabeled medicament removal ± standard error of the mean. $*P < .05$ tested by the Student *t* test ($n = 12$/group).

Fig. 12.12 Dentin conditioning for 7 days with medicaments used in REPs has a profound effect on SCAP survival. Standardized dentin disks were treated for 7 days with TAP or double antibiotic paste (DAP) (concentrations of 1000 mg/mL or 1 mg/mL), $Ca(OH)_2$ (Ultracal), or sterile saline (control).

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SCAP in a Matrigel scaffold (BD Biosciences; Bedford, MA) was seeded into the lumen of the disks after the medicaments were removed and cultured for 7 days. Cell viability (survival) was determined using a luminescent assay. SCAP culture on dentin treated with TAP or DAP at the concentration of 1000 mg/mL resulted in no viable cells. Conversely, dentin conditioning with TAP or DAP at the concentration of 1 mg/mL supported cell viability with no difference from untreated dentin disks (control). Greater survival and proliferation were detected in the group treated with $Ca(OH)_2$. Data are presented as mean ± standard deviation of relative luminescence units (n = 12/group). *P < .05; ***P < .001; n.s., no statistical difference as tested by 1-way analysis of variance.

Fig. 12.13 Revascularization case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma, with a class 3 fracture in tooth #8 and a class 2 fracture in tooth #9 (**A**). The patient reported moderate to severe pain in both teeth. The teeth were isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 55 days. Upon recall, the teeth were isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in tooth #9 but not in tooth #8, where CollaCote was placed prior to mineral trioxide aggregate (MTA). The root canal systems were sealed with white MTA and a composite restoration (**B**).

Fig. 12.14 Regenerative endodontic case illustrating treatment delivered to a 13-year-old female patient with a diagnosis of pulpal necrosis secondary to caries, with an unspecified prior history of trauma (**A**). The tooth was isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 21 days. Upon recall, the tooth was isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in both teeth, and the root canal system was sealed with white mineral trioxide aggregate and a composite restoration (**B**).

Fig. 12.15 Regenerative endodontic case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma and periradicular chronic apical abscess on tooth #9 seen on preoperative periapical radiograph (**A**) with sinus track tracing with a gutta-

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percha cone **(A1),** and with CBCT (**B**). The patient was asymptomatic and has draining facial sinus tract. The tooth was treated with a regenerative procedure using double antibiotic paste (DAP), a mixture of ciprofloxacin and metronidazole for 1 month. At the second appointment, the sinus tract had resolved. Then, the tooth was isolated, and the double antibiotic paste was removed by irrigation with 20 mL 17% EDTA. Intracanal bleeding was evoked from the apical tissues using a precurved #25 hand-file extending approximately 2 mm beyond the root apex. A Collaplug barrier was placed at the mid-root level, then covered with 3-mm white MTA. Fuji II LC glass ionomer was used as a coronal seal of the MTA. The access was then restored with a composite resin and polished (**C**). At the 1-year follow-up, the patient was asymptomatic, responsive to electrical pulp test (EPT), periodontal probings were no greater than 3 mm, and the tooth exhibited grade I mobility without coronal discoloration. Importantly there was appreciable root maturation in addition to resolution of apical radiolucency seen on CBCT (**D**) and periapical radiograph (**E**).

Fig. 12.16 Regenerative endodontic therapy case illustrating treatment delivered to a 9-year-old female patient with a diagnosis of previously initiated endodontic therapy (pulpectomy) due to pain from pulpal inflammation on #29. Bilateral examination revealed presence of a talon cusp (dens evaginatus) which may have been the etiology of pulpal inflammation on #29. Radiographic examination revealed large periapical radiolucency on a periapical radiograph (**A**) and cone-beam computed tomography (CBCT) (**B**). The tooth was treated with a regenerative procedure using double antibiotic paste as an intracanal medicament for 48 days. On the second visit, the tooth was isolated, and the double antibiotic paste removed by saline irrigation followed by a final rinse with 17% EDTA. The canal was dried and bleeding was evoked followed by placement of an internal matrix of Collaplug 3–4 mm below the CEJ. Mineral trioxide aggregate (MTA) was then placed over it and the tooth was restored with Fuji II LC and Build It. There was complete resolution of apical radiolucency on 1-month recall (**C**), appreciable root development at 5 months recall (**D**) and complete root maturation seen at 1-year recall on both periapical radiograph and CBCT (**E** and **F**, respectively). Greater root maturation is observed at 2.5 years recall periapical radiograph (**G**) and CBCT (**H** and **I**). In addition, the tooth responded to EPT on the 1- and 2.5-year recall visits.

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Fig. 12.17 Retrospective analysis of the percentage of change in dentinal wall thickness from preoperative image to postoperative image, measured at the apical third of the root (position of apical third defined in the preoperative image) in 40 control patients and in 48 patients following a revascularization procedure. *, $P < .05$ for each $P < .05$; ***, $P < .001$ versus mineral trioxide aggregate (MTA) apexification control group and NSRCT control group. $P < .05$ versus NSRCT control group only. $P < .05$ versus calcium hydroxide ($Ca[OH]_2$) and formocresol groups. $P < .05$ versus NSRCT control group only.

Fig. 12.18 Retrospective analysis of the percentage of change in root length from preoperative image to postoperative image, measured from the cementoenamel junction (CEJ) to the root apex in 40 control patients and in 48 patients following a revascularization procedure. ***$P < .001$ versus mineral trioxide aggregate (MTA) apexification control group ($n = 20$) and NSRCT control group ($n = 20$). $P < .05$ versus MTA control group only. Median values for each group are depicted by horizontal line, and individual cases are indicated by the corresponding symbol.

Fig. 12.19 Example of a revascularization case. Treatment was delivered to a 12-year-old boy who suffered trauma on tooth#9 2 years prior to the appointment. First appointment: (**A**), Clinical examination revealed a pain upon percussion and palpation. A working length file was placed into the root canal system (**B**). The tooth was slowly irrigated with 20 mL of 1.5% sodium hypochlorite (NaOCl) followed by 20 mL of saline using a Max-I-Probe needle inserted to the apical third (**C**). The canals were dried and medicated with calcium hydroxide paste (Ultracal) (**D**). The patient returned asymptomatic 1 month later. The tooth was isolated, accessed, and the medicament removed by slow irrigation with 1.5% NaOCl followed by 17% EDTA. After the canal was adequately dried, intracanal bleeding was achieved by lacerating the apical tissues (**E**). Blood clotting was observed after approximately 10 minutes **(F).** CollaPlug was placed below the CEJ (**G**) to serve as a matrix to position the white mineral trioxide aggregate coronal to the blood clot (**H**). The tooth was then sealed with a layer of Fuji IX (**I**), etched and restored with a composite (**J**). A final radiograph was taken (**K**). The tooth responded to electrical pulp tested at the 1-year recall, and demonstrated apical closure and moderate dentinal wall thickening (**L**).

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Fig. 12.20 Trilevel outcome assessment pyramid. Regenerative endodontic procedures must be assessed and evaluated in a systematic manner, acknowledging that there are different levels of possible outcomes that carry different meanings and value for patients, clinicians, and scientists. The patient-centered outcomes represent the base of this pyramid, symbolizing the fundamental importance of achieving healing, reestablishment of function, and patient satisfaction and overall well-being.

Fig. 12.21 Schematic illustrating the different possible outcomes in root development following regenerative endodontic procedures. Type I shows root development and narrowing of the apical foramen. Type II shows root development but failure of apex closure. Type III shows no root development but closure of the apical foramen. Type IV shows no developments and failure to apex closure.

Fig. 12.22 (**A–C**) Representative hematoxylin-eosin staining of longitudinal sections of a healthy mature tooth #28, tooth #29, and tooth #9. (**D**) An enlarged section from **B** demonstrating mature, secretory odontoblasts lining newly formed tubular dentin *(arrows)*. (**E**) An enlarged section from **C** showing the presence of biofilm colonies attached and penetrated into dentinal tubules of tooth #9 *(arrows)*.

Fig. 12.23 (**A–C**) Representative hematoxylin-eosin staining of sections of a healthy mature tooth #28, tooth #29, and tooth #9. (**D–F**) Immunohistochemical staining showing von Willebrand factor and podoplanin (PDPN) immunoreactivity in a healthy mature tooth #28, tooth #29 and tooth #9. (**G–I**) Immunohistochemical staining showing colocalization of calcitonin gene–related peptide (CGRP) and class III beta-tubulin (TUBB3) in a healthy mature tooth #28 and tooth #29. (I) Tooth #9 shows TUBB3 immunoreactivity but no colocalization with calcitonin gene–related peptide. (**J–L**) Immunohistochemical staining demonstrating colocalization of Schwan cell marker (S100) with TUBB3 in a healthy mature tooth #28, tooth #29, and tooth #9. (**M–O**) Immunohistochemical staining showing close proximity of blood vessels stained with von Willebrand factor and a marker for antigen-presenting cells (human leukocyte antigen–antigen D related; HLA-DR)) in a healthy mature tooth #28, tooth #29, and tooth #9. (**P–R**) Immunohistochemical staining demonstrating close proximity of blood vessels stained with von Willebrand factor and a pan-leukocytic marker (CD45) in a healthy mature tooth, tooth #29, and tooth #9.

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Arrows indicate positive expression of the various proteins.
Fig. 13.1 Morphologic zones of the mature pulp.
Fig. 13.2 Diagrammatic representation of the odontoblast layer and subodontoblastic region of the pulp.
Fig. 13.3 **A,** Electron micrograph of a mouse molar odontoblast demonstrating gap junctions *(arrows)*, nucleus *(N)*, mitochondria *(M)*, Golgi complex *(G)*, and rough endoplasmic reticulum *(RER)*. **B,** High magnification of a section fixed and stained with lanthanum nitrate to demonstrate a typical gap junction.
Fig. 13.4 Diagram of a fully differentiated odontoblast. *RER*, Rough endoplasmic reticulum.
Fig. 13.5 Confocal microscopic image showing an odontoblast with its process expressing neurokinin 2 receptor. Neurokinin 2 has affinity to all the neuropeptides of the neurokinin family. The dotted line represents the border of the predentin.
Fig. 13.6 Autoradiograph demonstrating odontoblasts and predentin in a developing rat molar 1 hour after intraperitoneal injection of ^{3}H-proline.
Fig. 13.7 Morphology of cultured pulpal cells. Many of the cells will have mesenchymal stem cell characteristics.
Fig. 13.8 Function of major histocompatibility complex *(MHC)* class II molecule-expressing cells. They act as antigen-presenting cells that are essential for the induction of helper T-cell-dependent immune responses.
Fig. 13.9 Class II antigen-expressing dendritic cells in the pulp and dentin border zone in normal human pulp, as demonstrated by immunocytochemistry. *D*, Dentin; *OB*, odontoblastic layer.
Fig. 13.10 Immunoelectron micrograph of a cell resembling a dendritic cell and a lymphocyte. They show cell-to-cell contact.
Fig. 13.11 Delicate network of pulpal collagen fibers as demonstrated by the Pearson silver impregnation method.
Fig. 13.12 Dense bundles of collagen fibers *(CF)* in the apical pulp.
Fig. 13.13 Schematic drawing illustrating the convergence of sensory information from teeth to higher brain centers. *TMJ*, Temporomandibular joint.
Fig. 13.14 Histologic section, immunohistologically stained for neuropeptide Y (NPY), shows the distribution of sympathetic nerves in the root pulp of a

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rat molar. NPY fibers are seen associated with blood vessels.

Fig. 13.15 Schematic drawing illustrating the location of A and C fibers in the dental pulp. Myelinated A fibers are located in the periphery of the pulp, penetrating the inner part of dentin. Unmyelinated C fibers are located in the deeper part of the pulp proper.

Fig. 13.16 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows distribution of sensory nerves in the apical area of a rat molar. Nerve fibers are seen associated with blood vessels and enter the dental pulp in nerve bundles.

Fig. 13.17 Electron micrograph of the apical pulp of a young canine tooth, showing in cross section myelinated nerve axons *(M)* within Schwann cells. Smaller, unmyelinated axons *(U)* are enclosed singly and in groups by Schwann cells.

Fig. 13.18 Branches of a single nerve fiber may innervate more than a single tooth. Nerve fiber activation in one tooth may thus result in neurogenic inflammation in adjacent teeth innervated by the same axon. The mechanism is known as the axonal reflex. This phenomenon may also contribute to the poor localization of pain reported by patients.

Fig. 13.19 Histological sections from a human premolar labeled with the neurochemical marker protein gene product (PGP) 9.5, showing the dental pulp and surrounding dentin. **A,** Nerve fibers entering the coronal pulp *(P)* form a dense network beneath the odontoblasts *(OB)*, known as the plexus of Raschkow *(*)*. **B,** Nerves are entering the apical area and pass through the radicular pulp as nerve bundles *(arrows)*. Attached and free denticles *(dt)* are seen close to dentin in the apical area. **C** and **D,** Compared to dense peripheral branching of nerves in the coronal pulp **(C)**, the branching of nerves in the radicular pulp is sparse **(D)**.

Fig. 13.20 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows the distribution of sensory nerves in a rat molar. Nerves enter the coronal pulp in bundles and ramify in a network beneath the odontoblasts (i.e., plexus of Raschkow), before entering between the odontoblasts and the inner part of dentin.

Fig. 13.21 Unmyelinated nerve fiber *(NF)* without a Schwann cell covering located between adjacent odontoblasts *(O)* overlying pulp horn of a mouse molar tooth. Predentin *(PD)* can be seen at upper right. Within the nerve, there are longitudinally oriented fine neurofilaments, microvesicles, and

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mitochondria.

Fig. 13.22 Schematic drawing showing distribution of nerve fibers in the dentin-pulp border zone. **A,** Fibers running from the subodontoblastic plexus to the odontoblast layer. **B,** Fibers extending into the dentinal tubules in the predentin. **C,** Complex fibers that branch extensively in the predentin. **D,** Intratubular fibers extending into the dentin. *D,* Dentin; *Fb,* fibroblast; *OL,* odontoblast layer; *OP,* odontoblast process; *PD,* predentin.

Fig. 13.23 Schematic diagram of the pathway of transmission of nociceptive information from the orofacial region. The trigeminal pain system is a complex multilevel system that begins with the *detection* of tissue damaging stimuli in the periphery, the *processing* of that input at the level of the medullary spinal cord, and the final *perception* of what is felt as pain in the cerebral cortex. There is growing appreciation for the concept that once a noxious stimulus is detected in the periphery, there is the opportunity for a great deal of modification of the message prior to ultimate perception. *RF,* Reticular formation.

Fig. 13.24 Response of a single dog pulp nerve fiber to repeated hydrostatic pressure stimulation pulses. Lower solid wavy line of each recording indicates the stimulation pressure applied to the pulp. Upper line *(kPa)* is the femoral artery blood pressure curve recorded to indicate the relative changes in the pulse pressure and the heart cycle.

Fig. 13.25 The average number of impulses recorded from dentine after application of pressure stimuli to the dentine. More impulses are recorded after application of negative pressure (outward fluid flow) than after positive pressure (inward fluid flow).

Fig. 13.26 Diagram illustrating movement of fluid in the dentinal tubules resulting from the dehydrating effect of a blast of air from an air syringe.

Fig. 13.27 Scanning electron micrograph of a shallow groove *(between white arrowheads)* created in polished dentin by a dental explorer tine under a force of 30 g (30 cN). Note the partial occlusion of the tubules by smeared matrix.

Fig. 13.28 Odontoblasts *(arrows)* displaced upward into the dentinal tubules.

Fig. 13.29 **A,** Normal distribution of calcitonin gene–related peptide (CGRP)-immunoreactive sensory fibers in adult rat molar. Nerve fibers typically are unbranched in the root *(R)*, avoid interradicular dentin *(ir)*, and form many branches in coronal pulp *(C)* and dentin *(D)*. Nerve distribution is often asymmetric, with endings concentrated near the most columnar

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odontoblasts (in this case on the left side of the crown). When reparative dentin *(rd)* forms, it alters conditions so that dentinal innervation is reduced. **B,** Shallow class I cavity preparation on the cervical root of a rat molar was made 4 days earlier. Primary odontoblast *(O)* layer survived, and many new CGRP-immunoreactive terminal branches spread beneath and into the injured pulp and dentin. Terminal arbor can be seen branching *(arrowhead)* from a larger axon and growing into the injury site. Scale bar: 0.1 mm.

Fig. 13.30 NGF-mRNA is upregulated in the mesial pulp horn 6 hours after cavity preparation.

Fig. 13.31 Plastic changes in the trigeminal system. After injury, trigeminal neurons upregulate production of autonomic neuropeptides like neuropeptide Y (NPY) (**A**) and growth-associated protein 43 (GAP 43) connected to neurite outgrowth and regeneration (**B**). **C,** Confocal image showing that NPY is upregulated in injured neurons expressing GAP 43. **D,** The interstitial cells of the trigeminal tract of medullary dorsal horn express c-*fos* during chronic tooth pulp inflammation in rat.

Fig. 13.32 Schematic diagram of the perception and modulation of orofacial pain. Activation of primary afferent fibers (in this example from an inflamed maxillary molar) leads to the entry of a nociceptive signal that is conveyed across a synapse in the subnucleus caudalis of the trigeminal spinal nucleus. The second-order neuron projects to the thalamus; the information is then relayed to the cortex. A great deal of processing of nociceptive input can occur at the level of the medullary dorsal horn (MDH). The *inset* depicts a typical wide dynamic range (WDR) projection neuron and its relationship with other components of the MDH. Primary afferent fibers release the excitatory amino acid, glutamate—which binds and activates either AMPA or NMDA receptors—and substance P—which activates NK-1 receptors on the WDR neuron or excitatory interneurons. Descending fibers from the locus coeruleus *(LC)* and nucleus raphe magnus *(NRM)* secrete serotonin *(5HT)* and norepinephrine *(NE)*, respectively, which inhibit transmission. Release of γ amino butyric acid *(GABA)*, the amino acid, glycine, and endogenous opioid peptides such as met-enkephalin *(M-ENK)* also inhibit transmission of nociceptive information. Projection neurons may have autocrine or paracrine effects by the synthesis and release of prostaglandins *(PGs)* and nitric oxide *(NO)* via the action of cyclooxygenase *(COX)* and nitric oxide synthase *(NOS)*, respectively. Glial cells can modulate nociceptive processing by the

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release of cytokines such as tumor necrosis factor α *(TNF-α)* and interleukin 1 β *(IL-1β)*. The + sign indicates an excitatory action, whereas the—sign denotes an inhibitory action. *AMPA,* α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; *NK,* neurokinin; *NMDA, N*-methyl-d-aspartate; *SP,* substance P; *TGG,* trigeminal ganglion.

Fig. 13.33 Substance P–positive nerve fibers in the wall of pulpal blood vessels.

Fig. 13.34 High-power scanning electron micrograph of vascular network in the radicular pulp of a dog molar showing the configuration of the subodontoblastic terminal capillary network *(TCN)*. Venules *(VL)* and arterioles *(AL)* are indicated.

Fig. 13.35 Subodontoblastic terminal capillary network *(TCN)*, arterioles *(AL)*, and venules *(VL)* of young canine pulp. Dentin would be to the far left and the central pulp to the right. Scale bar: 100 μm.

Fig. 13.36 Blood vessels in the pulp horn fan out into the odontoblast layer.

Fig. 13.37 Postcapillary venule draining blood from subodontoblastic capillary plexus.

Fig. 13.38 Effect of antagonist infusion of h-*CGRP(8-37)* (calcitonin gene–related peptide inhibitor) and *SR* 140.33 (substance P inhibitor) on basal pulpal blood flow *(PBF)* and gingival blood flow *(GBF)*. *MAP,* Mean arterial pressure. ***, $p < 0.001$, **, $p < 0.005$, *, $p < 0.05$, as compared with control recordings before antagonist infusion.

Fig. 13.39 Immune cells in normal human pulp are immunopositive to *LYVE-1*, known as a lymphatic vessel marker. The $CD68^+/LYVE\text{-}1^+$ cells derive from the monocytic lineage of cells. Immunostaining demonstrated the lack of $LYVE\text{-}1^+$ lymphatic vessels in the pulp.

Fig. 13.40 A lymphatic vessel *(arrows)* in the mandibular canal in a rat jaw below the apex *(A)* of the molars. *ab,* Alveolar bone; *NB,* nerve bundle; *PDL,* periodontal ligament.

Fig. 13.41 Interstitial structure and pressures that govern transcapillary fluid transport. *Kf,* Capillary filtration coefficient; σ, capillary reflection coefficient for plasma proteins.

Fig. 13.42 Original simultaneous recordings of percent change in pulpal blood flow *(*ΔLDF%), interstitial fluid pressure *(IFP)*, and systemic blood pressure *(P_A* mm Hg) in a cat during electrical tooth stimulation. Note that

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when IFP is first decreasing after an initial rise, the pulpal blood flow reaches its maximal level *(arrows)*, demonstrating compression of vessels in the first phase.

Fig. 13.43 Reparative dentin *(RD)* deposited in response to a carious lesion in the dentin.

Fig. 13.44 Layer of cells forming reparative dentin. Note the decreased tubularity of reparative dentin compared with the developmental dentin above it.

Fig. 13.45 Swiss-cheese type of reparative dentin. Note the numerous areas of soft-tissue inclusion and infiltration of inflammatory cells in the pulp.

Fig. 13.46 Autoradiographs from dog molars illustrating uptake of ^{3}H-thymidine by pulp cells preparing to undergo cell division after pulpotomy and pulp capping with calcium hydroxide. **A,** Two days after pulp capping. Fibroblasts, endothelial cells, and pericytes beneath the exposure site are labeled. **B,** By the fourth day, fibroblasts *(F)* and preodontoblasts adjacent to the predentin *(PD)* are labeled, which suggests that differentiation of preodontoblasts occurred within 2 days. **C,** Six days after pulp capping, new odontoblasts are labeled, and tubular dentin is being formed. (Titrated thymidine was injected 2 days after the pulp capping procedures in [**B]** and [**C]**.)

Fig. 13.47 Diffusion of dye from the pulp into reparative dentin. Note atubular zone between reparative dentin (RD) and primary dentin on the left.

Fig. 13.48 Fibrosis of dental pulp showing replacement of pulp tissue by large collagen bundles *(CB)*.

Fig. 13.49 Immature tooth with a necrotic infected canal with apical periodontitis. The canal is disinfected with copious irrigation with sodium hypochlorite and an antibiotic paste. Seven months after treatment, the patient is asymptomatic, and the apex shows healing of the apical periodontitis and some closure of the apex.

Fig. 13.50 Pulp stone with a smooth surface and concentric laminations in the pulp of a newly erupted premolar extracted in the course of orthodontic treatment.

Fig. 13.51 Diffuse calcification near the apical foramen.

Fig. 13.52 Pulp stones occupying much of the pulp chamber.

Fig. 13.53 Rough surface form of pulp stone. Note hyalinization of collagen

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fibers.

Fig. 13.54 High-power view of a pulp stone from Fig. 13.53, showing the relationship of mineralization fronts to collagen fibers.

Fig. 13.55 **A,** Calcific metamorphosis of pulp tissue after luxation of tooth as a result of trauma. Note presence of soft-tissue inclusion. **B,** High-power view showing cementoblasts *(arrows)* lining cementum *(C)*, which has been deposited on the dentin walls.

Fig. 13.56 Anatomical structures in the apical area. **A**, Cementum apposition during life will change the apical anatomical landmarks. Number *1* denotes the radiographic apex, *2* the apical foramen (major) and *3* the apical constriction (the minor apical foramen) representing the transition between the pulpal and periodontal tissues. The line that goes through *1* and *2* is often denoted the anatomical apex. **B**, Age-related sclerosis, making the dentinal tubules impermeable to bacteria, is a progressive event starting in the apical area *(white arrow)*.

Fig. 14.1 **A,** Whitlockite crystals occlude the dentinal tubules in sclerotic dentin. **B,** Dentinal sclerosis is radiographically apparent beneath a deep class II lesion.

Fig. 14.2 Reactionary dentinogenesis *(TD)*. Note the tubular morphology and the discontinuity of the tubules at the interface of secondary and reactionary dentin. Resident odontoblasts are still present.

Fig. 14.3 Tertiary dentin. The strong stimulus of the impinging infection is cytocidal for odontoblasts. The resultant dentin is irregular with soft-tissue inclusions. Intact secondary dentin is depicted by *arrow A*, and tertiary dentin is designated by *arrow B*.

Fig. 14.4 The early pulpal response to caries is represented by a focal accumulation of chronic inflammatory cells *(arrowheads)*. Note that peripheral to the inflammation the pulpal parenchyma is relatively unaffected.

Fig. 14.5 Odontoblasts exposed to lipopolysaccharide (LPS) in an in vitro culture model express IL-8 as evidenced by immunostaining with anti-IL-8 antibodies. **A,** Control cultures not exposed to LPS (negative control). **B,** Immunostaining for IL-8 in cultures exposed to LPS.

Fig. 14.6 Dental caries stimulates the accumulation of pulpal dendritic cells in and around the odontoblastic layer.

Fig. 14.7 Innate immunity in the odontoblast layer (ODL). Bacterial components from caries activate cytokine/chemokine release from

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odontoblasts, dendritic cells, and/or macrophages via Toll-like receptors *(TLRs)*. Proinflammatory cytokines released from these cells act as autocrine and paracrine signals to amplify cytokine responses including antimicrobial peptide, cytokine, and chemokine production. The release of chemokines creates a migration gradient for immune cells to ODL while antimicrobial peptides reduce bacterial load.

Fig. 14.8 A proliferative response to caries in a young tooth, typically referred to as proliferative pulpitis, hyperplastic pulpitis, or pulp polyp.

Fig. 14.9 Histological photomicrograph of a molar with carious pulp exposure. The exposure had been capped but had failed and the patient presented with symptoms. The photomicrograph shows an area of necrosis and extensive inflammation throughout the coronal pulp.

Fig. 14.10 Scanning electron microscope (SEM) image of an exposed dentin surface of a hypersensitive area. **A,** A large proportion of dentinal tubules *(arrows)* are seen to be open. **B,** SEM image of a fractured dentinal tubule of a hypersensitive area. The lumen of the dentinal tubule is partitioned by membranous structures *(arrow)*. **C,** SEM image of exposed dentin surface of a naturally desensitized area. The lumens of dentinal tubules *(arrows)* are mostly occluded, and the surface is extremely smooth. **D**, SEM image of a fractured dentinal tubule of a naturally-desensitized area. Rhombohedral platelike crystals of from 0.1 to 0.3 μm *(arrow)* are present.

Fig. 14.11 Smear layer treated with 30% dipotassium oxalate for 2 minutes plus 3% monopotassium and monohydrogen oxalate for 2 minutes. Dentin surface is completely covered with calcium oxalate crystals. (Original magnification ×1900.)

Fig. 14.12 Effects of infiltration anesthesia (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the maxillary canine teeth of dogs. There is a drastic decrease in pulpal blood flow soon after the injection. *Arrow* indicates the time of injection. *Bars* depict standard deviation.

Fig. 14.13 Effects of ligamental injection (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the mandibular canine and premolar teeth of dogs. Injection was given in the mesial and distal sulcus of premolar teeth. Injection caused total cessation of pulpal blood flow which lasted about 30 minutes in the premolar teeth. *Arrow* indicates time of injection.

Fig. 14.14 With adequate water and air spray coolant, the same cutting tools, and a comparable remaining dentin thickness, the intensity of the pulpal

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response with high-speed techniques (i.e., decreasing force) is considerably less traumatic than with lower speed techniques (i.e., increasing force).
Fig. 14.15 Without adequate water coolant, larger cutting tools (e.g., no. 37 diamond point), create typical burn lesions within the pulp when the remaining dentin thickness becomes less than 1.5 mm.
Fig. 14.16 Burn lesion with necrosis and expanding abscess formation in a 10-day specimen. Cavity preparation dry at 20,000 rpm, with remaining dentin thickness is 0.23 mm.
Fig. 14.17 Schematic of convergence of tubules toward the pulp. **A,** Periphery of the dentin. Most surface area is occupied by intertubular dentin (☆), with a few tubules surrounded by hypermineralized peritubular dentin (✪). **B,** Near the pulp, the increase in tubule diameter has occurred largely at the expense of the peritubular dentin. This substrate has high protein content. As the remaining dentin is made thinner (from A to B), the permeability of the dentin increases, because both diameter and density of dentinal tubules are increased.
Fig. 14.18 Effects of crown preparation in dogs, with and without water and air spray (at 350,000 rpm) on pulpal blood flow. Tooth preparation without water and air spray caused substantial decrease in pulpal blood flow, whereas that with water and air spray caused insignificant changes in the flow
Fig. 14.19 Transmission electron microscope (TEM) images of undemineralized specimens of resin-bonded caries-affected dentin. **A,** Stained TEM image of undemineralized specimens following the application of the self-etch ABF system to caries-affected dentin. The hybrid layer (*H,* between *arrows*) was about 3 μm thick, and the underlying un-demineralized dentin *U* was highly porous *(arrowhead)*. The dentinal tubule was covered with a smear plug *(SP)* and was partially obliterated with large caries crystals (pointer). *A,* Filled adhesive. **B,** Stained section of the total etch Single Bond adhesive bonded to caries-affected dentin. A hybrid layer *H* between 15 and 19 μm thick could be seen, with a partially demineralized zone *(open arrows)* above the undemineralized caries affected dentin *U. C*, Composite; *T*, dentinal tubule. **C,** Higher magnification of the basal part of the unusually thick hybrid layer *H* shown in *A*. Banded collagen fibrils *(open arrow)* were separated by unusually wide and porous interfibrillar spaces *(open arrowheads)*. A partially demineralized zone *(Pd)* was present along the demineralization front. This zone was not seen in phosphoric-acid-etched

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sound dentin (B). *U*, Undemineralized caries-affected dentin.

Fig. 14.20 Calcium hydroxide produces an inflammatory response that stimulates dentinal bridge formation. The dentin bridge forms lower in the tooth with calcium hydroxide paste **(A)** versus hard setting calcium hydroxide **(B)**.

Fig. 14.21 Currently available dental wavelengths on the electromagnetic spectrum. Note all of the wavelengths are nonionizing.

Fig. 14.22 **A**, Dental caries. **B**, Pulsed Nd:YAG ablation by-products (160 mJ, 10 Hz). **C**, Debris removed with acid etch and polishing. Enamel surface was faceted for reflection spectroscopy.

Fig. 14.23 **A** and **B,** Histopathologic picture of Nd:YAG laser specimen with an increasing power. There is direct relationship between the degree of pathologic changes and the increasing power of the laser. In fact, 1.5 W and the greater power cause permanent damage to the pulp.

Fig. 14.24 **A,** Control; normal odontoblastic process *(arrow)* and a few nerve terminals *(arrowheads)* are seen in a dental tubule of rat upper first molar. **B,** Six hours after Er:YAG laser irradiation; disrupted cell membrane of a nerve terminal that contains some granular vesicles *(arrow)* and shrinkage of an odontoblastic process *(arrowhead)* are noted in dentinal tubules just under the ablated area. An asterisk indicates the irradiated side. *TEM*, ×13,700; *bar*, 1 μm.

Fig. 14.25 Mandibular first molar of 49-year-old female, no pain. Clinically caries free. Calculus, periodontal disease, and bone loss from two thirds to three fourths of root. **A,** Note narrowness of pulp chamber seen throughout all serial sections (original magnification ×8.5). **B,** Bacterial plaque and adjacent dentinal tubules with bacteria (*oblique arrows* in **A**) (original magnification ×400). **C,** Farthest penetration of bacteria in dentinal tubules (*vertical arrow* in **A**) (original magnification ×400). **D,** Where dentinal tubules invaded by bacteria terminate in the pulp (*horizontal arrow* in **A**), a small but dense accumulation of lymphocytes and macrophages. *a*, Artifact; pulp tissue torn away from dentin during processing. (Original magnification ×400.)

Fig. 14.26 **A,** The postoperative change of the mean pulpal blood flow in the upper incisors of the two groups. **B,** The postoperative change in the percentages of teeth (upper incisors) with positive pulpal sensibility in the two groups. *d*, Day(s) after the operation; *M*, months after the operation; *Pre*,

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before the operation; *bars,* SD; *, $P < .05$; **, $P < .01$.

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Fig. 15.1 Drawings from Miller's classic paper showing different bacterial forms in a root canal sample observed by microscopy.
Fig. 15.2 **A,** Scanning electron micrographs of dentin showing tubules in cross-sectional view (magnification ×850). **B,** Longitudinal view (magnification ×130).
Fig. 15.3 Periodontal disease **(A)** mostly affects the pulp vitality when the subgingival biofilm reaches the apical foramen **(B)**.
Fig. 15.4 Scanning electron micrograph showing a bacterial biofilm covering dentin in a deep carious lesion. Note the presence of different bacterial morphotypes (magnification ×3500).
Fig. 15.5 Dynamics of pulp response from caries exposure **(A)** to pulp inflammation **(B)** to pulp necrosis **(C)** to apical periodontitis formation **(D)**.
Fig. 15.6 Bacterial cell and its structural components that can act as virulence factors. *Right:* A detailed scheme of the bacterial cell walls from gram-positive and gram-negative bacteria. *CM,* Cytoplasmic membrane; *LPS,* lipopolysaccharide (endotoxin); *LPtn,* lipoproteins; *LTA,* lipoteichoic acid; *OM,* outer membrane; *OMP,* outer membrane protein; *PG,* peptidoglycan.
Fig. 15.7 Mixed bacterial population colonizing the root canal wall. Cocci are the predominant forms, but rods, filaments, and spirochetes are also observed. In some areas, coccoid cells are relatively apart from each other (magnification ×2200).
Fig. 15.8 Heavy colonization of yeast cells in the root canal of an extracted tooth with primary infection associated with apical periodontitis (magnification ×300). Note that some cells are in the stage of budding. A daughter cell is growing on the surface of the mother cell (insets: magnification ×2700 [*bottom*], magnification ×3500[*top*]).
Fig. 15.9 **A,** Biofilm on the walls of a mesial root canal from a mandibular first molar (magnification ×100). The tooth was symptomatic, and an apical periodontitis lesion was present. Sections in **B** and **C** correspond to higher magnifications of the larger and smaller insets, respectively. Note the accumulation of polymorphonuclear neutrophils in the canal near the biofilm. (**B,** magnification ×400; **C,** magnification ×1000). Sections stained with a Taylor-modified Brown and Brenn technique.
Fig. 15.10 **A,** Bacterial biofilm in the necrotic canal and in an apical ramification contiguous to inflamed periradicular tissues (magnification ×25). **B,** Higher magnification of **A** (magnification ×100). Sections stained with a

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Taylor-modified Brown and Brenn technique.

Fig. 15.11 Transverse sections of a maxillary second molar with fused mesial and palatal roots. **A,** Heavy bacterial infection of the canal, spreading to an isthmus (magnification ×25). **B,** Higher magnification of the isthmus clogged with bacteria (magnification ×400). Sections stained with a Taylor-modified Brown and Brenn technique.

Fig. 15.12 Heavy infection of the root canal walls, mainly by cocci, but some small rods are also seen. Cocci are penetrating into dentinal tubules (magnification ×3500).

Fig. 15.13 Cocci in dentinal tubules approximately 300 μm from the main root canal (magnification ×5000).

Fig. 15.14 High interindividual variability in bacterial diversity (richness and abundance) at the phylum level in samples taken from cryopulverized apices of teeth with primary and posttreatment apical periodontitis. Variability is much more pronounced at the genus and species (data not shown).

Fig. 15.15 Mixed bacterial biofilm adhered to the tooth surface (Brown and Brenn staining, magnification ×1000).

Fig. 15.16 Molecular biology methods used (or with potential to be used) in the study of endodontic infections. The choice for a particular technique will depend on the type of analysis to be performed. Some methods can be used for detection of target microbial species or groups and others for a broader analysis of the microbiota**.**

Fig. 15.17 Percentage distribution of bacterial species/phylotypes found in endodontic infections according to the detection method. Data refer to the percentage of species or phylotypes overall or in each of the nine phyla that have endodontic representatives.

Fig. 15.18 Bacterial phyla with their respective endodontic representatives. *Right:* Example species or phylotypes for each phylum.

Fig. 15.19 Apical periodontitis due to primary intraradicular infection. The dental pulp is necrotic and the lesion size is usually directly proportional to the complexity of the microbiota involved.

Fig. 15.20 Prevalence of bacteria detected in primary infections of teeth with asymptomatic (chronic) apical periodontitis.

Fig. 15.21 Prevalence of bacteria detected in primary infections of teeth with symptomatic (acute) apical periodontitis.

Fig. 15.22 Prevalence of bacteria detected in primary infections of teeth with

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acute apical abscesses.

Fig. 15.23 Acute apical abscess. The infection has spread to other anatomic spaces to form cellulitis.

Fig. 15.24 Ecologic conditions in different areas of the root canal. A gradient of oxygen tension and nutrients (type and availability) is formed. Consequently, the microbiota residing in different parts can also differ in diversity, density, and accessibility to treatment procedures.

Fig. 15.25 Posttreatment apical periodontitis lesions in root canal–treated teeth. In inadequately treated canals, the microbiota is similar to primary infections. In cases apparently well treated, fewer species are found. Regardless of treatment quality, persistent or secondary intraradicular infections are the main causative agents of endodontic treatment failure.

Fig. 15.26 Prevalence of *Enterococcus faecalis* in samples from root canal–treated teeth with apical periodontitis.

Fig. 15.27 Dentinal tubule infection by *Enterococcus faecalis* in a dog's tooth after experimental infection. Notice that cells invaded the entire extent of some tubules up to the cementum (Brown and Brenn staining, ×1000).

Fig. 15.28 Prevalence of microorganisms detected in root canal–treated teeth with posttreatment disease.

Fig. 15.29 Interindividual variability in bacterial diversity (richness and abundance) at the genus levels in cryopulverized apices from teeth with posttreatment apical periodontitis. Notice that no two samples are the same in terms of bacterial composition.

Fig. 15.30 Bacterial aggregate in an epithelialized apical periodontitis lesion, suggestive of actinomycosis. This case presented a severe flare-up in the past, but the tooth was asymptomatic at the time of extraction. **A,** Section not passing through the canal; note cyst cavity and bacterial aggregation on the left (magnification ×25). **B,** Detail of area with colony (magnification ×100). **C,** Higher magnification of colony, which is surrounded by neutrophils (magnification ×400). Sections stained with a Taylor-modified Brown and Brenn technique.

Fig. 15.31 Scanning electron micrograph showing extensive bacterial colonization in the very apical part of the canal, near and at the apical foramen (**A–B,** magnification ×550 and magnification ×850, respectively). **C,** Higher magnification of left inset in **B** showing a bacterial biofilm adhered to the very apical canal walls (magnification ×3700). **D,** Higher magnification

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of right inset in **B** showing a fully developed “corncob” in tissue meshwork adjacent to the apical foramen (magnification ×4000).

Fig. 15.32 **A,** Large bacterial floc located in the center of the apical periodontitis lesion, at some distance from the apical foramen. This represents an independent extraradicular infection that was the cause of persistent disease (magnification ×16). **B,** Higher magnification of the periphery of the large colony, showing intense accumulation of inflammatory cells (magnification ×400).

Fig. 16.1 **A,** Inflammation of the pulp tissue in the apical root canal extends into the periapical tissues in a mature tooth (H&E, magnification: c100). **B,** Arrow in **A**. High magnification of the pulp tissue in the apical root canal in **A**. The pulp tissue is vital and infiltrated with chronic inflammatory cells. Note resorption of canal wall and multinucleated clast cells (H&E magnification: ×200).

Fig. 16.2 Major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis. *APC,* Antigen presenting cell; *BV,* blood vessels; *CGRP*, calcitonin gene–related peptide; *GM-CSF*, granulocyte/monocyte colony-stimulating factor; *INF*, interferon-*r*; *MC,* mast cell; *Mφ,* macrophage; *OB,* osteoblast; *OC,* osteoclast; *PAMPs*, pathogen-associated molecular patterns; *PMN*, polymorphonuclear leukocyte; *RANKL*, receptor activator of nuclear factor κB ligand; *TLR,* Toll-like receptor; *TNF*, tumor necrosis factor.

Fig. 16.3 Structure of a secondary periapical abscess. **A,** Axial section of an abscessed apical periodontitis. The microabscess *(AB)* contains a focus of neutrophils (*NG* inset in **A**). Note the phagocytosed bacteria in one of the neutrophils (further magnified in large inset in **B**). A secondary abscess forms when bacteria (*BA* in oval inset) from the apical root canal *(RC)* advance into the chronic apical periodontitis lesion (**B**). Note the tissue necrosis immediately in front of the apical foramen and the bacterial front in the body of the lesion *(arrowheads in lower inset). BV,* Blood vessels; *D,* dentin. (**A,** Magnification: ×130; **B,** magnification: ×100; oval inset, magnification: ×400; inset in **A**, magnification: ×2680; upper inset in **B**, magnification: ×4900; lower inset in **B**, magnification: ×250.)

Fig. 16.4 Bone resorption by osteoclasts in apical periodontitis. *DC,* Dendritic cell; *GM-CSF*, granulocyte/monocyte colony-stimulating factor; *HSC,* hematopoietic stem cell; *IL*, interleukin; *Mφ,* macrophage; *MSC,*

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mesenchymal stromal cell; *Nφ*, neutrophil; *OB*, osteoblast; *OC*, osteoclast; *OCP*, osteoclast precursor; *OPG*, osteoprotegerin; *PGE*, prostaglandin E; *RANK*, receptor activator of nuclear factor κB; *RANKL*, receptor activator of nuclear factor κB ligand; *TGF*-β, transforming growth factor-β; *TNF*, tumor necrosis factor.

Fig. 16.5 Schematic illustration of the major mechanisms that activate proliferation of epithelial cell rests in apical periodontitis. *EGF*, Epidermal growth factor; EO, eosinophil; *ERM*, epithelial cell rests of Malassez; *FGF*, fibroblast growth factor; *KGF*, keratinocyte growth factor; *IGF*, insulin-like growth factor; *IL*, interleukin; *Mφ*, macrophage; *OC*, osteoclast; *PMN*, polymorphonuclear leukocyte; *TGF*, transforming growth factor; *TNF*, tumor necrosis factor.

Fig. 16.6 Chronic asymptomatic apical periodontitis without **(A)** and with **(B)** epithelium *(EP)*. The root canal contains bacteria *(BA)* in **A** and **B.** The lesion in **A** has no acute inflammatory cells, even at the mouth of the root canal with visible bacteria at the apical foramen *(BA)*. Note the collagen-rich maturing granulation tissue *(GR)* infiltrated with plasma cells and lymphocytes (inset in **A** and **B**). *BV*, Blood vessels; *D*, dentin. (**A,** Magnification: ×80; **B,** magnification: ×60; inset in **A,** magnification: ×250; inset in **B,** magnification: ×400.)

Fig. 16.7 Presence of intact myelinated and nonmyelinated nerve fibers in chronic apical periodontitis lesion. *ly*, Lymphocyte; *M*, myelinated nerve fiber; *N*, nucleus of Schwann cell; *nl*, neutrophilic leukocyte; *NM*, nonmyelinated nerve fiber; *p*, plasma cell. (Electron micrograph, magnification: ×1600.)

Fig. 16.8 Bacteria phagocytosed by phagocytes in chronic apical periodontitis lesion. **A,** Bacteria *(arrows)* in phagosome of a neutrophil. *ls*, Lysosome; *N*, part of nucleus; *Pv*, phagocytic vacuole. (EM, magnification: ×6300.) **B,** Part of the cell structure of a macrophage. Note longitudinally and cross-cut bacteria cells with unit membrane *(arrows)* in phagocytic vacuoles. (EM, magnification: ×10,000).

Fig. 16.9 **A,** A well-developed pocket cyst in apical periodontitis lesion. **B,** Note the saclike epithelial lumen. **C** and **D,** Sequential axial serial section passing through the apical foramen in the root canal plane *(RC)* shows the continuity of the lumen with the root canal. *D*, Dentin; *EP*, epithelium; *LU*, Lumen. (**A** and **C,** Magnification: ×16; **B** and **D,** magnification: ×40.)

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Fig. 16.10 **A,** Two distinct cysts lined by epithelium in apical periodontitis lesion. There is no evidence of communication with the foramen in serial sections (H&E, magnification: ×25). **B,** Cyst cavity on the left side in **A.** Accumulation of foamy macrophages (H&E, magnification: ×50; inset magnification: ×1000). **C,** Lower part of cavity in **B.** Dense infiltration of neutrophilic leukocytes (H&E, magnification: ×1000). **D,** Cyst cavity on the right side in **A.** Necrotic tissue debris in the lumen; infiltration of inflammatory cells in the epithelium (H&E, magnification: ×50). **E,** Cystic wall in **D.** Cyst's lining epithelium is infiltrated with acute and chronic inflammatory cells (H&E, magnification: ×1000). **F,** Foamy macrophages, neutrophilic leukocytes, and necrotic tissue inside the cyst *(inset),* but no bacteria (Brown & Brenn, magnification: ×25, inset magnification: ×1000). **G,** Area indicated by an *open arrow* in **F.** Bacterial colonies in the apical foramen (Brown & Brenn, magnification: ×1000). *LU,* Lumen.

Fig. 16.11 **A,** A radicular cyst in which the lumen is completely filled with cholesterol crystals *(CC)* (H&E, magnification: ×25). **B,** High magnification of cholesterol crystals in **A** (H&E, magnification: ×100). **C,** High magnification of rectangular area in **B.** Multinucleated giant cells associated with cholesterol crystals (H&E, magnification: ×400). *EP*, Epithelium.

Fig. 16.12 **A,** An apical true cyst in apical periodontitis lesion. Bacterial colonies are seen in the foraminal areas (Brown & Brenn, magnification: ×25). **B,** Bacterial colonies in the cyst lumen, surrounded by inflammatory cells (Brown & Brenn, magnification: ×400).

Fig. 17.1 Histologic view of intact calcific bridge formation following pulpotomy.

Fig. 17.2 Immature maxillary right lateral and central incisors **(A)** showing continued root development following pulpotomies **(B)**.

Fig. 17.3 Low-power view of healing by cementum formation when Sealpex is used (black particles are residual Sealapex and root filling material).

Fig. 17.4 Resolution of periapical disease after root canal treatment of the mandibular left molar with preoperative periapical radiolucency associated with mesial and distal roots **(A)**, delayed healing caused by extruded filling material from the distal canal **(B)** and nearing complete healing as extruded material is resorbed **(C)**.

Fig. 17.5 Histologic view of fibrous periapical healing.

Fig. 17.6 Axial sections through the surgically removed apical portion of the

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root with a therapy-resistant periapical lesion *(GR)*. Note the cluster of bacteria *(BA)* visible in the root canal. Parts **(B)**–**(E)** show serial semithin sections taken at varying distances from the section plane of **(A)** to reveal the emerging **(B)** and gradually widening **(C–E)** profiles of an accessory root canal *(AC)*. Note that the accessory canal is clogged with bacteria *(BA)*. Original magnification: **A,** ×52; **B–E,** ×62.

Fig. 17.7 **A,** Adequate root filling demonstrated on radiograph. **B,** View of the periapical surgery and root resection of the tooth shown in **(A)** shows stained root dentine. **C,** Resected root showing stained/infected dentine. **D,** Histologic view of the root end shown in **(C)** showing infected dentinal tubules *(S)*.

Fig. 17.8 **Actinomyces in the body of a human periapical granuloma.**The actinomycotic colony *(AC)* in (**A**) is magnified in (**B**). The rectangular area demarcated in **(B)** is magnified in **(C)**. Note the starburst appearance of the colony with needle-like peripheral filaments surrounded by few layers of neutrophilic granulocytes *(NG)*, some of which contain phagocytosed bacteria. A dividing peripheral filament *(FI)* is magnified in the inset. Note the typical gram-positive wall cell wall *(CW)*. *D*, Dentine. Original magnification: **A,** ×60; **B,** ×430; **C,** ×1680; inset, ×6700.

Fig. 17.9 **A,** A maxillary right central incisor having undergone periapical surgery. **B,** Incomplete periapical healing at 1 year postoperatively. Complete healing at 3 **(C)** and 4 years **(D)** postoperatively.

Fig. 17.10 Example of incomplete healing with isolated scar tissue.

Fig. 17.11 Example of uncertain healing. Periapical radiographs of a maxillary left central incisor taken immediately after periapical surgery **(A)**, at 2 years postoperatively **(B)**, and at 3 years postoperatively **(C)**.

Fig. 17.12 Forest plot showing individual study and weighted pooled probabilities of success for one-step indirect pulp capping.

Fig. 17.13 Forest plot showing individual study and weighted pooled probabilities of success for stepwise indirect pulp capping.

Fig. 17.14 Forest plot showing individual study and weighted pooled probabilities of success for direct pulp capping.

Fig. 17.15 Forest plot showing individual study and weighted pooled probabilities of success for partial pulpotomy.

Fig. 17.16 Forest plot showing individual study and weighted pooled probabilities of success for full pulpotomy.

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Fig. 17.17 Forest plot showing results of pooled and individual study's probability of maintained periapical health for preoperatively vital teeth undergoing root canal treatment (pooled probability = 0.83; 95% confidence interval: 0.77, 0.89).

Fig. 17.18 Forest plot showing results of pooled and individual study's probability of periapical health for teeth associated with nonvital pulps and periapical radiolucencies undergoing root canal treatment (pooled probability = 0.72; 95% confidence interval: 0.67, 0.78).

Fig. 17.19 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with preobturation negative versus positive culture test results (pooled OR = 2.1; 95% CI: 1.5, 2.9).

Fig. 17.20 Forest plot showing pooled and individual study's odds ratio *(OR)* for periapical health of teeth undergoing root canal treatment with preoperative nonvital pulps and absence of periapical radiolucencies versus teeth with nonvital pulps and presence of periapical radiolucencies (pooled OR = 2.4; 95% CI: 1.7, 3.5).

Fig. 17.21 Forest plot showing pooled and individual study's odds ratio *(OR)* for periapical health of teeth undergoing root canal treatment with preoperative large (>5 mm) versus small (<5 mm) periapical radiolucencies (pooled OR = 2.2; 95% CI: 1.3, 3.7).

Fig. 17.22 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment with underextended (0.76 [0.71, 0.82]) **(A)**, flush (0.81 [0.76, 0.86]) **(B)**, or overextended (0.66 [0.56, 0.75]) **(C)** root fillings.

Fig. 17.23 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with good quality versus suboptimal quality root filling (pooled OR = 3.9; 95% CI: 2.5, 6.2).

Fig. 17.24 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with satisfactory versus unsatisfactory coronal restoration at follow-up (pooled OR = 1.9; 95% CI: 1.5, 2.5).

Fig. 17.25 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment by "decade of publication" and "criteria for success."

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Fig. 17.26 Forest plot showing pooled and individual study's probability of 2-year (pooled probability = 0.93; 95% CI: 0.90, 0.95) **(A)**, 5-year (pooled probability = 0.92; 95% CI: 0.91, 0.93) **(B)**, and 10-year (pooled probability = 0.88; 95% CI: 0.84, 0.92) **(C)** survival of teeth undergoing root canal treatment.
Fig. 17.27 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for nonmolar versus molar teeth (pooled OR = 1.4; 95% CI: 1.1, 1.6).
Fig. 17.28 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth with both mesial and distal contacts present versus absent (pooled OR = 2.6; 95% CI: 1.8, 3.7).
Fig. 17.29 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth restored with cast versus plastic restoration (pooled OR = 3.5; 95% CI: 2.6, 4.7).
Fig. 17.30 Tooth with technically deficient root canal treatment **(A)**; having undergone (**B** and **C**) root canal retreatment.
Fig. 17.31 Forest plot showing pooled and individual study's probability of complete periapical healing following root canal retreatment.
Fig. 17.32 Forest plot showing pooled and individual study's odds ratio of periapical health for teeth with preoperative periapical radiolucency undergoing primary root canal treatment versus root canal retreatment (pooled OR = 1.5; 95% CI: 1.0, 2.1).
Fig. 17.33 Forest plot showing pooled and individual study's probability of complete periapical healing following apical surgery.
Fig. 18.1 Light microscopy images of a deciduous tooth showing both physiologic and pathologic (inflammatory) resorption. **A,** Tooth 7.5 in a 9-year-old boy. Extensive exposure of a mesial pulp horn occurred during caries excavation, and pulp capping was performed. The patient was brought to the dental office 4 months after the treatment because of severe spontaneous pain. The radiograph shows radiolucencies on both apices and interradicularly. The parents did not accept any treatment and requested extraction. **B,** Longitudinal section taken on a mesiodistal plane, passing approximately at the center of the mesial root. (Hematoxylin-eosin [H&E] stain; original magnification ×25.) **C,** Detail of the resorption area apically in **B** (original magnification ×100). **D,** Magnification of the area demarcated by the rectangle in **C.** Two odontoclasts are present in an area of dentin being

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resorbed, surrounded by fibroblasts and inflammatory cells (original magnification ×400). **E,** High-power view of the area indicated by the *arrow* in **C.** An odontoclast is in close contact with the dentin in a Howship lacuna. Its cytoplasm appears vacuolated and shows a more intense staining reaction than the cytoplasm of the adjacent cells (original magnification ×1000). **F,** High-power view of the upper odontoclast in **D.** The characteristic ruffled border can be distinguished (original magnification ×1000). **G,** Howship lacuna with an odontoclast in another area (original magnification ×400). **H–J,** Progressive magnification of the mesial root apex. The resorption process may be physiologic at this level (original magnification ×16, ×100, and ×400).

Fig. 18.2 External apical inflammatory resorption. **A,** Maxillary first molar in a 46-year-old female patient that was causing severe pain. The radiographs revealed material in the pulp chamber, a large distal caries, and apical radiolucencies. The tooth was extracted. **B,** Palatal root apex extracted with the pathologic tissue attached. Note the ingrowth of granulation tissue into the foramen and the massive resorption of the apical profile. Also note the resorption of lacunae on the left radicular profile. (H&E stain; original magnification ×25.) **C,** High magnification of the area of the external radicular profile indicated by the *arrow* in **B.** A resorption lacuna can be seen in the cementum. (H&E stain; original magnification ×400.) **D,** View from the center of the apical periodontitis lesion showing severe concentration of chronic inflammatory cells (mostly plasma cells) (original magnification ×400).

Fig. 18.3 External apical inflammatory resorption. **A,** Mandibular second premolar extracted with the periapical lesion attached. This section, which did not pass through the canal, shows extensive apical resorption. (H&E stain; original magnification ×25.) **B,** Section taken approximately 120 sections away encompasses the apical foramen. In addition to resorption, the opposite phenomenon can be observed; that is, a large calcification partly embedded in the right apical dentin wall (original magnification ×25).

Fig. 18.4 External apical inflammatory resorption. **A,** Mesial root of a mandibular first molar extracted with a periapical lesion attached. This section passes through the canal and the foramen. Extensive resorption of the foramen has occurred, and a calcification can be seen more coronally. Note that the tissue in the apical canal and in the resorptive defect appears

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structured and continuous with the apical periodontitis lesion. A thick biofilm can be discerned layering the root canal walls more coronally. (Taylor's modified Brown & Brenn stain; original magnification ×25.) **B,** Palatal root of a maxillary first molar extracted with the periapical lesion attached. A cyst cavity is present in the body of the lesion. Resorption of the foramen is present, and a portion of the right dentin wall has been replaced by bone *(arrowheads).* (H&E stain; original magnification ×25.)

Fig. 18.5 Apical resorption. **A,** 22-year-old male patient complained of pain on chewing. This was caused by a molar that had been treated endodontically 1 year earlier. A radiograph showed obturation material only at the root canal orifices and a periapical radiolucency on the mesial root. Endodontic retreatment was recommended, but the patient declined this treatment. **B,** The patient returned 3 years and 3 months later because of an abscess that was causing severe pain. A radiograph disclosed destructive mesial caries and large periapical radiolucencies on both roots, along with resorption of the whole apical third of the distal root.

Fig. 18.6 External cervical resorption with replacement. **A,** Asymptomatic mandibular third molar. ECR was diagnosed on a radiograph taken for neighboring teeth. The tooth was extracted. Overview of the tooth (H&E stain; original magnification ×2). **B,** High power view of the resorptive area occupied by granulation tissue indicated by the *arrow* in **A**. Severe concentration of acute and chronic inflammatory cells (original magnification ×400). **C,** Detail of the area demarcated by the *rectangle* in **A**. The large area of resorbed dentin has been partly repaired with bonelike tissue. Note the thin layer of dentin remaining. No inflammatory changes can be observed in the adjacent pulp (original magnification ×25). **D,** Detail from **C**. Normal appearance of dentin, predentin, and odontoblast layer. Absence of inflammation (original magnification ×50).

Fig. 18.7 NanoCT imaging of tooth 13 and 3D modeling using CTan, CTvol, and CTvox software, showing the thickness distribution of the pericanalar resorption resistant sheet (PRRS).

Fig. 18.8 **A,** Structure of pericanalar resorption resistant sheet *(PRRS)* and pulp tissue. PRRS is consisted of predentin, dentin and occasionally bonelike tissue. **B,** Calcification inside the pulp. Histologic staining was performed with a combination of Stevenel's blue and Von Gieson's picrofuchsin, visualizing mineralized tissue *(red)* and nonmineralized tissue *(blue-green)*.

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Fig. 18.9 External cervical resorption (ECR). **A,** A 55-year-old female patient presented with an asymptomatic "pink spot." She had no history of any predisposing factors. **B,** A periapical radiograph revealed radiolucent defects on the proximal aspects of the upper left central incisor; note the ragged borders. **C,** A reconstructed coronal cone beam computed tomography (CBCT) slice reveals the true extent of the ECR lesion. Note that the root canal wall appears to be intact. Inhibitory factors in the root canal wall/odontoblastic prevented the ECR lesion from penetrating the root canal. **D,** A 41-year-old male patient presented as a new patient. Routine radiographic examination revealed a poorly defined periapical radiolucency in the root of the upper right central incisor. The appearance is suggestive of ECR; the patient had orthodontic treatment in his early teens and remembered "knocking the tooth" at least twice when he was very young. Reconstructed sagittal **(E)** and coronal **(F)** CBCT slices revealed the true nature of the ECR lesion and showed that the lesion was not amenable to treatment. **G,** The treatment options were discussed with the patient, and it was decided to review the tooth periodically. The 4-year radiograph shows no change in size of this asymptomatic lesion.

Fig. 18.10 **A,** This patient's upper left central incisor had a pink spot and was cavitated. **B,** A radiograph revealed an unusual presentation of ECR; the lesion is circular and has well-defined margins. Note that the outline of the root canal is visible and intact through the radiolucent lesion. **C,** The tooth was unrestorable and was extracted; note the large amount of granulation tissue. **D,** Three-dimensional reconstruction of the extracted tooth from microtomography data revealed bonelike tissue below the overlying granulation tissue. Note the intact root canal wall *(red arrow)*. **E,** Coronal reconstruction from a microtomography scan reveals how the predentin *(red arrow)* prevented the ECR defect from invading the root canal. In addition, bonelike tissue can be seen *(yellow arrow)*. Posttreatment view **(F)** and radiograph **(G)** after replacement of the upper left central incisor with an implant-retained crown.

Fig. 18.11 External cervical resorption (ECR) with replacement. **A,** Radiograph of a maxillary central incisor of a 34-year-old female patient who recalled being struck in the face with a cricket ball when she was 11 years old. The dentist mistakenly diagnosed the resorptive defect as caries and attempted to manage it accordingly. The tooth was asymptomatic; however, a

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4-mm periodontal probing depth was identified on the palatal aspect of the tooth. A cone beam computed tomography (CBCT) scan confirmed an ECR defect. The tooth was deemed unrestorable and was extracted with the patient's consent. **B,** Palatal view of the tooth at the end of the demineralization process, while immersed in the clearing agent. The tooth was separated into four portions, which were embedded separately in paraffin blocks. **C,** CBCT axial section passing through the coronal third at the level of *line 1* in **B.** The corresponding histologic section shows that most of the dentin has been replaced by a bonelike tissue. The root canal is no longer present. (H&E stain; original magnification ×8.) **D,** CBCT axial section passing through the middle third at the level of *line 2* in **B.** The root canal can be appreciated at this level, although reduced in size, but it appears to be encircled by bonelike tissue (original magnification ×8). **E,** CBCT axial section taken from the apical third at the level of *line 3* in **B.** At this level, the canal is the same size of that of the contralateral tooth. The histologic section confirmed the absence of bonelike tissue (original magnification ×8). **F,** Detail from **C** showing the transition from dentin to bonelike tissue (original magnification ×100). **G,** Higher magnification of **F.** The metaplastic tissue does not show the lamellar structure typical of bone (original magnification ×400). **H,** Detail of the canal in **D.** The metaplastic bonelike tissue has concentrically replaced a consistent portion of dentin, leaving in place a reduced layer of the original dentin (original magnification ×25). **I,** High-power view confirms that the tissue surrounding the canal is dentin (original magnification ×400). *Considerations:* The resorption process, followed by replacement with a bonelike tissue, started in the cervical area and extended in an apical direction, tunneling dentin circumferentially up to the transition between the middle and apical thirds of the tooth.

Fig. 18.12 External cervical resorption with replacement. **A,** Mandibular first and second molars in a 27-year-old female patient. The patient had symptoms of irreversible pulpitis associated with the lower right second molar; the lower right first molar was asymptomatic. **B,** CBCT scan taken through the second molar at the level of the area indicated by the *arrow* in **A.** Note massive resorption involving the crown and root. **C,** Mesial portion of the crown of the second molar after clearing. **D,** Section taken on a buccolingual plane. Overview shows resorption and replacement on the lingual side, corresponding to the area indicated by the *arrow* in **C.** (H&E stain; original

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magnification ×6.) **E,** Detail of the area indicated by the *arrow* in **C.** Several areas of resorption, with replacement by metaplastic tissue, can be seen (original magnification ×100). **F,** Magnification of the area indicated by the right *lower arrow* in **E.** The metaplastic tissue closely resembles bone (original magnification ×400). **G,** High-power view of the area indicated by the right *upper arrow* in **E.** Note the lacuna in dentin occupied by a multinucleated clastic cell (original magnification ×1000). **H,** High-power view of the metaplastic bone tissue in the area indicated by the *arrow* in **F.** A bone trabecula is being resorbed by a typical osteoclast (original magnification ×1000). **I,** High-power view of the area indicated by the left *lower arrow* in **E.** A Howship lacuna with odontoclasts can be seen (original magnification ×1000). **J,** High-power view of the area indicated by the left *upper arrow* in **E.** Note the islands of bone tissue, with typical osteocytes, surrounded by dentin (original magnification ×1000). *Considerations:* In this case the metaplastic tissue was similar to normal lamellar bone. It is interesting that the bone tissue was undergoing remodeling, as evidenced by the presence of osteoclasts. Osteoclasts and odontoclasts can be observed in the same area and show morphologic similarity.

Fig. 18.13 Early internal root resorption. **A,** A 54-year-old male patient presented with a long story of pain on chewing and with cold stimuli. Recently the pain had become continuous and severe. The maxillary first molar, which had a mesial amalgam restoration, did not respond to sensitivity tests. A radiograph showed a periapical radiolucency on the palatal root. After removal of the restorative materials, a crack line involving the pulp chamber floor was diagnosed, and the tooth was extracted. **B,** Some pathologic tissue remained attached to the palatal root apex at extraction. A longitudinal section passing at the center of the canal demonstrated vital connective tissue in the apical third. (H&E stain; original magnification ×16.) **C,** Detail of the canal in **B.** Note the connective tissue with vessels and relatively few inflammatory cells, in addition to the resorption lacunae on the root canal walls (original magnification ×1000). **D,** High-power view of the area of the canal wall indicated by the upper *arrow* in **C.** Note the resorption lacuna with odontoclasts (original magnification ×1000). **E** and **F,** Progressive magnification of the area of the canal wall indicated by the lower *arrow* in **C.** Note the Howship lacuna housing clastic cells. Predentin is present in some areas of the wall, but it is absent in the areas with active

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resorption (original magnification ×400 and ×1000).

Fig. 18.14 **A** and **B,** Parallax views of the maxillary left lateral incisor showing internal root resorption with necrosis. A gutta-percha point has been used to track the sinus. The reconstructed sagittal **(C)** and axial **(D)** slices from a CBCT scan reveal that the lesion has resorbed the palatal aspect of the root *(arrows)* and has nearly perforated the root wall. **E,** The tooth has been obturated with gutta-percha using a thermoplasticized technique.

Fig. 18.15 Light microscopy images of a tooth with internal (root canal) replacement resorption. The tooth belonged to a 44-year-old male patient who was referred to the first author for management of a perforated root. The tooth was asymptomatic on examination, but there was a history of previous trauma. **A,** Radiograph of a maxillary central incisor with a radiolucent lesion in the middle third of the root canal. The radiolucent lesion appears to be mottled, which suggests internal root resorption with metaplasia. **B,** Clinical radiograph of the tooth after extraction, taken at a 90-degree angle, showing the continuity of the resorptive lesion with the canal space. **C,** Cross section taken approximately at the level of *line 1* in *B.* The low-magnification overview shows that the dentin around the root canal had been replaced by an ingrowth of bone tissue, and the root appears to have been perforated on the distopalatal aspect. (H&E stain; ×8.) **D,** Higher magnification of *C.* (H&E stain; ×16.) **E,** High magnification of the area demarcated by the rectangle in *D.* The intraradicular dentin has been resorbed. (H&E stain; ×100.) **F,** High-magnification view taken from the right part of *C,* showing that the resorbed dentin has been replaced by lamellar bone. Osteocytes are present in lacunae between the lamellae. A characteristic cross section of an osteon can be seen on the right *(open arrows),* with concentric lamellae surrounding a vascular structure. (H&E stain; ×100.) **G,** High-magnification view of the area indicated by the left open *arrow* in *E.* A multinucleated resorbing cell (odontoclast) can be seen in a dentinal lacuna, indicating active resorption of the dentinal wall. (H&E stain; ×1000.) **H,** High-magnification view of the bone surface indicated by the right *arrow* in *E.* The large cells are osteoblast-like cells. Once they produced mineralized tissue, they were embedded in the bone lacunae, assuming the characteristics of osteocytes. (H&E stain; ×1000.) **I,** Cross section taken approximately at the level of *line 2* in *B.* The root canal is still large at this level and is surrounded by a relatively thin layer of newly formed bone. (H&E stain; ×16.) **J,** Cross section taken

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approximately at the level of *line 3* in *B.* At this level the root canal appears consistently narrowed by a dense layer of newly formed bone. (H&E stain; ×16.)

Fig. 18.16 Light microscopy images of a variant of internal (root canal) replacement resorption with tunneling resorption. The lower right lateral incisor belonged to a 39-year-old former boxer who had suffered a jaw fracture in a boxing match in his early twenties and was placed in intermaxillary fixation. The patient developed symptoms 20 years later and complained of pain associated with his lower incisors. **A,** Radiograph of the mandibular right incisors. The lower right central incisor had asymptomatic apical periodontitis associated with a necrotic and infected pulp. The lower right lateral incisor showed a large area of internal root resorption. The tooth did not respond to sensitivity tests. **B,** Sagittal CBCT slice shows some calcified tissue in the resorptive defect. **C,** Cross section taken at the level of *line 1* in *A* and *B.* The overview shows that the canal was apparently empty at this level. (H&E stain; ×6.) **D,** High magnification of the area indicated by the *arrow* in *C.* Lamellar bone filling an area of previous resorption. Note the osteon structure *(arrow).* (H&E stain; ×100.) **E,** Cross section taken at the level of line 2 in *A* and *B.* Overview shows that the canal lumen was partly occupied by necrotic remnants, partly by bonelike tissue. (H&E stain; ×8.) **F,** High magnification of the lower part in *E.* (H&E stain; ×50.) **G,** Higher magnification of *F.* Bone trabeculae are surrounded by necrotic debris. (H&E stain; ×100.) **H,** Cross section taken from the same area as that in *E.* (Taylor's modified Brown & Brenn [TBB] stain; ×16.) **I,** High magnification of the area indicated by the *arrow* in *H.* A fragment of bonelike tissue can be seen surrounded by bacteria-colonized necrotic tissues. (TBB stain; ×100; inset ×1000.) **J,** Longitudinal section passing approximately through the center of the root apex. Dentin walls have been resorbed and replaced by a bonelike tissue. (H&E stain; ×16.).

Fig. 19.1 **A,** Patient complained of acute pain on biting at the upper left molar (tooth #15). **B,** Clinical thermal tests revealed pulpal necrosis and a preoperative radiograph taken (periapical lesions noted). **C,** The tooth was split in half.

Fig. 19.2 The American Association of Endodontists *(AAE)* Endodontic Case Difficulty Assessment Form and Guidelines, developed to assist the clinician in assessing the level of difficulty of a given endodontic case and to help

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determine when referral may be necessary.

Fig. 19.3 Drainage of pus through the root canal. **A,** Acute apical abscess arising from the lower right lateral incisor with radiographic radiolucency. **B,** Initial drainage through the canal. **C,** Persistent drainage through the canal. **D,** Aspiration of the content with a plastic suction tip. **E,** Irrigation with NaOCl. **F,** Mechanical debridement. **G,** Placement of calcium hydroxide. **H,** Obturation of root canals during second visit.

Fig. 19.4 **A,** Spread of odontogenic infections. **B,** Mandibular buccal vestibule (posterior tooth). **C,** Mandibular buccal vestibule (anterior tooth). **D,** Mental space. **E,** Submental space. **F,** Sublingual space. **G,** Submandibular space. **H,** Maxillary buccal vestibule. **I,** Buccal space. **J,** Submasseteric space. **K,** Temporal space. **L,** Pterygomandibular space. **M,** Parapharyngeal spaces. **N,** Cervical spaces. **O,** Canine (infraorbital) and periorbital space.

Fig. 19.5 **A,** Preoperative radiograph. **B,** Cellulitis.

Fig. 19.6 **A,** Intraoral localized swelling. **B,** Preparation for incision. **C,** Incision. **D,** Purulence discharge.

Fig. 19.7 **A,** Iodoform gauze cut to proper length. **B,** Iodoform gauze drain after 24 hours.

Fig. 19.8 Nonvital infected tooth with active drainage from the periapical area through the canal. **A,** Access opened and draining for 1 minute. **B,** Drainage after 2 minutes. **C,** Canal space dried after 3 minutes.

Fig. 19.9 Foreign object in tooth left open to drain. Patient used a sewing needle to clear out food particles that were blocking the canal and broke the needle in the tooth.

Fig. 19.10 **A,** Preoperative radiograph. **B,** Transillumination.

Fig. 19.11 **A,** Preoperative radiograph. **B,** Clinical view. **C,** Transillumination. **D,** Extracted tooth showing the extension of the stained longitudinal fracture.

Fig. 20.1 Femur shaft cross sections (**A**) untreated bone section; (**B**) saline-treated bone; (**C**) sodium hypochlorite (NaOCl)-treated bone section. Grossly, NaOCl caused significant changes in cancellous structure, leaving large structural craters of apparent demineralization.

Fig. 20.2 **A,** Clinical presentation 3 days following the extrusion of sodium hypochlorite (NaOCl) during treatment of the maxillary right canine. The patient had difficulty opening her right eye, and the swelling had extended to

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the submandibular/sublingual regions on the affected side; she also experienced limited mouth opening. There was altered sensation infraorbitally and in the region of the upper right lip, but no reported paresthesia of the alveolar or facial nerves. **B,** One-month postextrusion, the ecchymosis had resolved, with a return of sensation and full function. Therapy was completed without incident.

Fig. 20.3 **A,** Radiograph of the maxillary left lateral incisor (#10, #22) before post removal. Radicular root resection had been completed, and the canal space is empty. Prior to recementation, the canal was irrigated with 3% NaOCl, precipitating the extrusion event. **B,** Twenty-four hours postextrusion, there was altered sensation infraorbitally and from the upper left lip to the left lip corner. In addition, the buccal branch of the facial nerve was affected, as evidenced by a distinct loss of upper lip and cheek function (the corner of the mouth could not be pulled up by the mimic musculature). The mouth opening was limited to 20 mm (**C**), 3 years postincident. The weakness of the mimic musculature of the left face side is clearly visible. Attempts to laugh resulted in a hanging left lip corner secondary to the weakness of the motor innervation by the facial nerve. In the gray-marked area there is a permanent hypoesthesia.

Fig. 20.4 **A,** Radiograph of the mandibular left first molar with a long indistinct separated instrument obviously longer than 4.5 mm in the mesial root that is extruded beyond the radicular foramen. **B1,** Sagittal view of cone-beam computed tomography (CBCT) showing the radicular end of the separated instrument is extended into the IAN and it measures 14 mm on it. **B2,** Sagittal view of CBCT showing root perforation in the distal canal *(red arrow)*. **B3,** Axial view of CBCT showing the perforation in the distal canal *(red arrow)*. **B4,** Coronal view of CBCT showing the radicular end of the separated instrument is in the IAN. **C,** Separated instrument in the mesiobuccal canal upon removal of root filling materials *(red arrow)*. **D,** Radiograph showing removal of the root fillings in the mesiobuccal canal revealed the overall length of the separated instrument *(red arrow)*. **E,** Retrieved separated instruments measuring 14 mm on the ruler. **F,** Radiograph showing the completion of the separated instrument removal. **G,** Perforation site in the distal canal *(yellow arrow)*. **H,** Perforation repaired with mineral trioxide aggregate (MTA) *(yellow arrow)*. **I,** Postoperative radiograph showing root canal fillings in all the canals and perforation repair

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(yellow arrow). **J,** One-month postoperative radiograph revealing the periradicular lesions still present. **K,** Three-month postoperative radiograph showing periradicular healing.

Fig. 20.5 **A,** Separated file covered with debris. **B,** Mineral oil immediately permeated the spaces between the canal walls and the separated file revealing the surface of the file and broke through the entire file. **C,** Separated file covered with debris. **D,** Water did not permeate those spaces at all.

Fig. 20.6 **A**, Ninety-degree semicircular space created on the inner canal wall with the spoon tip on the microscopic view *(white arrow)*. **B**, A 150-degree semicircular space created on the inner canal wall with the straight tip, which is still not wide enough to make it "dance" because of the canal walls on both sides still holding the separated file *(white arrows)*. **C**, More than 180-degree semicircular space created on the inner canal wall with the straight tip, which results in loosening the separated file. **D**, Magnification of the spoon tip. **E**, Magnification of the Katana tip; a frontal view on the left (0.5 mm in width) and a side view on the right (0.1 mm in thickness). **F**, Magnification of the straight tip (0.1 mm in diameter).

Fig. 20.7 **A**, Thicker ultrasonic tip touches the top of the separated instrument before being placed into the space on the canal wall *(red arrow)*. **B**, Side view shows the thicker ultrasonic tip in relation to the separated instrument, which may result in pushing it into a radicular direction. **C**, Thinner ultrasonic tip can be placed into the space on the canal wall without touching the top of the separated instrument *(red arrow)*. **D**, Side view shows the thinner ultrasonic tip in relation to the separated instrument.

Fig. 20.8 **A,** Canal enlarged to the separated file with a large diameter rotary file. **B,** Space created on the inside curve with the spoon ultrasonic tip, followed by the straight tip. **C,** Space extended to about one third of the file length until the separated file dances. **D,** Ultrasonic activation from the outer curvature. **E,** Separated file redirected in a radicular direction by ultrasonic activation from the outer wall. **F,** Ultrasonic activation from the inner curvature. **G,** Separated file redirected in a coronal direction by ultrasonic activation from the inner wall. **H,** Ultrasonic tip placed on the separated file where there is no canal wall behind it, which may result in secondary fracture.

Fig. 20.9 The coronal one third of the file length is typically the part to be stuck in the canal walls, whereas the radicular portion of the file is typically

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not in contact with the canal walls. Stress is concentrated mostly on the coronal one third of the file length.

Fig. 20.10 **A,** Separated file may accidentally come out with ultrasonic activation during a preparation phase. **B,** EDTA solution is put into the canal to take advantage of cavitation and acoustic streaming effects for instrument retrieval. **C,** Ultrasonics must be activated within the space created. **D,** Separated file is removed from the canal with ultrasonics in the presence of EDTA solution or oil.

Fig. 20.11 **A**, Separated file in the canal with the space created on the inner wall *(right side)*. **B**, Separated file shifted to a different place in the space created *(red arrow)*. This movement shows the separated file is "dancing." **C**, Separated file in the canal with the space created on the inner wall *(right side)*. **D**, Separated file flexing to a different place *(red arrow)*. **E**. Separated file returning to the original place *(red arrow)*. This movement shows the separated file is "flexing."

Fig. 20.12 **A**, Nonvisible separated file around the second curve, beyond the first curve. **B**, Thin ultrasonic tip placed in the space on the inner canal wall of the second curve.

Fig. 20.13 **A**, Thin ultrasonic tip placed into the gap between the inner wall and the separated file and activated. **B,** Ninety-degree semicircular space created in this gap with ultrasonics. **C,** Ultrasonics activated directly on the separated file from this space. **D,** Canal walls contacting the separated file eliminated with ultrasonic oscillation transferred to the separated file from the vibrating ultrasonic tip. **D,** Separated file loosened and dislodged from the original position *(red arrow)*.

Fig. 20.14 **A,** Separated file beyond the severe curve in relation to the file placed in the mesiobuccal (MB) canal. **B,** Thin ultrasonic tip placed in the gap between the inner wall and the separated file. **C,** Separated file is not visible in the MB. **D,** Separated file coming out of the canal. **E,** Removal of the separated file confirmed on a radiograph. **F,** Retrieved separated file along with the original file

Fig. 20.15 **A**, Separated file loose beyond the radicular foramen. **B,** Ultrasonic irrigation such as ProUltra PiezoFlow using saline may help retrieve the separated file which is not engaged in the canal wall.

Fig. 20.16 **A,** Separated file pushed beyond the radicular foramen. **B,** PiezoFlow activated using saline to create cavitation and acoustic stream. **C,**

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Separated file approaching the radicular foramen with the PiezoFlow. **D,** Separated file brought back into the canal. **E,** Separated file retrieval confirmed on the radiograph. **F,** Retrieved separated file on a ruler. **G,** Root canals obturated with mineral trioxide aggregate (MTA).

Fig. 20.17 **A**, Preoperative radiograph of a maxillary left first molar showing a separated instrument beyond the curve in the mesiobuccal root canal with large periapical lesions *(red arrow)*. **B**, Coronal view of cone-beam computed tomography (CBCT) shows the separated instrument in the buccally curved MB2 canal with a large periapical lesion developing into the sinus floor *(red arrow)*. **C**, Separated instrument measures 2.26 mm on the coronal view. **D** and **E**, Axial view shows the separated instrument in the MB2 canal directed toward the MB1 canal. **F**, Sagittal view shows the separated instrument in the MB2 canal lying in a distal direction with a large periapical lesion *(red arrow)*. **G**, Magnification of a partially visible portion of the separated instrument in the MB2 canal *(white arrow)*. **H**, Magnification of the separated instrument that came out of the canal with ultraspnics *(white arrow)*. **I**, Retrieved separated instrument measured 2.3 mm on a ruler. **J**, Interoperative radiograph confirming removal of the separated instrument from the MB2. **K,** Postoperative radiograph showing the final root fillings with mineral trioxide aggregate (MTA), which reveals dentin sacrifice for removal of the separated instrument was minimum. **L**, Three-month postoperative radiograph showing nice periapical healing. **M**, Three-month postoperative coronal view of CBCT showing periapical bone reformation associated with the mesiobuccal root *(white arrow)*. **N**, Three-month postoperative sagittal view of CBCT showing periapical bone reformation associated with the mesiobuccal root *(white arrow)*. **O**, Three-month postoperative axial view of CBCT showing root fillings in MB, MB2, DB, and P root canals.

Fig. 20.18 **A**, Space between the ultrasonic tip and the canal wall is smaller than the diameter of the separated file. **B,** Separated file comes out and bumps against the ultrasonic tip. **C**, Separated file bounces back into the original position. **D**, Space between the ultrasonic tip and the canal wall is wider than the diameter of the separated file. **E**, Separated file comes out through the space. **F,** Ultrasonic tip placed on the top of the separated file in the space on the outer wall.

Fig. 20.19 **A,** Fluid filled in the canal and the pulp chamber. **B,** Separated file

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is coming out of the canal to the pulp chamber in the fluid with ultrasonics *(white arrow)*. **C,** Fluid filled only in the canal. **D,** Separated file fluctuating within the canal space filled with fluid *(red arrows)*.

Fig. 20.20 Terauchi File Retrieval Kit (TFRK). **A,** Modified #3 Gates Glidden bur that is also called GG-3M *(upper)* and microtrephine bur that is also called TFRK-MT *(lower)*. **B1,** TFRK-6 ultrasonic tip (the tip portion looks like a spoon and is also called a spoon tip. It has a micro-concave on the spoon, which turns toward the handpiece at the 6 o'clock position. **B2,** TFRK-S ultrasonic tip (the tip portion looks like a sharp spear and is also called a straight tip). **C1,** Yoshi loop (TFRK-L), which captures a separated instrument. This micro-lasso is comprised of a tiny wire loop at the end of a stainless steel cannula, with a sliding handle that tightens the loop when pulled. **C2,** Yoshi loop holding a separated file from an actual case. **D,** Terauchi File Retrieval Kit (TFRK) in an autoclavable cassette case. The kit includes a GG-3M, TFRK-MT, TFRK-6 ultrasonic tip, TFRK-12 ultrasonic tip, TFRK-S ultrasonic tip, Yoshi loop, TFRK- GPR, and TFRK-ME, High Speed Polishing Point (HSPP), NiTi GT #70/.12 Rotary File (GT70.12).

Fig. 20.21 **A,** Coronal portion of the separated file should be exposed at least 0.7 mm above the bottom of the space created around it. A #40 plugger should be placed in the space to assess the available space prior to the placement of the loop. **B,** Approaching the separated file with the loop bent to 45 degrees against it. **C,** Loop is pushed back to 90 degrees as it is placed over the separated file. Removal with the loop requires the diameters of the loop device (0.4 mm) and the separated instrument and a depth of at least 0.7 mm. **D,** Separated file is pulled out with the loop device in various directions. **E,** Loop prebent to 90 degrees occupying more room in the canal. **F,** Loop pre-bent to 45 degrees requiring minimum room in the canal. **G,** Loop size is adjusted using an endodontic explorer.

Fig. 20.22 Schematic diagrams showing the most common preparation errors. **A,** Radicular zip. **B,** Ledge. **C,** Radicular zip with perforation. **D,** Ledge with perforation.

Fig. 20.23 **A,** Preoperative radiograph showing the failing outcome of endodontic surgery for the mandibular right first premolar with the root canal filling in one root canal. **B,** Cone-beam computed tomography (CBCT) image in coronal view showing a missed lingual canal *(red arrow)*. **C,** CBCT image in axial view showing the missed lingual canal lingual *(red arrow)* to the

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mesiodistal midline in relation to the buccal canal buccal to the midline with the same distance between the midline and the lingual canal. **D,** Magnification of the missed lingual canal *(red arrow)*. **E,** Postoperative radiograph showing root canal obturation in both the buccal and the lingual root canals.

Fig. 20.24 **A,** Precurved microinstruments such as EndoHandle with a precurved #25 Hedstrom File (Venta Endo, Logan, UT) and micro-Opener (Dentsply Sirona) to explore a ledged canal for the original pathway and to reduce irregularities over the ledge. **B,** Ledge is formed with perforation in a radicular straight direction. **C,** Microscopic view showing the ledged canal in the center and a narrow access to the original pathway on the right side. **D,** The canal orifice as well as the access to the original pathway needs to be flared in an anticurvature direction *(red portions)*. **E,** Microscopic view showing red portions to be removed for easy access to the original pathway. **F,** Access to the original pathway was flared to facilitate file insertion. **G,** Bypassing file used in short push-pull strokes to create a distinct pathway to the original canal as the final glide path.

Fig. 20.25 **A,** Micro-hand instrument (TFRK-ME) to explore canals for the original pathway and bypass a ledged canal. **B,** Magnified image of a TFRK-ME.

Fig. 20.26 Micro-hand instrument with diamond coating *(ELES)* to bypass/eliminate/reduce a ledge and enlarge the orifice of the original pathway.

Fig. 20.27 Schematic diagrams showing bypassing the ledge with ledge removal hand instruments. **A,** Ledge is created in a radicular straight direction around a curve. **B,** Original pathway is explored and opened up with the TFRK-ME in short push-pull strokes. **C,** Original pathway is widened with the ELES instrument. **D,** ELES instrument used in push-pull motions to reduce the ledge. **E,** Precurved TFRK-GPR is placed into the original canal and moved with short up and down strokes over the ledge. **F,** Ledge was reduced with the precurved TFRK-GPR. **G,** Precurved straight ultrasonic tip can also be used here to reduce the ledge instead of the ELES instrument. **H,** File with a biconical tip is brought into the canal to eliminate/reduce the ledge and negotiate the original canal. The primary cone of the biconical tip has no cutting tip. As it hits the curve or irregularities on the canal wall, it will slip alongside the curve and scoot over the irregularities. **I,** Secondary

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cone of the biconical tip removes the points at the transition angles and provides a smooth surface that guides the tip portion into the original pathway. **J,** Orifice of the original canal over the ledge needs to be enlarged for easy file insertion after the ledge has been bypassed successfully. **K,** Orifice of the original canal can be efficiently widened with a precurved instrument such as a ProTaper Gold S1 rotary file and a #15/.04 Vortex Blue rotary file used as a hand file, and the apical patency established. **L,** Original canal can be sequentially prepared with larger NiTi files. **M,** Postoperative canal showing the ledge is significantly reduced.

Fig. 20.28 **A,** Preoperative radiograph of a mandibular right first molar showing a periradicular lesion associated with the mesial root and two separated files in the mesial and the distal root *(red arrows)*. Note there are two separated files in the mesial and the distal roots in addition to the fact that the root filling in the mesial root deviates away from the center of the root. **B,** Cone-beam computed tomography (CBCT) image in sagittal view showing the original canal *(red arrow)* in the center of the root while the ledge *(white arrow)* turns away in the straight line in the mesial root. **C,** CBCT image in coronal view showing the MB and the ML canals merge into one canal *(red arrow)* in the apical third. **D1** and **D2,** Consecutive CBCT images in axial view showing the MB canal and the ML canal *(white arrows)* join into one oval canal *(red arrow)*. **E,** Magnification of the MB canal showing a separated file *(red arrow)*. **F,** Retrieved separated files from the mesial root and the distal root. **G,** Interoperative radiograph showing removal of the two separated files. **H,** Placement of a small file into the MB canal reveals a perforation on the outer wall in the straight line form from the orifice on the interoperative radiograph. **I,** Ledge *(red arrow)* and the original pathway *(white arrow)* were identified in the MB canal under the DOM based on the axial view of CBCT and the orifice of the original canal was enlarged with a precurved #10/.06 micro-opener with short push-pull strokes. **J,** Placement of a precurved small hand file (ProTaper S1) reveals it in the original canal reaching the working length on the interoperative radiograph. **K,** Placement of the final shaping file into the MB canal confirms it in the original canal reaching the working length without transportation on the interoperative radiograph. **L,** Postoperative radiograph shows root canal fillings with mineral trioxide aggregate (MTA) including the ledged canal with the perforation. **M,** Three-month postoperative radiograph shows a

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dissolving periapical lesion.

Fig. 20.29 Nonvisible ledge removal with ultrasonics. **A,** Ledge formed beyond the curve. Cone-beam computed tomography (CBCT) is taken to locate both the ledge and the original pathway. **B,** Canal coronal to the ledge is flared in an anticurvature direction with NiTi rotary files in brushing motions to possibly visualize the ledge and the original pathway. Original pathway to the canal terminus is explored with a #10 precurved K-file or the precurved TFRK-ME for a sticky feeling and then radiographs are taken with it inserted in the canal to see if it is in the original canal beyond the ledge. **C,** Straight ultrasonic tip is precurved the same way as the hand instrument used and placed into the original canal to eliminate/reduce the ledge and to open up the entrance to the original pathway. The red portion needs to be smoothed out to facilitate the insertion of the next larger file into the original pathway. **D,** Postoperative canal showing the ledge is significantly reduced.

Fig. 20.30 **A,** Preoperative radiograph of a maxillary right second molar showing periradicular lesions. Note the root filling in MB is overextended beyond the radicular foramen. **B,** A series of cone-beam computed tomography (CBCT) images in axial view from the apical third to the radicular foramen (**B1** to **B3**) reveals an oval MB canal turning round toward the radicular foramen and the original pathway *(red arrows)* curves in a distopalatal direction with the root filling turning away from the original canal (off-center). **C1** and **C2,** A pair of CBCT images in sagittal view clearly show both the overextended root filling (*red arrow* in **C1**) and the presence of the distally curved original canal (*red arrow* in **C2**). **D,** CBCT image in coronal view also shows a large lesion associated with the MB canal, with the root filling extruded beyond the radicular foramen (perforation). **E1 to E3,** A series of microscopic images shows the overextended root fillings being removed with the TFRK-GPR (**E1** and **E2**) and the original canal (**E3,** *red arrow*) was located in the MB canal and enlarged with ultrasonics. **F,** Original canal was negotiated and then the apical patency was established with a precurved micro-opener followed by a precurved ProTaper S1. **G,** Postoperative radiograph showing the completed root fillings including the original canal and the ledged canal with perforation *(red arrows)*. **H,** Three-month follow-up showing healing in progress. **I,** Six-month follow-up showing nice periapical healing. **J,** A series of 6-month postoperative CBCT images in axial view from the apical third to the

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radicular foramen (**J1** to **J3**) reveals root fillings in both the original canal and the ledged canal with nice periapical healing. **K,** A pair of 6-month postoperative CBCT images in sagittal view (**K1** and **K2**) shows nice periapical healing compared to the preoperative CBCT images. **L,** Six-month CBCT image in coronal view also shows nice periapical healing.

Fig. 20.31 **A,** Canal coronal to the ledge is enlarged with a #3 GG bur. **B,** Small straight rotary file (#10/.02 or #10/.04) rotating at 300 RPM or faster will not get into the original pathway. **C,** Small precurved rotary file rotating at 100 RPM or 150 RPM with 90-degree clockwise and 30-degree counterclockwise reciprocating motions will most likely get into the original pathway. **D,** Small precurved rotary file rotating at 300 RPM ended up going into the ledged canal *(red arrow)*. **E,** Small precurved rotary file rotating at 100 RPM was able to get into the original canal *(white arrow)*.

Fig. 20.32 **A,** Radiograph taken in 2010 for evaluation of the ledge *(red arrow)* in the mandibular left first premolar and the separated file *(white arrow)* in the mandibular left second premolar showing no radiographic periapical pathosis and both of the teeth were asymptomatic. **B,** Radiograph taken in 2019 for evaluation of those teeth showing the same without any sign of periapical pathosis *(red and white arrows)*.

Fig. 20.33 Maxillary sinus aspergillosis. **A,** Preoperative radiograph showing a lesion associated with the palatal root, from which the extrusion of the root filling materials occurred in the maxillary sinus. **B,** Cone-beam computed tomography (CBCT) image in sagittal view showing the relation between mucositis on the maxillary sinus floor and the gutta-percha filling materials. **C,** CBCT image in axial view showing the relation between mucositis on the mesial wall of the maxillary sinus and the extruded root filling materials. **D,** CBCT image in coronal view showing the relation between the palatal root and the extruded root filling materials. **E,** Postoperative radiograph of orthograde root canal retreatment on the maxillary left first molar showing the mesiobuccal, distobuccal, and palatal root canals were adequately filled without overextension of the filling materials. **F,** Aspergillus was found on the filling materials retrieved from the maxillary sinus.

Fig. 20.34 Root filling materials extruded beyond the radicular foramen. **A,** Preoperative radiograph showing overextension of the root filling material with a large lesion associated with the root. **B,** Root filling in the canal was mechanically removed, but the extruded portion of it remained in the

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periradicular tissues. **C,** Mineral trioxide aggregate (MTA) was filled in the canal with the extruded portion remaining in the lesion. **D,** Six-month postoperative radiograph showing the reduced size of the lesion, but the lesion surrounding the extruded portion stayed the same in size for the next 6 months, indicating infection of the extruded gutta-percha point. The extruded portion was surgically removed from the lesion at this point. **E,** Three-month follow-up radiograph after the surgery showing the resolving lesion. **F,** Twelve-month follow-up radiograph after the surgery showing a more periradicular healing. **G,** Gutta-percha point retrieved from the lesion.

Fig. 20.35 **A,** Preoperative radiograph showing a large periradicular lesion associated with the root of the mandibular left incisor. **B,** Interoperative radiograph showing the cast post and the root filling in the canal was mechanically removed. Calcium hydroxide was placed as an intracanal dressing on the first appointment. **C,** Postoperative radiograph on the second appointment showing the canal was obturated with mineral trioxide aggregate (MTA), which resulted in overextension of the material into the periradicular tissues. **D,** Six-month postoperative radiograph showing the significantly reduced size of the lesion. **E,** One-year postoperative radiograph showing a nice periradicular healing with hard tissues formed on the radicular end of the MTA filling, resulting in a longer root aside from a residual of the MTA root filling extruded into the periradicular tissues. **F,** Three-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with the extruded MTA filling reduced in size. **G,** Twelve-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with the extruded MTA filling completely dissolved in the bone. **H,** Sixteen-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with healthy lamina dura surrounding the root.

Fig. 20.36 **A,** Preoperative radiograph of the mandibular right first molar showing extruded root filling materials into periapical tissues *(red arrows)*. **B1** and **B2,** Cone-beam computed tomography (CBCT) images in axial view showing the perforation site from the mesiobuccal canal into the furcation *(red arrow)*. **C,** CBCT image in sagittal view showing extruded root fillings from the distal root *(red arrow)* and the perforation *(white arrow)*. **D1** and **D2,** CBCT images in coronal view showing the mesial root without extruded root fillings *(red arrow)* (**D1**) and the distal root with extruded root fillings *(red arrow)* (**D2**). **E,** Magnification of the MB canal with perforation *(red*

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arrow). **F,** Interoperative radiograph showing removal of the intracanal root fillings. **G,** Magnification of the MB canal showing removal of the extruded root fillings from the perforation with an XP-Endo Shaper. **H,** Magnification of the distal canal showing removal of the extruded root fillings from the distal root end with an XP-Endo Shaper. **I,** Magnification of the root canals after the removal of extruded root fillings *(white arrows)*. **J,** Postoperative radiograph showing all the root canals including the perforation were successfully filled with mineral trioxide aggregate (MTA). **K,** Three-month postoperative radiograph showing reduced periapical lesions. **L,** Ten-month postoperative radiograph showing nice periapical healing. **M,** Ten-month postoperative CBCT image in sagittal view showing furcal healing. **N,** Ten-month postoperative CBCT image in sagittal view showing nice periapical healing around the perforation site *(white arrow)*. **O,** Ten-month postoperative CBCT image in coronal view showing nice periapical healing associated with the mesial root *(white arrow)*. **P,** Ten-month postoperative CBCT image in coronal view showing nice periapical healing associated with the distal root *(white arrow)*. **Q** and **R,** Ten-month postoperative CBCT image in axial view showing periapical healing associated with the perforation site *(white arrows)*.

Fig. 20.37 Diagrams showing removal of gutta-percha filling materials extruded beyond the radicular foramen. **A,** Solid gutta-percha filling extruded beyond the radicular foramen. **B,** Small space is created between the canal wall and the gutta-percha filling with a thin ultrasonic tip (such as TFRK-6/12 and TFRK-S ultrasonic tips). **C,** Gutta-percha removal hand instrument, such as the TFRK-GPR and the EGPR, is brought into the space with the tiny projection pressed against the gutta-percha filling to pull it out. **D,** Extruded gutta-percha root filling portion separated from the coronal portion within the canal. **E,** Chloroform or some other solvent is filled in the canal to agitate it beyond the widened radicular foramen with a gutta-percha removal hand instrument or expandable rotary files to dissolve the extruded portion. **F,** Gutta-percha filling material is diluted, dissolved with the hand instrument in push-and-pull motions, and is irrigated out with a minimally invasive irrigant such as saline. **G,** Extruded gutta-percha filling material can also be braided out with those expandable rotary files with caution not to break them or not to damage the inferior alveolar nerve if close to it.

Fig. 20.38 **A,** Panoramic radiograph demonstrating significant overfilling and

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extension in the radicular vacuoles and overlying the mandibular canal. **B,** A coronal CT section demonstrating that the extruded material is lateral and below the mandibular bundle *(arrows).* The patient's sensory complications were temporary.

Fig. 20.39 Cone-beam computed tomography (CBCT) image in coronal view showing that the mesiobuccal root apex of a maxillary second molar is in close proximity with the sinus floor.

Fig. 20.40 Cross section of mandible in the molar region showing the presence of bone vacuoles *(yellow arrows)*.

Fig. 20.41 Longitudinal mandible cross section in the molar region showing the spongy bone cover of the neurovascular bundle. Note the multiple fenestrations and honeycombed trabeculations.

Fig. 20.42 Overfill of the distal root under direct observation. Note the proximity of the extruded material to the artery and nerve bundle (pedicle).

Fig. 20.43 Full-thickness reflection of the flap in the region of the premolar root apices reveals the mental nerve exiting the foramen; note how the tissue "funnels" from the reflected flap to the foramen.

Fig. 20.44 Infiltration dentistry is dependent upon the site and procedure.

Fig. 20.45 **A,** Nerve injury proximal to endodontically treated tooth. Cause = endo. **B,** Inferior dental block nerve injury initially will cause neuropathy of the whole dermatome.

Fig. 20.46 **A,** Facial presentation after the rubber dam was removed. **B,** Close-up view reveals marked distention of the left suborbital region, with ptosis of the eye and loss of the nasolabial fold. There was marked crepitus upon palpation, but the patient was pain-free. **C,** She was placed on an antibiotic regime, as per the protocol, 2 days after the incident, the emphysema has resolved and the tissues are normal in color and texture.

Fig. 20.47 **A,** Preoperative radiograph tooth #10[158]; dystrophic calcification has obliterated the pulp chamber and the coronal half of the canal space. **B,** A #10 file inserted to estimated working length; the file is actually through a facial perforation of the root at the radicular extent of the ultrasonic excavation. Repeated drying of the canal space using an air syringe forced air through the perforation site and precipitated the emphysema seen in Fig. 20.26, *A* and *B*. **C,** The radicular perforation site was sealed with mineral trioxide aggregate (MTA) before the canal space was obturated. The tooth

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has remained asymptomatic since the incident.

Fig. 21.1 Dichlorodifluoromethane (−40°F [−40°C]) gas is sprayed on a cotton pellet and then placed on the incisal edge of the maxillary incisor.

Fig. 21.2 Laser Doppler machine.

Fig. 21.3 An occlusal film of a luxation injury of central incisors. Both were diagnosed to be laterally luxated with apical translocation. Note that the left central is completely obliterated and has a history of being luxated some years prior.

Fig. 21.4 **A,** Panoramic radiograph taken on a patient with past history of dentoalveolar trauma. Tooth #9 appeared to have arrested root development with a periapical radiolucency. **B,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 had arrested root development, a large periapical radiolucency, and extensive root resorption along the palatal surface that was *not* evident on the panoramic image. **C,** Periapical radiograph of tooth #9 with extensive complicated crown fracture. The clinical examination revealed the crown fracture extended subgingivally on the palatal aspect. **D,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that the apical extent of the fractured palatal portion extended just apical to the crest of bone. The correct diagnosis of this injury was complicated crown-root fracture. **E,** Periapical radiograph of teeth #8, #9 revealed crown fractures. **F,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 suffered a lateral luxation displacement injury with extensive concomitant alveolar fracture. The necessary treatment of repositioning and splinting was apparent. **G,** Periapical radiograph of tooth #9 following dentoalveolar trauma. Tooth #8 was avulsed and tooth #9 appeared to be severely intruded. **H,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed the labial position of tooth #9. **I,** Periapical radiograph of tooth #8 revealed a lateral luxation injury. **J,** Sagittal view of tooth #8 from CBCT imaging. The extensive tooth displacement and concomitant alveolar are evident. **K,** Periapical radiograph of teeth #8 to #10, that had recently been splinted in an emergency room. This image revealed that these teeth were not properly repositioned before splinting, evidenced by the spaces along each root. **L,** Axial view of maxillary anterior region of same patient from CBCT imaging. This revealed that teeth #8 to #10 are labially displaced. **M,** Sagittal view of tooth #10 of same patient revealed how severe the displacement was.

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Following consent and local anesthesia, the splint was removed and teeth #8 to #10 were repositioned and resplinted.

Fig. 21.5 Patient sustained complicated crown fractures on the left lower incisors and later on the canine tooth. **A,** An emergency pulp capping; Band-Aid composite was placed on the teeth. Six days later the patient was referred to the clinic because of a poorly healed laceration on the lip. **B,** Radiograph of the lip revealed a portion of the lateral crown still in the lip. The patient was anesthetized and the crown removed. **C,** Healing was quick and uneventful.

Fig. 21.6 **A1,** A clinical photograph of a maxillary right central incisor that sustained a crown-root fracture. **A2,** A periapical radiograph that failed to show the extent of the crown-root fracture. **A3,** A CBCT image revealed the extent of the crown-root fracture. **B1,** A clinical photograph of a right maxillary central incisor that had sustained a crown fracture that was restored with composite resin by the referring dentist. **B2,** A periapical radiograph revealed a vertical fracture in the crown and the coronal third of the root. An area of bone loss just below the alveolar crest on the mesial aspect of the root suggested further injury. **B3,** A CBCT image revealed the further injury as a transverse root fracture.

Fig. 21.7 Complicated crown fracture involving enamel, dentin, and pulp.

Fig. 21.8 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferated over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

Fig. 21.9 Histologic appearance of the pulp days after a traumatic exposure. Superficial necrosis above a zone of inflamed pulp is seen.

Fig. 21.10 Pulp necrosis of 1.5 mm as a result of the high pH of calcium hydroxide.

Fig. 21.11 Hard-tissue barrier after calcium hydroxide partial pulpotomy. **A,** Histologic appearance of replacement odontoblasts and a hard-tissue barrier. **B,** Clinical appearance of the barrier on removal of the coronal restoration 3 months after placement of the calcium hydroxide. **C,** Radiographic appearance of the hard-tissue barrier *(arrow)*.

Fig. 21.12 Cvek partial pulpotomy. **A,** The fractured teeth are cleaned and disinfected; a rubber dam is placed. **B,** Cavities are prepared at high speed with a round diamond bur 1 to 2 mm into the pulpal tissue. **C,** Calcium hydroxide on a plugger **(D)** is placed on the soft tissue of the pulp. **E,** Care is

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taken to avoid smearing the walls of the preparation with the calcium hydroxide. **F,** Cavity preparations are filled with glass ionomer cement. The exposed dentin is etched **(G)** and then covered with composite resin **(H)**. **I,** Radiograph 6 months later shows formation of hard-tissue barriers in both teeth *(arrows)*.

Fig. 21.13 Continued root development after partial pulpotomy. **A,** Radiograph of immature tooth with a complicated crown fracture. **B,** At the time of placement of calcium hydroxide after partial pulpotomy *(arrow)*. **C,** Follow-up radiograph confirming that the pulp maintained vitality and the root continued to develop.

Fig. 21.14 Successful pulpotomy followed by a pulpectomy at 18 months.

Fig. 21.15 Creamy mix of calcium hydroxide on a Lentulo spiral instrument ready for placement into the canal.

Fig. 21.16 **A** and **B,** Root canal that "disappears" after placement of a thick mix of pure calcium hydroxide and then over time washes out again.

Fig. 21.17 Apexification with mineral trioxide aggregate. **A,** The canal is disinfected with light instrumentation, copious irrigation, and a creamy mix of calcium hydroxide for 1 month. **B,** Calcium sulfate is placed through the apex as a barrier against which the mineral trioxide aggregate (MTA) is placed. **C,** A 4-mm MTA plug is placed at the apex. **D,** The body of the canal is filled with the Resilon obturation system. **E,** A bonded resin is placed below the cementoenamel junction (CEJ) to strengthen the root.

Fig. 21.18 Histologic appearance of hard-tissue barrier after calcium hydroxide apexification. The barrier is composed of cementum and bone with soft-tissue inclusions.

Fig. 21.19 Root filing with a soft technique after calcium hydroxide apexification. Sealer and softened filling material are expressed through the "Swiss cheese" holes in the barrier.

Fig. 21.20 **A,** Preoperative radiograph showing an immature incisor (tooth #9) that sustained an uncomplicated crown fracture where pulp necrosis developed as shown by cessation of root development when compared with tooth #8. **B,** A 6-year review radiograph showing the tooth after treatment with a regenerative endodontic protocol. There were no signs of infection (primary goal). Calcific material at the apex is present indicating apical closure. A quantitative analysis as described by Bose et al.[44] reports a 1.9%

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increase in root length and a 5% increase in canal wall width (secondary goal). The tooth was responsive to electric pulp testing in a comparable range to tooth #8 (tertiary goal).

Fig. 21.21 Extracted root after a root fracture. Note the oblique angle of the fracture.

Fig. 21.22 Radiographs showing the importance of different vertical angulations for diagnosis of root fracture. All three radiographs were taken within minutes of each other.

Fig. 21.23 Figure of facial root fracture manipulated back into place.

Fig. 21.24 Healing patterns after horizontal root fractures. **A1,** A transverse root fracture in a 7.5-year-old boy. **A2,** Healing with calcified tissue with internal thickening of root dentin into the pulp chamber at a 3-year review. **B,** Healing with interproximal connective tissue. **C,** Healing with bone and connective tissue. Note extensive intracanal calcification of tooth #9. **D,** Interproximal connective tissue without healing.

Fig. 21.25 **A,** Sagittal histologic section in a labiopalatal plane showing pulp and hard-tissue deposition between the fractured segments on the labial and to a greater extent on the palatal aspect (Van Gieson stain, original magnification ×4). **B,** Labial section showing hard-tissue deposition between approximately half of the fractured dentin surfaces and bulging into the root canal space (Van Gieson stain, original magnification ×10). **C,** Higher-power view of 25A showing fibrillar dentin deposit *(FD)* with some small and larger cell/tissue inclusions and tubular dentin *(TD)* with a sunray orientation bulging into the root canal and lined by pulp (original magnification ×20). **D,** Polarized light view of the pulpal extremity of dentin deposition showing the tubular orientation and deeper irregular hard-tissue formation with cell/tissue inclusions (original magnification ×25). **E,** Palatal section showing hard-tissue deposition between the majority of the fractured dentin surfaces and budging into the root canal. The calcified tissue deposits consist of tubular dentin *(TD)* fibrillar dentin *(FD)* with cell/tissue inclusions, considered to be of pulpal origin *(P)*, and sclerotic calcified tissue *(SCT)*, considered to be of periodontal ligament origin (Van Gieson stain, original magnification ×10). **F,** Higher-power view of the calcified tissue considered to have been pulpally deposited showing further details of the orientation of the dentinal tubules in the tubular dentin layers *(TD)* and the deeper fibrillar dentin layers *(FD)* (original magnification ×20).

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Fig. 21.26 **A,** The intact central incisor after extraction and formalin fixation. **B,** The histologic appearance of the extracted tooth sectioned in a mesiodistal sagittal plane showing the interposition of fibrous connective tissue *(FCT)* calcification of the root canals but evidence of residual pulp tissue *(P)* (H&E, original magnification ×4). **C,** View of the fracture site showing dentin resorption lines *(RL)* repaired with reparative hard tissue *(RHT)* and the interposed dense fibrous connective tissue *(DFCT)* (H&E, original magnification ×10). **D,** Higher-power view showing dense connective tissue, generally with a parallel orientation to the fractured dentin surfaces that show reparative hard tissue laid down on previously resorbed dentin. There is minimal vascularity, but some extravasated erythrocytes are evident (Van Gieson stain, original magnification ×20). **E,** Highlighted section from **(C)** using polarised light to demonstrate Sharpey fiber insertion into the reparative hard tissue *(arrows)* (original magnification ×40).

Fig. 21.27 **A,** Pulp canal calcification after a luxation injury. Radiographic appearance of a calcified maxillary lateral incisor; note that despite calcification, it has now become necrotic and infected, evident by the periapical lesion. **B,** The typical yellow appearance of the tooth caused by thickened dentin in the pulp chamber.

Fig. 21.28 Internal root resorption in the root fracture area; tooth was diagnosed with compound midroot fracture. **A,** Coronal portion was repositioned and then splinted to the lateral incisor with a composite splint. **B,** At 6-month recall, the pulp responded normally to cold, but an internal root-resorptive defect was noted on radiograph. No treatment was indicated, because the pulp did respond normally to vitality tests. **C,** At 42-month recall, the tooth was still asymptomatic, and the pulp still responded to vitality tests. The resorptive defect had healed, and signs of dystrophic calcifications were evident in the apical and coronal segments.

Fig. 21.29 Histologic section showing previous root resorptive defect healed with new cementum and periodontal ligament.

Fig. 21.30 Histologic appearance of replacement resorption. There is direct contact between bone and root structure. Resorptive defects are seen in the bone and root tissue, which is the normal physiologic process of bone turnover. The resorptive defects are filled by new bone and additional areas resorbed. In this way the entire root is replaced by bone at a rate dependent on the metabolic rate of the patient.

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Fig. 21.31 Radiographic appearance of replacement resorption. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are not present.
Fig. 21.32 Inflammatory root resorption caused by a pulp space infection. Note the radiolucencies in the root and surrounding bone.
Fig. 21.33 **A,** Periapical radiograph of tooth #9 of a 16-year-old boy showing a mature tooth 6 months after avulsion and replantation within 5 minutes. Inflammatory root resorption is occurring as endodontic treatment was not commenced. **B** and **C,** CBCT sagittal images taken same day as the periapical radiograph. **D,** CBCT axial images.
Fig. 21.34 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the dentin of the root.
Fig. 21.35 Revascularization of immature root. A tooth with an open apex was replanted soon after the avulsion. The checkup radiograph 12 years later confirms that regrowth has taken place into the pulp chamber. It appears that a bone plug has grown in, and new periodontal ligament has formed, with a lamina dura within the pulp canal.
Fig. 21.36 High pH of calcium hydroxide. The root was filled with calcium hydroxide and then cut in cross section. A pH indication shows the high pH in the canal and surrounding root, whereas the surrounding tissue is a neutral pH.
Fig. 21.37 Healing of external inflammatory root resorption after calcium hydroxide treatment. The radiolucencies seen before treatment have disappeared with the reestablishment of the lamina dura.
Fig. 21.38 Histologic appearance of internal root resorption. **A,** Section stained with Brown and Brenn. Bacteria are seen (in the dentinal tubules) communicating between the necrotic coronal segment and the apical granulomatous tissue and resorbing cells. **B,** An area of active internal root resorption.
Fig. 21.39 Angled radiographs to show internal resorption. Radiographs from two different horizontal projections depict **(A)** the lesion within the confines of the canal on both views and **(B)** the adjacent bone intact on both views.
Fig. 21.40 A maxillary incisor with internal root resorption. Uniform enlargement of the pulp space is apparent. Outline of the canal cannot be seen in the resorptive defect.

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Fig. 21.41 **A,** Internal root resorption in a maxillary premolar with a history of trauma 7 years before the diagnosis (patient's head slammed against side window during an automobile accident). **B,** Three-year follow-up radiograph after endodontic treatment.
Fig. 21.42 External root resorption. Radiographs from two different horizontal projections depict movement of the lesion to outside the confines of the root canal.
Fig. 21.43 **A,** Periapical radiograph of invasive cervical root resorption in tooth #8 and #9 in a 68-year-old female where the only known trauma was a fall onto bitumen aged 7 years when hit by a car. These teeth are diagnosed as having Heithersay Grade IV invasive cervical resorptive lesions which can be confirmed by CBCT imaging. **B** and **C,** CBCT coronal images. **D,** CBCT sagittal image of tooth #8. **E,** CBCT axial image. The outline of the canal is evident in Panels A, B, D, and E. The pulp does not appear exposed due to the anticlastic properties of predentin.
Fig. 21.44 Tooth #9 exhibiting invasive cervical resorption in a 31-year-old male. The tooth was injured 3 years prior after a fall from a scooter and was root filled and restored with a post crown. The resorption commences in the cervical external root and extends deeper into the root consistent with a Heithersay Grade III invasive cervical resorptive lesion. A resorptive defect is also present in the adjacent bone.
Fig. 21.45 Pink spot of invasive cervical root resorption. **A,** Radiographic appearance. **B,** Clinical appearance.
Fig. 21.46 **A,** Avulsed tooth soaking in doxycycline. **B,** Minocycline powder placed on the root surface before replantation.
Fig. 21.47 Titanium trauma splint (TTS) in place.
Fig. 21.48 **A,** An occlusograph showing root fillings in teeth #9 and #10 in a 44-year-old male 25 years after the teeth had been traumatized. Symptoms and infection returned after endodontic treatment and surgical correction was required. **B,** CBCT imaging revealed internal resorption within the canal.
Fig. 21.49 **A,** An occlusograph showing extensive periapical radiolucencies associated with the maxillary incisor teeth in a 23-year-old male. The patient sustained a traumatic injury when he was 8 years of age. Tooth #8 has extruded gutta-percha through the open apex indicating either a failed apexification procedure or poor obturation of the canal due to the open apex. Tooth #9 has an open apex indication pulp necrosis likely occurred at or soon

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after the traumatic injury but no treatment was undertaken. **B,** CBCT imaging revealing the immature root formation of tooth #9 and periapical lesion. Surface resorption on the palatal cervical root is suggestive of a prior palatal luxation injury.

Fig. 22.1 Complete vertical root fracture. A cross section of a mesial root of a mandibular molar with a complete vertical root fracture. The fracture is in the buccolingual plane and extends from the buccal convexity of the root to its lingual convexity.

Fig. 22.2 The Tooth Slooth device. Application for a bite test: the tip of the pyramid is touching the tested cusp while the wide base is supported by multiple contacts.

Fig. 22.3 Transillumination for the detection of a cracked cusp/tooth. An intense but small light source is applied to the suspect tooth, preferably in relative darkness. The light is transmitted through the tooth structure but is reflected from the crack plane, leaving the area behind the crack in darkness.

Fig. 22.4 A fractured cusp. **A,** The mesiopalatal cusp of a maxillary first right molar was fractured. The fractured cusp was movable and was retained by the periodontal ligament. **B,** The cusp was removed, and the tooth was considered restorable. It was treated with root canal treatment and a crown.

Fig. 22.5 A case of fracture-induced necrosis. A tooth with minimal or no restoration or caries is unlikely to become nonvital. **A,** This radiograph of a mandibular second molar shows a restoration that is distant from the pulp chamber, yet the tooth is nonvital and symptomatic. **B,** On occlusal examination, a slight crack is observed on the distal marginal ridge. **C,** After extraction, the mesial aspect of the crown and root shows no indication of a fracture. **D,** However, the distal aspect of the crown and coronal root shows the fracture. **E** and **F,** When the crown is sectioned, the crack can be observed to extend well into the pulp chamber.

Fig. 22.6 **A,** Observing split tooth by probing or pushing cusp. **B,** Radiographic evidence of split tooth.

Fig. 22.7 Diagonal vertical root fracture (VRF) in a molar. A lingual view of a right first mandibular molar revealing a diagonal VRF in the mesial root.

Fig. 22.8 Three types of vertical root fractures (VRFs). **A,** A coronally located VRF extending apically as far as one third of the root. **B,** A midroot VRF extending along the middle third of the root. **C,** An apically located VRF extending coronally as far as the apical two thirds of the root.

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Fig. 22.9 **A** to **E,** Vertical root fractures observed with periapical radiograph, cone-beam computed tomography and clinically after extraction.
Fig. 22.10 Coronally located sinus tracts. **A,** A draining sinus tract at a coronal location originating from a buccal vertical root fracture (VRF) in the first left maxillary premolar. **B,** A draining sinus tract at the gingival margin of a right first mandibular molar with a buccal VRF in the mesial root.
Fig. 22.11 Radiographic examination of filled versus empty canals. **A,** The buccolingual projection of a filled root will fail to detect a vertical root fracture (VRF) at an early stage. **B,** The removal of the root filling and use of radiography at different mesiodistal angulations may reveal the VRF.
Fig. 22.12 Early versus late radiographic presentation of a vertical root fracture–associated bone defect. At an early stage, a bone defect *(red)* is not likely to be detected in a periapical radiograph, as the root will overlap with the defect **(A** and **B).** At later stages, when major damage has occurred to the cortical plate **(C),** the bone defect may be large enough to extend beyond the silhouette of the root **(C** and **D)** and appear as a radiolucent defect along the root **(E).**
Fig. 22.13 Radiographic presentations of longstanding vertical root fractures (VRFs). **A,** J-shaped "halo" associated with a VRF in a second right mandibular premolar. **B,** Extensive bone damage associated with a complete VRF in a second right maxillary premolar. **C,** Limited bone damage associated with a midroot VRF in a second left maxillary premolar. **D,** Bone damage associated with a VRF in the mesial root of a first left mandibular molar. All such presentations are typical of longstanding VRFs.
Fig. 22.14 Radiographs of longstanding vertical root fractures. No special diagnostic skills are needed to diagnose such cases.
Fig. 22.15 Vertical root fracture *(VRF)* pocket. **A,** Periodontal pockets *(left)* are wide coronally, whereas VRF pockets *(right)* are narrow and deep. **B,** Periodontal pockets *(left)* are loose and allow probing at various sites, whereas VRF pockets *(right)* are narrow and tight. If not checked carefully at every millimeter of the sulcus, an early VRF pocket can easily be missed. Note that periodontal pockets appear more commonly in the proximal sides of the root, whereas VRF pockets are more common on the buccal or lingual sides.
Fig. 22.16 Tight vertical root fracture (VRF) pockets. **A,** The distal root of a second right mandibular molar with a VRF. VRF pockets were found on both

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the lingual **(B)** and the buccal **(C)** sides. VRF pockets are tight, so inserting a probe into these pockets causes pressure blanching of the surrounding tissues.
Fig. 22.17 Rigid versus flexible probes. **A,** In a loose periodontal pocket, a rigid metal probe can easily reach the depth of the pocket. **B,** In a tight, early-stage vertical root fracture (VRF) pocket, a rigid probe may be of limited value, as the bulge of the crown often prevents the insertion of the probe into the tight, deep pocket. **C,** A flexible probe **(D)** is more likely to detect VRF pockets at an early stage.
Fig. 22.18 Radiography of an empty canal: a clinical case. The right maxillary lateral incisor underwent a root canal treatment a few years ago. The patient complained about occasional pain on the palatal side. The tooth was sensitive to percussion and palpation on the palatal side. Radiography revealed a periapical radiolucency **(A).** An isolated, deep, and narrow pocket was found on the palatal side of the root. Both the patient and the referring dentist were reluctant to extract the tooth, assuming that the pocket could potentially be a sinus tract, and decided on retreatment. A radiograph taken with a file during the process **(B)** could have missed essential information that was obscured by the file, which was an evident vertical root fracture **(C).**
Fig. 22.19 Diagonal radiographs for detection of vertical root fracture (VRF). **A,** Orthoradial radiograph: very limited bone loss is seen. **B,** A different horizontal angulation reveals a radiolucent lesion along the root. **C,** Schematic presentation. Although a radiograph in an orthoradial direction *(blue lines)* cannot pick up the radiolucency along the root, diagonal angulation *(black lines)* may do so, as in **B.**
Fig. 22.20 **A,** Periapical radiograph of maxillary left first molar. **B,** Cone beam computed tomography (CBCT) coronal view revealing bone loss between the buccal and palatal roots, suggestive of a vertical root fracture. **C,** CBCT axial view revealing bone loss between the buccal and palatal roots, suggestive of a vertical root fracture.
Fig. 22.21 Clinical image of a vertical root fracture, observed using applied dye.
Fig. 22.22 Anatomic predisposing factors. **A,** An axial view of cone-beam computed tomography (CBCT) scan of a maxilla, revealing oval canals in the maxillary canine and second premolar. **B,** An axial view of a mandible, revealing oval canals in incisors, canine, premolars, and distal roots of the mandibular molars. Oval anatomy combined with endodontic treatment has

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been associated with a higher incidence of vertical root fractures (VRFs). **C** and **D,** Concavities on the distal aspect of the mesial roots of mandibular molar may establish a "danger zone" in which excessive instrumentation, combined with straightening of the canal, may result in a thinner dentin wall that may allow strain concentration. **E** and **F,** Concavities in the palatal side of the buccal root of maxillary first premolar (**E**, sections; **F**, axial view from a CBCT). These depressions may also represent a potential danger zone. Neither the concavity in **C** and **D** nor the concavity in **E** and **F** would be evident in a planar periapical radiograph. It should be noted that CBCT scans should not be used for routine screening but should be limited to the indications delineated in the joint statement of the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR).[5,6] **G,** Histology of VRF. **H,** Radiograph revealing peri-radicular bone loss.

Fig. 22.23 Finite element analysis of the strain distribution in an oval root. Note the strain concentration on the inner side of the highest convexity of the remaining dentin wall. Red and orange represent areas of higher strains than blue areas.

Fig. 22.24 Typical bucco-lingual fracture that includes the isthmus in a maxillary premolar with two canals.

Fig. 22.25 **A,** Periapical radiograph revealing bone loss from crestal bone to apex. **B,** As viewed clinically after full thickness flap. **C,** Mesial root resection performed and can see how the root has split. **D,** Final radiograph after mesial root resection.

Fig. 22.26 **A,** Periapical radiograph of mandibular right first molar revealing suspected periradicular bone loss. **B,** Cone-beam computed tomography (CBCT) sagittal view of same tooth revealing bone loss from crestal bone to apex. **C,** CBCT anatomic view revealing mesial osseous defect. **D,** CBCT axial view revealing buccal and lingual bone loss midroot.

Fig. 23.1 The negative impact of poor initial biomechanical status on restoration success. **A** and **B,** Preoperative radiographic views following the removal of the old metallic foundation. **C,** A new amalgam core, using post and self-anchorage into mesial root structure, was performed. **D,** Prosthetic restorations on working model. **E,** Full arch view after 3 years. **F,** The tooth is symptomatic due to furcation involvement and periapical lesion. **G,** This

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untreatable tooth was finally extracted and replaced by an implant. **H,** Eight-year postoperative radiograph showing a stable situation. Other teeth with less extensive biomechanical damage overcame functional stresses.

Fig. 23.2 Severe discoloration can significantly disturb aesthetics, even in the lateral area of the smile. When not treatable with bleaching agents or veneers, this condition might justify tooth preparation for a full crown.

Fig. 23.3 **A,** Stress distribution within a metallic post and core foundation and residual tooth structure, according to photoelastic studies. The post is cemented and usually penetrates the apical portion of the root. Functional stresses accumulate inside the foundation, slightly around the post and further inside the canal, around the apex of the post; there is less stress buildup in the cervical area compared to that with a fiber post (as shown in Fig. 23.3, *B*). This configuration more ideally protects the coronocervical structures, but when failing, it results in severe untreatable root fractures. **B,** Stress distribution within a fiber post/composite foundation and residual tooth structure, according to photoelastic and FEM studies. The post is bonded to the canal walls and penetrates the canal less apically. Functional stresses mainly accumulate around the post in the cervical area. This configuration protects the cervical area less efficiently but tends to prevent untreatable root fractures. The presence of a ferrule is mandatory.

Fig. 23.4 Failure of prosthetic foundations can have dramatic consequences on both overlying restorations and surrounding tissues. A better understanding of compositional and structural changes that affect tooth resistance to repeated functional forces is mandatory to improve treatment success in endodontically treated teeth.

Fig. 23.5 **A,** Current recommendations for the treatment of nonvital anterior teeth. *Normal function and anterior guidance; **moderate to severe parafunctions and abnormal occlusion/anterior guidance. **B,** Current recommendations for the treatment of nonvital posterior teeth. *Relatively flat anatomy and canine guidance, normal function; **group guidance, steep occlusal anatomy, parafunctions.

Fig. 23.6 Direct composite restorations on front teeth (following bleaching). Nonvital anterior tooth with sufficient amount of residual tissue and intact, healthy neighboring teeth, usually in rather young patients. **A,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha were removed from the cavity to expose a clean dentin substrate.

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Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. **B,** Bleaching procedures. Two sessions of internal bleaching were needed to adapt the color of both traumatized anterior teeth (tooth #11), using a mixture of 3% hydrogen peroxide and sodium perborate powder. Each bleaching session extended over a 10-day period. Between sessions and until the given delay to proceed with final restoration (1 to 2 weeks up to 6 to 8 weeks), access cavity is closed with either a temporary filling material or flowable resin composite. **C** and **D,** Restorative procedures. A layer of adhesive is applied over all cavity surfaces. An etch-and-rinse system is frequently preferred when the dentinal tissue is highly sclerotic or was contaminated (e.g., by eugenol). A hard cement such as glass ionomer (not a resin-modified glass ionomer because of water uptake and further expansion) was used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. Two or three layers are usually used to replace the missing tissue, preferably following the "natural layering concept," which aims to build up the restoration with two basic masses, namely dentin and enamel, with the help of a silicone index. **E** and **F,** Postoperative views. The two conservative restorations are shown after finishing and polishing, with the rubber dam still in place **(E)** and after several weeks **(F)**. Note that aesthetic integration cannot be evaluated before full rehydration has occurred (4 to 6 hours at least).

Fig. 23.7 Direct composite restoration on posterior tooth. Nonvital posterior tooth with sufficient amount of residual tissue and mostly intact, rather healthy, neighboring teeth. No significant discoloration of buccal tooth substrate and absence of severe parafunctions or group guidance or other unfavorable occluso-functional condition. **A,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for a later placement of a post. **B,** Adhesive procedure. An etch-and-rinse adhesive system was used here owing to the presence of sclerotic and contaminated dentin. **C** to **E,** Restoration base and layering technique. Resins

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or glass ionomer cements can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. A dentin composite mass is used to replace missing dentinal tissue, and enamel composite is used to build up proximal walls and the occlusal anatomy. **F,** The postoperative view demonstrates the advantage of this conservative approach in the context of teeth with appropriate biomechanical status.

Fig. 23.8 Veneer. Nonvital anterior tooth with sufficient amount of residual tissue and rather healthy neighboring teeth. Discoloration resisted bleaching or relapsed, despite several retreatments. Preferably thick biotype to limit the risk of gingival recession and loss of aesthetic integration. **A,** Closing the lingual cavity. A hard cement such as glass ionomer (not resin-modified glass ionomer because of water uptake and possible expansion) can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. **B,** Preparation. A sufficient amount of buccal tooth structure must be removed to allow for a good aesthetic result (preparation depth is dictated by the discoloration severity). Cervical margins closely follow gingival contours, preventing soft-tissue impingement, but they assume complete coverage of discolored tooth structure. **C** and **D,** Adhesive procedure. Hydrophobic bonding systems are preferred to wet-etched enamel when no dentin is exposed. An etch-and-rinse adhesive system is preferred when dentin is exposed (here, in the presence of significant areas of sclerotic dentin). Restoration. Ceramic is etched and silanated. Just before cementation, a last layer of bonding resin is applied but not light-cured until placement. **E,** Luting technique. A light-curing, highly filled, translucent and fluorescent restorative material is preferably used for cementation. This allows better control of restoration placement and minimal wear and fatigue of the cement layer. **F,** Postoperative view. The postoperative view demonstrates satisfactory aesthetics. This approach is sometimes suitable to meet a patient's high aesthetic demands.

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Fig. 23.9 Onlay/overlay. Nonvital posterior tooth with sufficient amount of residual tissue to justify partial restoration. Preparation involves both proximal surfaces or only two surfaces, but in a less favorable occluso-functional environment (thin walls). No significant discoloration of visible buccal tooth substrate. Option is favorable for short clinical crowns. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak occlusal walls are removed until reaching sufficient thickness for the restoration (1.5 to 2 mm in occlusal-free and occlusal areas, respectively). Adhesive procedure: an etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated dentin (a self-etch adhesive system can also be used). A resin composite base is made with a restorative composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **C** and **D,** After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. This treatment option provides optimal tissue conservation, function, and aesthetics and is a good alternative to full crowns.

Fig. 23.10 Endocrown. Nonvital posterior tooth with reduced amount of residual cervical tissue but sufficient tissue to provide supragingival restoration margins and good stabilization within former pulpal chamber. No or limited discoloration in visible buccal tooth substrate. Favorable option for short clinical crowns. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak walls are removed until reaching sufficient thickness (1.5 mm minimum); as much residual cervical structure as possible is then maintained to avoid restoration interference with periodontal tissues. **C,** Prosthetic restoration on working model. **D,** Restoration base. Adhesive procedures: An etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated

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dentin (a self-etch adhesive system can also be used). Restoration base: A resin composite base is made with a restorative composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **E,** Impression and restoration fabrication. After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. **F,** Postoperative result. Despite their relatively rare application, endocrowns combine biomechanical advantages with facilitation of clinical procedures and minimal if any involvement of biologic width. It is an interesting alternative to full crowns.

Fig. 23.11 Amalgam core. Nonvital posterior teeth with reduced tooth structure and wall height. Amalgam core stability is granted by extension of the restoration into the coronal portion of the canal (curved roots) and post (only if straight root anatomy) and remaining pulpal chamber undercuts. Today, this option is considered obsolete or historical because of the intrinsic staining of amalgam, the nonadhesive technique, and the less-than-ideal biomechanical behavior of amalgam. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Thin and weak walls are removed until reaching sufficient thickness (1.5 to 2 mm); existing retentions are used to stabilize the amalgam core, together with a post. The metal post (with passive and anatomic design, preferably made of titanium) extends into the root for about the same length as its coronal extension. **C** to **E,** Restoration fabrication. After placing a matrix or cupper ring to envelop the preparation, amalgam is compacted into the cavity, superior root canal portion, and around the post until the appropriate coronal volume is restored. Preparation of cores is made to manage optimal space for porcelain fused to metal (PFM) crowns. **F,** Posttreatment result. This treatment approach is feasible for teeth already restored with amalgam cores and when PFM restorations are mandated. Satisfactory aesthetics can be attained with intrasulcular restoration margins.

Fig. 23.12 Composite core (without post). Nonvital tooth with more than half the coronal structure left and sufficient wall thickness. Core stability is granted by adhesion, remaining pulpal chamber, and residual wall height. **A**

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and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. Half of the abutment height and more than 1.5 mm wall thickness must be present to restore the tooth without a post. If aforementioned conditions are met, a thin layer of hard cement (calcium hydroxide, zinc phosphate, or glass ionomer; not resin-modified glass ionomer) is applied at the entrance of the root canal to enable possible retreatment in the future. **C,** Adhesive procedures and core fabrication. All walls are covered with the adhesive system (etch-and-rinse or self-etch) before applying in layers a direct light-curing restorative composite or a self-curing material, in case of limited restoration volume. **D,** Postoperative view. The advantage of this approach is the ease of application and excellent aesthetics. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with limited tissue loss and discoloration.
Fig. 23.13 Composite core with white post (resin-reinforced fiber or zirconia post). Nonvital tooth with less than half the coronal structure left or insufficient wall thickness. Core stability is improved through adhesion and a tooth-colored post (resin-reinforced fiber or ceramic posts). **A,** Preoperative view. **B** and **C,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. If less than half of the abutment height or 1.5-mm wall thickness remain, the abutment requires a post to increase core retention and foundation stability. **D** and **E,** Post-and-core fabrication. Post placement: The post space is prepared with ad hoc instruments until reaching a length equivalent to core height or slightly less depending on remaining wall height. Increased length is not needed, because retention is predominantly achieved through adhesion. Any tooth-colored post can be used, but fiber posts are usually preferred to limit stress buildup into the root structure, even though they provide less rigidity to the foundation. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a direct light-curing restorative composite or with a single

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increment of a self-curing material when only limited volume is to be completed. **F,** Postoperative view. The primary advantage of this approach is the excellent aesthetic outcome. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with more tissue loss but moderate discoloration.

Fig. 23.14 Composite core with prefabricated metal post. Nonvital tooth with reduced coronal structure and ferrule effect or insufficient wall thickness. Core stability is improved through adhesion and a metal (opacified) post (titanium posts are usually preferred). **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. The absence of coronal tissue and ferrule effect mandates the use of a more rigid post (ceramic posts are considered too rigid and contraindicated in this situation). **C** to **F,** Post and core fabrication. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height. More length is not needed, because retention is predominantly achieved through adhesion. Post preparation: The metal post (titanium here) is sandblasted (silicoating can be used to increase adhesion; i.e., Rocatec or CoJet systems, 3M), followed by silane application. An opaque resin or flowable composite can be used to mask metal dark color and improve aesthetics. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a direct light-curing restorative composite or a single increment of a self-curing material when only limited volume is to be completed.

Fig. 23.15 Nonvital teeth with limited coronal structure or numerous abutments. Indirect post and core fabrication helps to achieve proper parallelism and core anatomy. Core retention is assumed by a conventional or adhesive cement. Today, this solution is considered mostly for extended metal-ceramic restorations or sometimes for multiple full-ceramic restorations. **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. Despite the indirect approach, all healthy coronal structures must

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be maintained to improve foundation stability. Access cavity and coronal walls are prepared with minimal convergence (6 to 10 degrees) to allow for easy insertion. **C** and **D,** Cast gold post and core fabrication and luting. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height or more. More length is needed here, as retention cannot always be achieved through adhesion (endodontic retreatment or highly sclerotic contaminated dentin). Then a conventional glass ionomer or zinc phosphate cement is used. Post and core preparation: The gold post is sandblasted (or eventually sandblasted and coated if adhesive procedures are possible) using Rocatec or CoJet systems, 3M + silane. Post cementation: The post and core is luted with the aforementioned conventional cements or adhesive technique. Next, a self-curing adhesive system is applied on all cavity walls and into post preparation (before the insertion of the post and core) and covered with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). **E** and **F,** Restoration fabrication and placement. A second impression is needed for the final prosthetic work to optimize restoration precision and quality. Lab-made PFM or full ceramic crowns are made with optimal foundation design and alignment.

Fig. 24.1 Radiographs of carious molars in patients aged 12 to 38 years. **A,** Mandibular left first molar in a 23-year-old with minor symptoms. **B,** Asymptomatic maxillary right second molar in a 16-year-old. **C,** Asymptomatic maxillary left first molar in a 38-year-old. **D,** Deep caries in the mandibular right first molar of a 12-year-old. All patients were referred to the endodontist for root canal treatment based on radiographic appearance. They exhibited normal vitality with cold testing, and all were treated successfully with vital pulp therapy.

Fig. 24.2 A 15-year-old female patient presented with full orthodontic banding and deep cervical caries of the mandibular right first molar (tooth #30). The tooth was sensitive to cold and biting pressure. **A,** Periapical radiograph revealing large carious lesion and normal periapical structures. **B,** Clinical photograph showing advanced cervical caries extending below crestal gingiva. **C,** View of initial pulp exposure during caries excavation guided by caries detector dye. **D,** Photograph after complete caries removal, large pulp exposure and 6% sodium hypochlorite hemostasis. **E,** Clinical photograph after placement of thick white mineral trioxide aggregate aliquot

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over entire pulp and surrounding dentin. **F,** Radiograph showing pulp capped molar with temporary restoration (Photocore). **G,** Four-year radiographic control showing recurrent caries and fractured distal marginal ridge tooth #30. **H,** Five-year radiographic review showing no detectable apical pathosis and recently cemented full coverage porcelain fused to metal crown. The molar was asymptomatic and responded normally to sensibility testing.
Fig. 24.3 A 24-year-old male patient presented with a deep distal carious lesion extending to the inner pulpal quarter in a maxillary right second premolar. The tooth was symptomatic but responded normally to sensibility testing. **A,** Magnified bitewing radiograph showing extension of advanced caries close to coronal pulp. **B,** Preoperative radiograph with period indentical device for precise comparison. **C,** Eighteen-month radiographic review after indirect pulp capping completed with calcium hydroxide. **D,** Five-year radiographic control. Pulp sensibility test response was positive with a well-defined lamina dura demonstrated on the periapical radiograph.
Fig. 24.4 An 7-year-old male patient referred after trauma displaying a horizontal coronal fracture of his maxillary right central incisor #8 with a pulp exposure covered with a glass ionomer cements by the general dentist and a retained deciduous left central incisor *(F)*. **A,** Clinical photograph showing horizontally fractured right maxillary incisor and retained deciduous left central incisor. **B,** Periapical radiograph reveals open apex #8 and unerupted maxillary left permanent incisor #9. In addition, a mesiodens is visible. **C,** Photograph after shallow Cvek coronal pulpotomy. **D,** View of access cavity after sodium hypochlorite hemostasis and placement of 3 to 4 mm Biodentine (Septodont, Saint-Maur-des-Fossés, France) plug. **E,** Radiograph after pulpotomy and temporary restoration placement. **F,** Clinical photograph 3 days after verified calcium silicate cements setting and reattachment of the fractured coronal segment after bonding. **G,** Radiograph of #8 with bonded crown segment reattachment showing immature open apex. **H,** Clinical photograph of full mouth banding after extraction of *(F)* and the mesiodens to permit normal eruption of tooth #9. **I,** Four-year clinical photograph with full orthodontic banding and complete eruption of #9 into the occlusal plane. Tooth #8 responded normally to cold sensibility testing at 1-, 2-, and 3-year recalls. **J.** Four-year periapical radiograph showing root-end closure of tooth #8 with normal periapical appearance. **K,** Five-year radiographic review of tooth #8 demonstrating complete radicular maturation.

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The incisor responded positive to cold testing. **L,** Clinical photograph at 5 years after finishing of a resin composite veneer created with a layering technique on tooth #8.

Fig. 24.5 A 10-year-old female was referred with a mandibular right first molar pulp exposure generated during caries excavation and temporized by the general dentist. **A,** Radiograph showing open apices with no evident apical pathosis. The tooth was diagnosed with reversible pulpitis. The patient received a new resin composite buildup to provide better isolation before a complete pulpotomy was performed. **B,** Clinical photograph of access opening through the buildup showing a sterile cotton pellet on the pulpal floor saturated with 5% sodium hypochlorite (NaOCl) for hemostasis. **C,** Photograph of radicular pulp in orifices after complete pulpotomy and 5% NaOCl hemostasis **D,** View through access opening after 5 mm thickness white mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) placement onto radicular pulp tissue and pulpal floor. **E,** Postoperative radiograph showing white MTA with wet cotton pellet and provisional restoration. One month later, MTA setting was verified and a permanent composite restoration provided. **F,** Radiographic review at 6 years shows apical maturation and thickening of the root walls. The patient remained asymptomatic and the tooth responded positively to electric pulp testing.

Fig. 24.6 Reparative bridge formation compared in dog pulps using mineral trioxide aggregate *(MTA)* and calcium hydroxide *(CH)*. **A,** Two-week pulp response to CH showing inflammatory cells *(IC)*. **B,** Tissue specimen showing lack of bridge formation and disorganized tissue proximal to CH. **C,** An 8-week specimen with partial reparative bridge *(RB)* formation subjacent to CH. **D,** Two-week pulpal response to MTA showing notable barrier formation and layer of organized odontoblast-like cells *(OLC)*. **E,** Pulp tissue section demonstrating complete calcific bridge formation proximal to MTA at 4 weeks. **F,** Sample section pulp capped with MTA at 8 weeks showing organized hard tissue formation with no inflammatory cell infiltrate.

Fig. 24.7 A 50-year-old male patient with deep recurrent caries associated with a mandibular right first molar (#30). The patient had no history of pain and responded normally to cold testing. **A,** Periapical radiograph showing distal caries in the mandibular right second premolar (#29) and large carious lesion below mesial aspect of composite restoration in the molar. Note

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retained deciduous root tip mesial totooth #29 and normal periapical appearance of #30. **B,** Pulpal hemorrhaging evident after complete caries removal with hand excavators. **C** and **D,** Clinical photographs after bleeding was controlled using cotton pellets soaked in buffered 0.5% sodium hypochlorite (NaOCl). Note presence of reparative dentin protecting mesiolingual pulp horn *(red arrow)*. A 2 mm–thick layer of white mineral trioxide aggregate (Dentsply, Tulsa Dental, Tulsa, OK) placed directly over the exposure and surrounding dentin and covered with a thin wet cotton pellet. The tooth was provisionalized for 1 week and restored permanently with composite resin. **E,** One-year radiographic follow-up; tooth responded normally to cold sensibility testing. **F,** Control radiograph at 3 years. Patient was asymptomatic and exhibited normal pulp testing response.

Fig. 24.8 Symptomatic mandibular left first molar in a 32-year-old male patient with temporary restoration placed over deep caries on the distal aspect and referred for root canal treatment. Sensibility testing elicited a normal pulpal response. **A,** Periapical film showing deep caries extending towards the distal pulp horn. **B,** Radiograph of direct mineral trioxide aggregate (Dentsply, Tulsa Dental, Tulsa, OK) pulp capping after 5.25% sodium hypochlorite hemostasis and placement of a three-surface bonded composite restoration. **C,** Nine-year 6-month radiographic control. The tooth was positive to cold testing and without symptoms. Note presence of mesial carious lesion in the second molar.

Fig. 24.9 **A,** Periapical radiograph of deep caries in a partially symptomatic mandibular left first molar in a 29-year-old female patient. **B,** Direct pulp capping completed with white mineral trioxide aggregate (MTA); the final bonded restoration was placed during the second visit after MTA curing. **C,** Radiographic recall at 7 years. Tooth was asymptomatic with normal response to CO_2 ice. **D,** Thirteen-year 10-month recall radiograph showing normal periapical appearance. The molar responded positive to cold testing and demonstrated normal mobility and periodontal probings.

Fig. 24.10 **A,** Bitewing radiograph of quadrants 2 and 3 showing defective restoration in a mandibular left first molar (#19) of a 27-year-old female patient. **B,** Clinical photograph showing pulp exposure *(white arrow)* after cavity preparation and complete caries excavation. **C,** Pulp capping and restorative base completed under slight pressure using Biodentine

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(Septodont; St. Maur-des-Fossés, France). **D,** Clinical photograph of final two-surface composite restoration placed after a setting time of 15 minutes. The restoration was placed after acid etching and bonded with a two-bottle dentin adhesive system. **E,** One-year periapical radiograph showing a normal periapical region and Biodentine radiopacity similar to that of surrounding dentin. **F,** Three-year radiographic review shows no evident pathological changes apically. The tooth registered normal responses to sensibility testing at both recall periods.

Fig. 24.11 **A,** Bitewing radiograph of quadrants 1 and 4 of an 18-year-old patient displaying a distal carious lesion in the maxillary right second premolar. The patient did not present at the scheduled appointment for restorative care. **B,** The patient returned 4 years later for treatment. Clinical photograph showing iatrogenic pulp exposure at two sites during complete caries excavation. **C,** Photograph of direct capping and base placement with Biodentine (Septodont; St. Maur-des-Fossés, France) applied to the cavity with cement pluggers using light pressure. **D,** Clinical view of cavity restored with bonded composite resin using a two-bottle dentin adhesive. **E,** Radiographic control recorded 4 years after direct capping showing normal periapical tissues. Tooth was asymptomatic with normal response to sensibility testing.

Fig. 24.12 A symptomatic 15-year-old patient presented for treatment with deep caries associated with the maxillary right first molar (#3). **A,** Preoperative radiograph showing deep caries but no evidence of apical pathosis. **B,** Clinical photograph of the carious molar. **C,** Caries removal using caries detector dye. **D,** Clinical view of pulp exposures after 5.25% sodium hypochlorite hemostasis. **E,** Periapical radiograph of direct mineral trioxide aggregate (MTA) pulp cap with wet cotton pellet and unbonded Photocore provisional. **F,** Permanent bonded composite placed 5 days after MTA direct pulp cap. **G,** Clinical photograph of final bonded composite restoration. **H,** Twenty-year 4-month recall radiograph without restorative intervention, patient was asymptomatic and responded normally to cold testing.

Fig. 24.13 A 23-year-old female patient presented with an IRM temporary restoration placed over a large pulp exposure after caries excavation of the maxillary left second molar (#15). The patient was referred for root canal treatment but had no history of pain and responded normally to sensibilty

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testing. **A,** Periapical radiograph showing temporary material in close proximity to mesial pulp horn. The tooth received a direct pulp cap with mineral trioxide aggregate (MTA) after further caries excavation and 5.25% sodium hypochlorite hemostasis. **B,** Radiograph showing direct MTA (Dentsply, Tulsa Dental, Tulsa, OK) pulp cap and bonded composite restoration placed at second visit. **C,** Two-year 5-month radiograph showing normal periapical structures and newly cemented porcelain onlay. **D,** Periapical radiographic review after 12 years and 8 months. The tooth responded positive to sensibility testing at both recall periods.

Fig. 24.14 A 12-year-old patient presented with a history of pain and cold sensitivity from a mandibular left first molar after having an occlusal composite restoration placed 16 weeks earlier. **A.** Periapical radiograph shows recurrent caries below base of the composite restoration. Note immature apices of the second molar. **B.** Clinical photograph of recently placed occlusal composite restoration. **C.** Photograph of initial caries removal showing remaining decay adjacent to pulpal roof. **D.** Photograph reveals three pulp horn exposures and presence of reparative dentin. **E.** Clinical view of three pulp horn exposures and hemostasis after application of 5% sodium hypochlorite. **F.** Photograph of bulk white mineral trioxide aggregate (MTA) (ProRoot MTA (Tulsa/Dentsply, Tulsa, OK) placement over exposures and surrounding dentin. The white MTA was covered with a flowable composite and the cavity restored with a bonded composite restoration using a two-bottle bonding system with enamel etching. **G,** One-year periapical radiograph showing normal apical structures. The tooth was asymptomatic and responded positively to vitality testing.

Fig. 24.15 Scanning electron micrographs of dentinal tubule obturation by calcium silicate cement (CSC) at the dentin/CSC interface after canals were seeded with *E. faecalis* in phosphate buffered saline (PBS). **A,** Tag formation in dentinal tubules at dentin interface in after application of OrthoMTA (BioMTA, Seoul, Korea) (×35,100). **B,** Four-week image showing *E. faecalis* entombed by growing crystalline structures within the dentinal tubule (×30,000). **C,** *White arrow* shows deformation and damaged cell membrane of *E. faecalis* within an aggregation of leaflet-like and needle-like crystals at 16 weeks (×20,000). **D,** Sixteen-week specimen showing complete blockage of the dentinal tubule lumen by CSC tight crystalline formation (×20,000).

Fig. 24.16 Eleven-year-old female presented 3 hours after trauma with a

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horizontal crown fracture of the maxillary left central incisor. **A.** Clinical photograph showing midcoronal horizontal fracture. The pulp was exposed and exhibited normal vitality. **B,** Photograph of access cavity after Cvek pulpotomy and placement of gray mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK). **C,** Photograph of fractured incisal segment before bonding and reattachment. **D,** Six-month radiograph with bonded fractured segment showing initial reparative bridge formation with absence of apical pathosis. The incisor was asymptomatic and responded normally to cold sensibility testing. **E,** Clinical photograph of incisor at 6-month recall. **F,** Eight-year radiographic review. **G,** Eleven-year radiographic recall. Tooth was asymptomatic wth normal mobility, probings, and positive response to cold test.

Fig. 24.17 Pulpotomy procedures completed for two patients clinically diagnosed with irreversible pulpitis using calcium enriched mixture (CEM) cement (BioniqueDent, Tehran, Iran). **A,** Preoperative radiograph from 27-year-old male patient with deep caries associated with maxillary right quadrant. The first molar was symptomatic with calcified tissue evident in the pulp chamber. **B,** Radiograph after complete pulpotomy using CEM cement restored with bonded composite. **C,** Five-year radiographic recall. The tooth was asymptomatic, firm, with normal probings. **D,** Preoperative radiograph showing deep caries in the mandibular left first (#19) and second (#18) molars in a 41-year-old male patient. Both teeth demonstrated lingering pain to cold testing and tooth #18 was sensitive to percussion. **E,** Postoperative radiograph showing completed miniature pulpotomy #19 and partial pulpotomy #18 after sodium hypochlorite hemostasis and application of CEM cement. Both molars were restored with bonded composites. Tooth #18 demonstrated resolution of symptoms after 24 hours. **F,** Six-year radiographic recall, patient remained asymptomatic and both teeth registered a positive response to sensibility testing.

Fig. 24.18 A 51-year-old patient who presented with a deeply carious but asymptomatic maxillary right first molar. **A,** Preoperative radiograph reveals extensive mesial caries and an occlusal amalgam. **B,** Postoperative radiograph after 1.5 mm pulpal exposure, sodium hypochlorite hemostasis, mineral trioxide aggregate (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) direct pulp cap, wet cotton pellet placement, and Photocore provisionalization. **C,** Radiograph 1 week after pulp capping and placement of a permanent bonded

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composite restoration. The patient was asymptomatic and showed a positive response to cold testing. **D,** Six-year radiographic recall; cold testing revealed normal vitality.

Fig. 24.19 Clinical examples of diseased pulp tissues after sodium hypochlorite hemostasis. **A,** Photograph of exposed pulp tissue of a mandibular right first molar in a 13-year-old patient. Note necrotic pulp tissue *(arrows)* that was subsequently removed with the remaining coronal pulp during a complete pulpotomy procedure. **B,** Clinical presentation of mandibular right molar in a 7-year-old patient after pulp exposure during caries excavation using a caries detector dye. Note the extruded seminecrotic, nonhemorrhagic tissue *(arrow)*. The tooth later underwent mineral trioxide aggregate partial pulpotomy and permanent restoration.

Fig. 24.20 Thirty-four-year-old female patient presented for root canal treatment with temporary restoration 1 week after caries excavation and pulp exposure of the maxillary right first molar (#3). The patient had mild discomfort and responded with a short lingering pain to cold testing. **A,** Periapical radiograph showing temporary restoration in tooth #3 with normal apical structures. **B,** Photograph of pulpal hemorrhage after temporary removal. **C,** Clinical photograph showing diminished bleeding after 5-minute exposure to 5.25% sodium hypochlorite. **D,** Photograph showing hemorrhage controlled in 10 minutes and 2 mm pulp exposure after continued caries excavation. The exposure and peripheral dentin received bulk OrthoMTA (BioMTA, Seoul, Korea) pulp capping and was covered with a thin wet cotton pellet and provisionalized with Photocore. The tooth was restored permanently with composite resin 1 week later. **E,** Two-year 6-month radiographic follow-up; tooth responded positive to sensibility testing. **F,** Control radiograph at 4 years. Patient exhibited normal pulp testing response and was asymptomatic with normal mobility and probings.

Fig. 24.21 An 8-year-old male presented following a traumatic injury to the maxillary left central incisor 2 months earlier. **A,** Periapical radiograph revealed an open apex with a developing radiolucency. The tooth was sensitive to palpation, percussion and negative to cold testing. The diagnosis was necrotic pulp and acute apical periodontitis. **B,** Clinical photograph after access cavity preparation, vital pulp tissue visible coronally. The diagnosis was changed to reversible pulpitis and a partial (Cvek) pulpotomy was performed. **C,** Photograph of white mineral trioxide aggregate (MTA)

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(ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) applied over the pulp to a thickness of approximately 3 mm. **D,** Radiograph after MTA pulpotomy with provisional restoration. A definitive composite restoration was placed 1 week later. **E,** One-year radiographic recall showing advancing apical closure. **F,** Four-year clinical photograph reveals a slight grey discoloration from the MTA in the cervical area of the tooth. Parents declined option of internal bleaching. **G,** Radiographic review at 9 years reveals apical maturation and thickening of the root walls. The tooth responded positively to cold sensibility testing.

Fig. 24.22 **A,** Preoperative radiograph of a 7-year-old male patient exhibiting a dens evaginatus anatomical anomaly associated with the maxillary right lateral incisor. The parents had been advised to extract the tooth, which was symptomatic and nonresponsive to vitality tests. **B,** A periapical radiograph after cleaning the coronal portion of the canal and irrigation with 2.12% sodium hypochlorite. A coronal plug using ProRoot MTA (Tulsa/Dentsply, Tulsa, OK) was placed and sealed with a provisional restoration. **C,** Four-year radiographic review demonstrates complete root formation with maturation and apical root closure. The tooth was asymptomatic with normal mobility and probings.

Fig. 25.1 Endodontic and periodontal pathways. **A,** Endodontic lesions. The pathway of inflammation is through the apical foramen, furcation canals, and lateral accessory canals to the periodontium. This results in a primary endodontic lesion, sometimes progressing to secondary periodontal involvement. **B,** Periodontal lesions. This is the progression of periodontitis by way of the lateral canal and apex to induce a secondary endodontic lesion. **C,** True combined endodontic and periodontal lesion and concomitant endodontic and periodontal lesions.

Fig. 25.2 Primary endodontic lesion. Mandibular molar showing endodontic filling material extending into the furcation and along the lateral root surface because of inadequate canal preparation. Furcation could be probed.

Fig. 25.3 Primary endodontic lesion on mandibular first molar. **A,** Mandibular first molar with deep restoration and pulpal necrosis; apical radiolucency on mesial and distal roots with furcation radiolucency. **B,** Probing of 12 mm with periodontal probe. **C,** X-ray image of completed root canal treatment. **D**, Eight months after root canal treatment, there is evidence of osseous healing at both the apex and furcation area.

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Fig. 25.4 Primary endodontic lesion with secondary periodontal involvement. **A,** The mesial aspect of the mandibular second premolar had deep periodontal pocketing (a periodontal probe in place demonstrated 6 mm of probing depth), even after periodontal therapy. **B,** Pulp tests were performed on the premolar; the tooth was nonresponsive. After endodontic treatment, the periodontal pocket resolved (filling of a lateral accessory canal after obturation is demonstrated).

Fig. 25.5 Primary periodontal lesion. **A,** Mandibular cuspid exhibits extensive periodontal destruction; the tooth responded normally to pulp tests. **B,** Extracted tooth depicts extensive calculus accumulation and root concavity.

Fig. 25.6 True combined pulpal and periodontal lesions on the mandibular second premolar and first molar. Periodontal probing depths were to the apices in both teeth.

Fig. 25.7 Concomitant pulpal and periodontal lesion on the maxillary second premolar. An endodontic lesion was noted at the apex, with a noncommunicating periodontal pocket on the distal side.

Fig. 25.8 Vertical root fracture. **A,** A radiograph revealed a widened periodontal ligament with a J-shaped radiolucency around the apex. **B,** A periodontal probe indicated more than 12 mm of probing depth. **C,** Exploratory surgery confirmed a vertical root fracture.

Fig. 25.9 Cemental tear. **A,** Radiographs revealed an endodontically treated tooth with post, separated instrument, and apical osseous breakdown; the tooth was very tender to both percussion and palpation. **B,** An “eggshell” piece of cementum on the buccal surface of the root before root-end resection; the cementum was removed with curettage. **C,** Postoperative radiographs after root-end resection, root-end preparation, and root-end filling. **D,** Six-month follow-up radiographs demonstrate periapical healing; the tooth was asymptomatic.

Fig. 25.10 Palatogingival groove on a maxillary lateral incisor with a periodontal defect.

Fig. 25.11 Cervical enamel projection on a mandibular molar.

Fig. 25.12 Lateral periodontal cyst. **A,** Clinical photograph showing mild gingivitis around tooth #24; there is a 5-mm probing defect on the mesial aspect of tooth #24. **B,** A radiograph depicts the beginning lesion 2 years earlier. **C,** A radiograph shows the extent of the lesion at the time of referral

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to the periodontist. **D,** Initial flap reflection; the lesion is present interproximally between teeth #24 and #25. **E,** The lesion was enucleated and submitted for histologic evaluation; the diagnosis was a lateral periodontal cyst. **F,** Three-month posttreatment clinical photograph; tooth #24 was nonvital and symptomatic and was referred for root canal treatment. **G,** One-year posttreatment radiograph showing completed root canal treatment and osseous defect still present; continuous recall recommended. **H,** One-year posttreatment clinical photograph; probing depths were within normal limits; continuous recall was recommended.

Fig. 25.13 Root resection to correct endodontic and periodontal defect. **A,** Pretreatment radiograph depicts severe periodontal involvement on the mesial. Endodontic treatment was performed before surgical removal of the mesial root. **B,** Five-year follow-up shows the remaining tooth well maintained.

Fig. 25.14 Hemisection. **A,** Initial presentation of mandibular molars with severe attachment loss on the mesial aspect of tooth #31 and the distal aspect of tooth #30. **B,** Radiograph exhibiting interproximal periodontal defects. **C,** Occlusal view of hemisection after endodontic treatment. **D,** Suturing after removal of mesial half of tooth #31 and distal half of tooth #30. **E,** Eighteen-month follow-up with fixed restoration in place. **F,** Postoperative radiograph at 18 months showing normal bony and periodontal architecture.

Fig. 25.15 Periradicular endodontic surgery using the combination of bone replacement grafts and collagen guided tissue regeneration barrier membranes. **A,** A buccal flap was reflected (note extensive apical lesion). **B,** An osseous window was created with a bur. **C,** The defect was repaired with demineralized freeze-dried bone allograft and covered with a collagen membrane. **D,** Radiographs show condition before surgery, at the time of surgery, and at 6 months and 2 years after surgery. At 2 years, the radiograph reveals complete bone regeneration.

Fig. 25.16 Periodontal surgery using guided bone regeneration. **A,** A pretreatment radiograph demonstrates a significant osseous defect on the mesial aspect of tooth #14. **B,** Pretreatment clinical photograph of teeth #12 to #16. **C,** A full-thickness mucoperiosteal flap reflected with a periodontal probe 7 mm into osseous defect. **D,** Osseous defect filled with BioOss (cancellous bovine bone). Root canal treatment (RCT) was started for the patient before the surgical appointment; RCT was completed 6 weeks after

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osseous graft placement. **E,** Ossix membrane (collagen) placed over the BioOss graft. **F,** One-year posttreatment radiograph with evidence of osseous repair. **G,** One-year posttreatment clinical photograph of area.

Fig. 25.17 Clinical case of forced eruption. **A,** Initial presentation of tooth #29 (buccal view). **B,** Bonded wire to help extrude tooth #29. **C,** Lingual view of tooth #29 after 3 weeks of extrusion. **D,** Lingual view of cemented porcelain crown on tooth #29; 4 months after extrusion and after 1 month of crown retention.

Fig. 26.1 Clinical case of a 19-year-old female patient illustrating discolorations from endodontic filling materials. **A,** Preoperative situation with history of endodontic treatment on both maxillary lateral incisors. **B and C,** Preoperative radiographs prior to nonsurgical retreatment and bleaching procedures. **D,** Postoperative situation after endodontic retreatment, bleaching procedures and new coronal restorations. Shades now match the maxillary arch with A2 (shade guide not shown). **E and F,** Postoperative radiographic control of procedures ca. 4 weeks after completion of endodontic treatment and new restorations.

Fig. 26.2 Chemistry of tooth whitening with hydrogen peroxide. Breakdown of pigmented organic materials consisting of carbon ring structures with unsaturated, double carbon bonds to hydrophilic, nonpigmented carbon structures with saturated carbon bonds, resulting in effective whitening. Further degradation over this step may complete disintegration of the organic matrix with the release of carbon dioxide and water.

Fig. 26.3 Walking bleach technique: Placement of barrier material at the cementoenamel junction *(CEJ)* level, bleaching agent, and coronal seal with temporary filling material.

Fig. 26.4 Clinical case illustrating the walking bleach technique. **A,** Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left canine due to pulp necrosis. **B,** Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C,** Preoperative situation. **D,** Control radiograph after application of sodium perborate for the walking bleach technique. **E,** Illustration of part D showing placement of barrier material at the cementoenamel junction *(CEJ)* level, bleaching agent, and coronal seal with temporary filling material. **F,** Postoperative situation after bleaching, definitive coronal seal with adhesive restoration.

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Fig. 26.5 Clinical case illustrating clinical steps of the walking bleach technique. **A,** Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left central incisor due to dental trauma. **B,** Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C,** Access cavity after barrier placement. **D,** Application of sodium perborate. **E,** Temporary coronal seal with adhesive restoration.
Fig. 26.6 Clinical case of a 33-year-old female patient illustrating extensive resorptive defects after nonvital bleaching efforts. **A,** Postoperative situation after endodontic treatments of the maxillary right canine and first premolar. The patient was referred back to the primary care provider for definitive restorations. **B,** Situation 12 months after the original endodontic treatments. The patient had spent 1 year overseas and received endodontic treatment on the second premolar, root-end surgery on the canine, as well as nonvital bleaching of the maxillary right canine and first premolar. Note the extensive resorptive defects on both teeth that received bleaching procedures and the missing restoration on the canine. **C** and **D,** Coronal CBCT views of canine and first premolar. Note location and dimensions of the resorptions *(arrows)*.
Fig. 27.1 Patient information form.
Fig. 27.2 Medical history form.
Fig. 27.3 Dental history record.
Fig. 27.4 **A,** Nail inside cranium? **B,** Nail in same patient as in **(A)**, showing nail taped to outside of skull. Alas, poor Yorick!
Fig. 27.5 Diagnosis and treatment form.
Fig. 27.6 Medication log.
Fig. 27.7 True/false endodontic informed consent form.
Fig. 27.8 **A,** Progress notes. **B,** Diagnosis and treatment form with notes.
Fig. 27.9 **A,** Distal open margin on tooth #30 (shown digitally). **B,** Distal open margin on tooth #30 not evident with plain-film radiography.
Fig. 27.10 **A,** Periapical (PA) view, which does not show full extent of transported and short-filled canal. Both deficiencies evident in **B** and **C** with cone-beam computed tomography (CBCT). **B,** CBCT Accuitomo of transported canal. **C,** CBCT Accuitomo of short-filled canal.
Fig. 27.11 **A,** Bifid inferior alveolar nerve canal (IANC) visible with cone-beam computed tomography (CBCT). **B,** Bifid canal visible with CBCT but not shown on Panorex. **C,** Branch of IANC apparent on CBCT but not shown

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on Panorex.
Fig. 27.12 Sample of written physician recommendations**.**
Fig. 27.13 Clinician and staff initials log.
Fig. 27.14 Postoperative instruction sheet.
Fig. 27.15 Belated record correction.
Fig. 27.16 A, Falsified records with belated entry of year of referral. **B,** Falsified records.
Fig. 27.17 American Association of Endodontists (AAE) Endodontic Case Difficulty Assessment Form and Guidelines.
Fig. 27.18 A general clinician's radiograph that ostensibly shows a complete endodontic fill within the root canal space.
Fig. 27.19 Endodontist's radiograph demonstrating a transported canal.
Fig. 27.20 Implant "harpooned" inferior alveolar nerve canal.
Fig. 27.21 Results of overheating a post with an ultrasonic device during post removal.
Fig. 27.22 Excessive local anesthetic resulting in palatal tissue sloughing with anterior middle superior alveolar (AMSA) (wand) injection.
Fig. 27.23 A, Preoperative radiograph. **B,** Measuring film without a rubber dam. **C,** Postoperative radiograph. **D,** Decompression microsurgery showing protruding "dagger" of bone to right of retractor at inferior border of inferior alveolar nerve, resulting from prior perforation of the mandibular canal. This is probably due to file overinstrumentation.
Fig. 27.24 Informed consent form.
Fig. 27.25 A, Preoperative panoramic radiograph showing a successful apicoectomy and retrograde seal. **B,** Postoperative panoramic radiograph showing a successful apicoectomy and retrograde seal.
Fig. 27.26 A, Nickel-titanium file broken inside the mesiobuccal canal of the mandibular first molar. **B,** Due to patient discomfort, the segment was surgically removed and mineral trioxide aggregate (MTA) was used as root-filling material. **C,** Periapical radiograph 2½ years posttreatment shows complete healing.
Fig. 27.27 A, Before injury. **B–D,** A clarinetist music student was burned by an inadequately maintained handpiece that overheated. This lawsuit was settled for $280,000.
Fig. 27.28 A, Unclear periapical radiograph shows both cortical borders of inferior alveolar nerve canal (IANC). **B,** Cones placed into IANC, showing

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measurements beyond apices. **C,** Overfill into IANC better shown on cone-beam computed tomography (CBCT) than periapicals of **A** and **B.**
Fig. 27.29 **A,** Tooth #31 with three separated files, one in each canal. K-files used for cleaning canals and also obturation (1996). **B,** No apparent radiographic changes in 1998.
Fig. 27.30 **A,** Apical radiolucent changes with draining 9-mm buccal abscess (August 2003). **B,** Endodontist retreatment (August 2003). **C,** Successful retreatment with no buccal pocket (September 2003).
Fig. 27.31 Swallowed endodontic instrument demonstrates the wisdom of using a dental dam.
Fig. 27.32 **A–B,** Calcium hydroxide overfill, causing permanent paresthesia.
Fig. 27.33 **A-C,** Overextended gutta-percha and extracted tooth with gutta-percha intact.
Fig. 27.34 **A,** Illustration showing implant placed into ascending inferior alveolar nerve canal (IANC). **B,** Cone-beam computed tomography (CBCT) image of implant into ascending IANC. Settlement of $915,000. **C,** Implant into IANC; immediate postoperative Panorex image. Same patient but with CBCT image.
Fig. 27.35 Medicolegal implant image.
Fig. 27.36 Gross overfill into sinus with a silver point, which ultimately caused sinusitis and loss of tooth #14 as a result of endodontic failure.
Fig. 27.37 **A** and **B,** Overextensions of Sargenti paste filling the inferior alveolar nerve canal. Both cases could have been avoided if the practitioners had selected a conventional sealing material and used a technique that emphasizes working length control.
Fig. 27.38 Implant “harpooned” the inferior alveolar nerve canal.
Fig. 27.39 Avoidable perforation.
Fig. 27.40 Post perforation of tooth #5.
Fig. 27.41 Bleach injury.
Fig. 27.42 **A,** Periapical tooth #19 showing fractured file but no perforation. **B,** Accuitomo cone-beam computed tomography (CBCT) showing perforation on the lingual side above the tooth #19 furcation. Perforation not evident in periapical view. Image demonstrates Accuitomo accuracy. **C,** Bleach extrusion through tooth #19 perforation, with resultant slough of lingual tissue.
Fig. 27.43 Tensile overload shown in elongated ductile dimple voids.

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Fig. 27.44 Needle blunting of broken needle tip demonstrating striking hard object (ramus).
Fig. 27.45 **A,** Needle fracture with scanning electron microscopy (SEM). **B,** Needle fracture, side view (SEM). **C,** Needle fracture at high SEM magnification.
Fig. 27.46 Cone-beam computed tomography usage for implant placement or apical surgery.
Fig. 27.47 Dental auxiliary confidentiality agreement.
Fig. 28.1 The keys to achieving practice management success.
Fig. 28.2 The impact of referral marketing. *Doctor A* does not have a formal referral marketing program in place. *Doctor B* does have a formal referral marketing program in place. The results are dramatic. *Doctor B* has many more referring doctors, who refer more patients for treatment.
Fig. 28.3 Key principles of endodontic practice management. *KPIs,* Key production indicators.

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PART I
The Core Science of Endodontics

OUTLINE

1. Diagnosis
2. Radiographic interpretation
3. Lesions that mimic endodontic pathosis
4. Diagnosis of the nonodontogenic toothache
5. Case assessment and treatment planning
6. Pain control
7. Tooth morphology and pulpal access cavities
8. Cleaning and shaping of the root canal system
9. Obturation of the cleaned and shaped root canal system
10. Nonsurgical retreatment
11. Periradicular surgery
12. Regenerative endodontics

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1: Diagnosis

Louis H. Berman, Ilan Rotstein

CHAPTER OUTLINE

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Art and science of diagnosis

Diagnosis is the art and science of detecting and distinguishing deviations from health and the cause and nature thereof.[6] The purpose of a diagnosis is to determine what problem the patient is having and why the patient is having that problem. Ultimately, this will directly relate to what treatment, if any, will be necessary. No appropriate treatment recommendation can be made until all of the *whys* are answered. Therefore, careful data gathering as well as a planned, methodical, and systematic approach to this investigatory process is crucial.

Gathering objective data and obtaining subjective findings are not enough to formulate an accurate clinical diagnosis. The data must be interpreted and processed to determine what information is significant, and what information might be questionable. The facts need to be collected with an active dialogue between the clinician and the patient, with the clinician asking the right questions and carefully interpreting the answers. In essence, the process of determining the existence of an oral pathosis is the culmination of the art and science of making an accurate diagnosis.

The process of making a diagnosis can be divided into five stages:

1. The patient tells the clinician the reasons for seeking advice.
2. The clinician questions the patient about the symptoms and history that led to the visit.
3. The clinician performs objective clinical tests.
4. The clinician correlates the objective findings with the subjective details and creates a tentative list of differential diagnoses.
5. The clinician formulates a definitive diagnosis.

This information is accumulated by means of an organized and systematic approach that requires considerable clinical judgment. The clinician must be able to approach the problem by crafting what questions to ask the patient and how to ask these pertinent questions. Careful listening is paramount to begin painting the picture that details the patient’s complaint. These subjective findings combined with the results of diagnostic tests provide the critical information needed to establish the diagnosis.

Neither the art nor the science is effective alone. Establishing a differential

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diagnosis in endodontics requires a unique blend of knowledge, skills, and the ability to interpret and interact with a patient in real time. Questioning, listening, testing, interpreting, and finally answering the ultimate question of *why* will lead to an accurate diagnosis and in turn result in a more successful treatment plan.

Chief complaint

On arrival for a dental consultation, the patient should complete a thorough registration that includes information pertaining to medical and dental history (Figs. 1.1 and 1.2). This should be signed and dated by the patient, as well as initialed by the clinician as verification that all of the submitted information has been reviewed (see Chapter 27 for more information).

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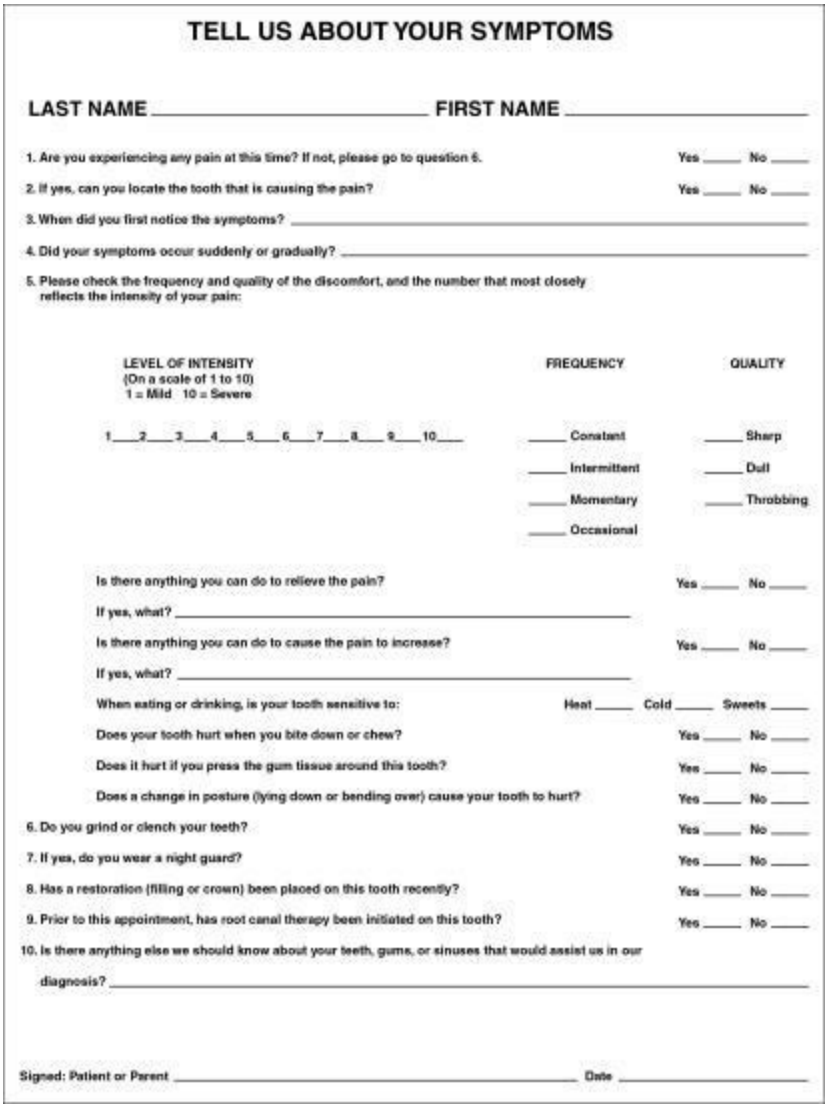

TELL US ABOUT YOUR SYMPTOMS

LAST NAME _____ **FIRST NAME** _____

1. Are you experiencing any pain at this time? If not, please go to question 6. Yes ____ No ____
2. If yes, can you locate the tooth that is causing the pain? Yes ____ No ____
3. When did you first notice the symptoms? _____
4. Did your symptoms occur suddenly or gradually? _____
5. Please check the frequency and quality of the discomfort, and the number that most closely reflects the intensity of your pain:

LEVEL OF INTENSITY (On a scale of 1 to 10) 1 = Mild 10 = Severe	FREQUENCY	QUALITY
1___2___3___4___5___6___7___8___9___10___	____ Constant	____ Sharp
	____ Intermittent	____ Dull
	____ Momentary	____ Throbbing
	____ Occasional	

Is there anything you can do to relieve the pain? Yes ____ No ____

If yes, what? _____

Is there anything you can do to cause the pain to increase? Yes ____ No ____

If yes, what? _____

When eating or drinking, is your tooth sensitive to: Heat ____ Cold ____ Sweets ____

Does your tooth hurt when you bite down or chew? Yes ____ No ____

Does it hurt if you press the gum tissue around this tooth? Yes ____ No ____

Does a change in posture (lying down or bending over) cause your tooth to hurt? Yes ____ No ____

6. Do you grind or clench your teeth? Yes ____ No ____
7. If yes, do you wear a night guard? Yes ____ No ____
8. Has a restoration (filling or crown) been placed on this tooth recently? Yes ____ No ____
9. Prior to this appointment, has root canal therapy been initiated on this tooth? Yes ____ No ____
10. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis? _____

Signed: Patient or Parent _____ Date _____

FIG. 1.1 Dental history form that also allows the patient to record pain experience in an organized and descriptive manner.

Dental history form, titled “tell us about your symptoms” is as follows:

Last name (followed by blank space) first name (followed by blank space).

1. Are you experiencing any pain at this time? If not, please go to question 6. (Yes or No)

2. If yes, can you locate the tooth that is causing the pain? (Yes or No)

3. When did you first notice the symptoms? Blank space

4. Did your symptoms occur suddenly or gradually? Blank space

5. Please check the frequency and quality of the discomfort, and the number that most closely

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Level of intensity (On a scale of 1 to 10) 1 = Mild 10 = Severe

Frequency: Constant, intermittent, momentary, occasional. (Each with a blank space)

Quality: Sharp, dull, throbbing. (Each with a blank space)

Is there anything you can do to relieve the pain? (Yes or No)

If yes, what? Blank space

Is there anything you can do to cause the pain to increase? (Yes or No)

If yes, what? Blank space

When eating or drinking, is your tooth sensitive to: heat, cold, sweets (each with a blank space)

Does your tooth hurt when you bite down or chew? (Yes or No)

Does it hurt if you press the gum tissue around this tooth? (Yes or No)

Does a change in posture (lying down or bending over) cause your tooth to hurt? (Yes or No)

6. Do you grind or clench your teeth? (Yes or No)

7. If yes, do you wear a night guard? (Yes or No)

8. Has a restoration (filling or crown) been placed on this tooth recently? (Yes or No)

9. Prior to this appointment, has root canal therapy been initiated on this tooth? (Yes or No)

10. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis? (blank space)

Signed: patient or parent (blank space), Date (blank space).

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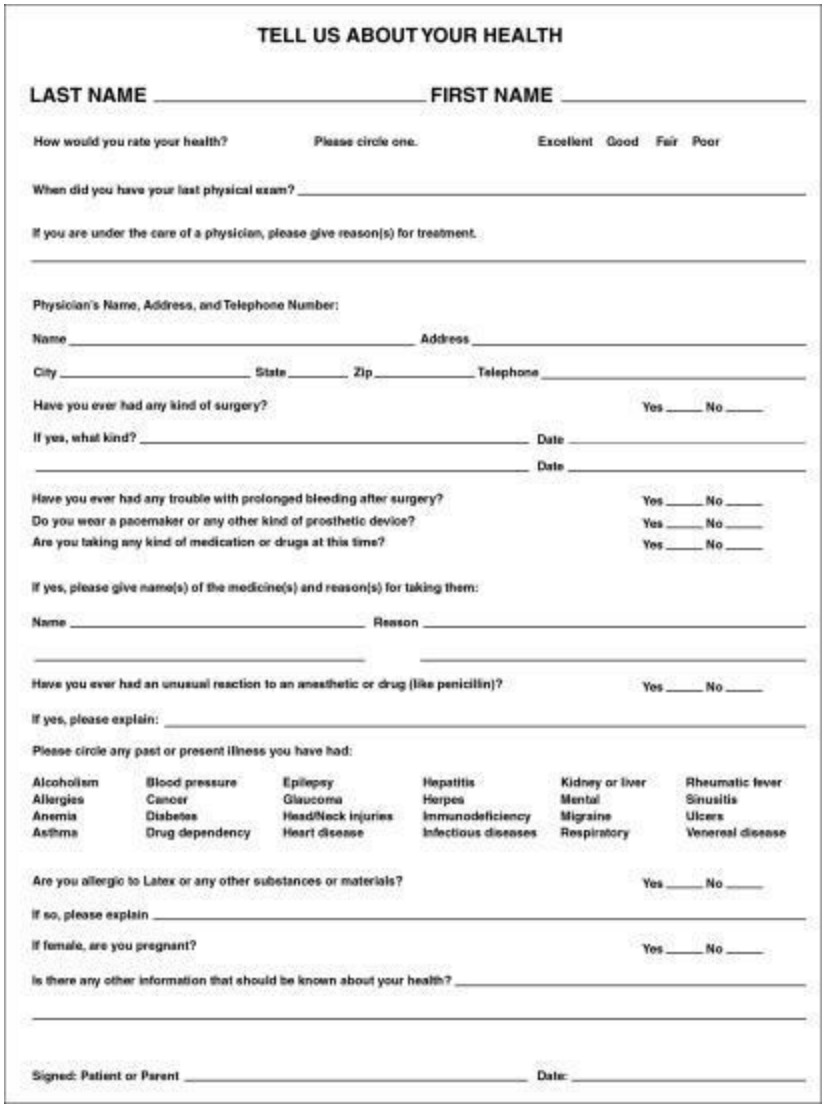

TELL US ABOUT YOUR HEALTH

LAST NAME _____ FIRST NAME _____

How would you rate your health? Please circle one. Excellent Good Fair Poor

When did you have your last physical exam? _____

If you are under the care of a physician, please give reason(s) for treatment.

_____

Physician's Name, Address, and Telephone Number:

Name _____ Address _____

City _____ State _____ Zip _____ Telephone _____

Have you ever had any kind of surgery? Yes ____ No ____

If yes, what kind? _____ Date _____

_____ Date _____

Have you ever had any trouble with prolonged bleeding after surgery? Yes ____ No ____

Do you wear a pacemaker or any other kind of prosthetic device? Yes ____ No ____

Are you taking any kind of medication or drugs at this time? Yes ____ No ____

If yes, please give name(s) of the medicine(s) and reason(s) for taking them:

Name _____ Reason _____

_____ _____

Have you ever had an unusual reaction to an anesthetic or drug (like penicillin)? Yes ____ No ____

If yes, please explain: _____

Please circle any past or present illness you have had:

Alcoholism	Blood pressure	Epilepsy	Hepatitis	Kidney or liver	Rheumatic fever
Allergies	Cancer	Glaucoma	Herpes	Mental	Sinusitis
Anemia	Diabetes	Head/Neck injuries	Immunodeficiency	Migraine	Ulcers
Asthma	Drug dependency	Heart disease	Infectious diseases	Respiratory	Venereal disease

Are you allergic to Latex or any other substances or materials? Yes ____ No ____

If so, please explain _____

If female, are you pregnant? Yes ____ No ____

Is there any other information that should be known about your health? _____

_____

Signed: Patient or Parent _____ Date: _____

FIG. 1.2 Succinct, comprehensive medical history form designed to provide insight into systemic conditions that could produce or affect the patient's symptoms, mandate alterations in treatment modality, or change the treatment plan.

Form, titled tells us about your health is as follows:

Last name (blank space) first name (blank space)

How would you rate your health? Please circle one from excellent, good, fair, and poor

When did you have your last physical exam? (blank space)

If you are under the care of a physician, please give reason(s) for treatment. (blank space)

Physician's Name, address, and telephone Number:

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Name, address, city, state, zip, telephone (each with a blank space)

Have you ever had any kind of surgery? (Yes or No)

If yes, what kind? Blank space, date (blank space), date (blank space).

Have you ever had any trouble with prolonged bleeding after surgery? (Yes or No)

Do you wear a pacemaker or any other kind of prosthetic device? (Yes or No)

Are you taking any kind of medication or drugs at this time? (Yes or No)

If yes, please give name(s) of the medicine(s) and reason(s) for taking them: Name (blank space) reason (blank space)

Have you ever had an unusual reaction to an anesthetic or drug (like penicillin)? (Yes or No)

If yes, please explain: (blank space)

Please circle any past or present illness you have had: Alcoholism, Allergies, Anemia, Asthma, Blood pressure, Cancer, Diabetes, Drug dependency, Epilepsy, Glaucoma, Head/Neck injuries, Heart disease, Hepatitis, Herpes, Immunodeficiency, Infectious diseases, Kidney or liver, Mental, Migraine, Respiratory, Rheumatic fever, Sinusitis, Ulcers, Venereal disease.

Are you allergic to Latex or any other substances or materials? (Yes or No)

If so, please explain (Yes or No)

If female, are you pregnant? (Yes or No)

Is there any other information that should be known about your health? Blank space

Signed: Patient or Parent: blank space, Date blank space.

The reasons patients give for consulting with a clinician are often as important as the diagnostic tests performed. Their remarks serve as initial important clues that will help the clinician to formulate a correct diagnosis. Without these direct and unbiased comments, objective findings may lead to an incorrect diagnosis. The clinician may find a dental pathosis, but it may not contribute to the pathologic condition that mediates the patient's chief complaint. Investigating these complaints may indicate that the patient's concerns are related to a medical condition or to recent dental treatment.

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Certain patients may even receive initial emergency treatment for pulpal or periapical symptoms in a general hospital.[93] On occasion, the chief complaint is simply that another clinician correctly or incorrectly advised the patient that he or she had a dental problem, with the patient not necessarily having any symptoms or any objective pathosis. Therefore, the clinician must pay close attention to the actual expressed complaint, determine the chronology of events that led to this complaint, and question the patient about other pertinent issues, including medical and dental history. For future reference and in order to ascertain a correct diagnosis, the patient's chief complaint should be properly documented, using *the patient's own words.*

Medical history

The clinician is responsible for taking a proper medical history from every patient who presents for treatment. Numerous examples of medical history forms are available from a variety of sources, or clinicians may choose to customize their own forms. After the form is completed by the patient, or by the parent or guardian in the case of a minor, the clinician should review the responses with the patient, parent, or guardian, and then initial the medical history form to indicate that this review has been done. The patient "of record" should be questioned at each treatment visit to determine whether there have been any changes in the patient's medical history or medications. A more thorough and complete update of the patient's medical history should be taken if the patient has not been seen for over a year.[51,52]

Baseline blood pressure and pulse should be recorded for the patient at each treatment visit. Elevation in blood pressure or a rapid pulse rate may indicate an anxious patient who may require a stress reduction protocol, or it may indicate that the patient has hypertension or other cardiovascular health problems. Referral to a physician or medical facility may be indicated. It is imperative that vital signs be gathered at each treatment visit for any patient with a history of major medical problems. The temperature of patients presenting with subjective fever or any signs or symptoms of a dental infection should be taken.[57,80,105]

The clinician should evaluate a patient's response to the health questionnaire from two perspectives: (1) those medical conditions and current medications that will necessitate altering the manner in which dental care will

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be provided and (2) those medical conditions that may have oral manifestations or mimic dental pathosis.

Patients with serious medical conditions may require either a modification in the manner in which the dental care will be delivered or a modification in the dental treatment plan (Box 1.1). In addition, the clinician should be aware if the patient has any drug allergies or interactions, allergies to dental products, an artificial joint prosthesis, organ transplants, or is taking medications that may negatively interact with common local anesthetics, analgesics, sedatives, and antibiotics.[80] This may seem overwhelming, but it emphasizes the importance of obtaining a thorough and accurate medical history while considering the various medical conditions and dental treatment modifications that may be necessary before dental treatment is provided.

Box 1.1

Medical Conditions That Warrant Modification of Dental Care or Treatment

Cardiovascular: High- and moderate-risk categories of endocarditis, pathologic heart murmurs, hypertension, unstable angina pectoris, recent myocardial infarction, cardiac arrhythmias, poorly managed congestive heart failure.[57,80,105]

Pulmonary: Chronic obstructive pulmonary disease, asthma, tuberculosis.[80,129]

Gastrointestinal and renal: End-stage renal disease; hemodialysis; viral hepatitis (types B, C, D, and E); alcoholic liver disease; peptic ulcer disease; inflammatory bowel disease; pseudomembranous colitis.[25,34,48,80]

Hematologic: Sexually transmitted diseases, human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS), diabetes mellitus, adrenal insufficiency, hyperthyroidism and hypothyroidism, pregnancy, bleeding disorders, cancer and leukemia, osteoarthritis and rheumatoid arthritis, systemic lupus erythematosus.[35,43,76,80,83,88,100,135]

Neurologic: Cerebrovascular accident, seizure disorders, anxiety, depression and bipolar disorders, presence or history of drug or alcohol abuse, Alzheimer disease, schizophrenia, eating disorders, neuralgias, multiple sclerosis, Parkinson disease.[36,44,80]

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Several medical conditions have oral manifestations, which must be carefully considered when attempting to arrive at an accurate dental diagnosis. Many of the oral soft-tissue changes that occur are more related to the medications used to treat the medical condition rather than to the condition itself. More common examples of medication side effects are stomatitis, xerostomia, petechiae, ecchymoses, lichenoid mucosal lesions, and bleeding of the oral soft tissues.[80]

When developing a dental diagnosis, a clinician must also be aware that some medical conditions can have clinical presentations that mimic oral pathologic lesions.[13,28,32,74,80,102,107,133] For example, tuberculosis involvement of the cervical and submandibular lymph nodes can lead to a misdiagnosis of lymph node enlargement secondary to an odontogenic infection. Lymphomas can involve these same lymph nodes.[80] Immunocompromised patients and patients with uncontrolled diabetes mellitus respond poorly to dental treatment and may exhibit recurring abscesses in the oral cavity that must be differentiated from abscesses of dental origin.[43,76,80,83] Patients with iron deficiency anemia, pernicious anemia, and leukemia frequently exhibit paresthesia of the oral soft tissues. This finding may complicate making a diagnosis when other dental pathosis is present in the same area of the oral cavity. Sickle cell anemia has the complicating factor of bone pain, which mimics odontogenic pain, and loss of trabecular bone pattern on radiographs, which can be confused with radiographic lesions of endodontic origin. Multiple myeloma can result in unexplained mobility of teeth. Radiation therapy to the head and neck region can result in increased sensitivity of the teeth and osteoradionecrosis.[80] Trigeminal neuralgia, referred pain from cardiac angina, and multiple sclerosis can also mimic dental pain (see also Chapter 4). Acute maxillary sinusitis is a common condition that may create diagnostic confusion because it may mimic tooth pain in the maxillary posterior quadrant. In this situation the teeth in the quadrant may be extremely sensitive to cold and percussion, thus mimicking the signs and symptoms of pulpitis. This is certainly not a complete list of all the medical entities that can mimic dental disease, but it should alert the clinician that a medical problem could confuse and complicate the diagnosis of dental pathosis; this issue is discussed in more detail in subsequent chapters.

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If, at the completion of a thorough dental examination, the subjective, objective, clinical testing, and radiographic findings do not result in a diagnosis with an obvious dental origin, then the clinician must consider that an existing medical problem could be the true source of the pathosis. In such instances, a consultation with the patient's physician is always appropriate.

Dental history

The chronology of events that lead up to the chief complaint is recorded as the *dental history*. This information will help guide the clinician as to which diagnostic tests are to be performed. The history should include any past and present symptoms, as well as any procedures or trauma that might have evoked the chief complaint. Proper documentation is imperative. It may be helpful to use a premade form to record the pertinent information obtained during the dental history interview and diagnostic examination. Often a SOAP format is used, with the history and findings documented under the categories of *S*ubjective, *O*bjective, *A*ppraisal, and *P*lan. There are also built-in features within some practice management software packages that allow digital entries into the patient's electronic file for the diagnostic workup (Figs. 1.3 and 1.4).

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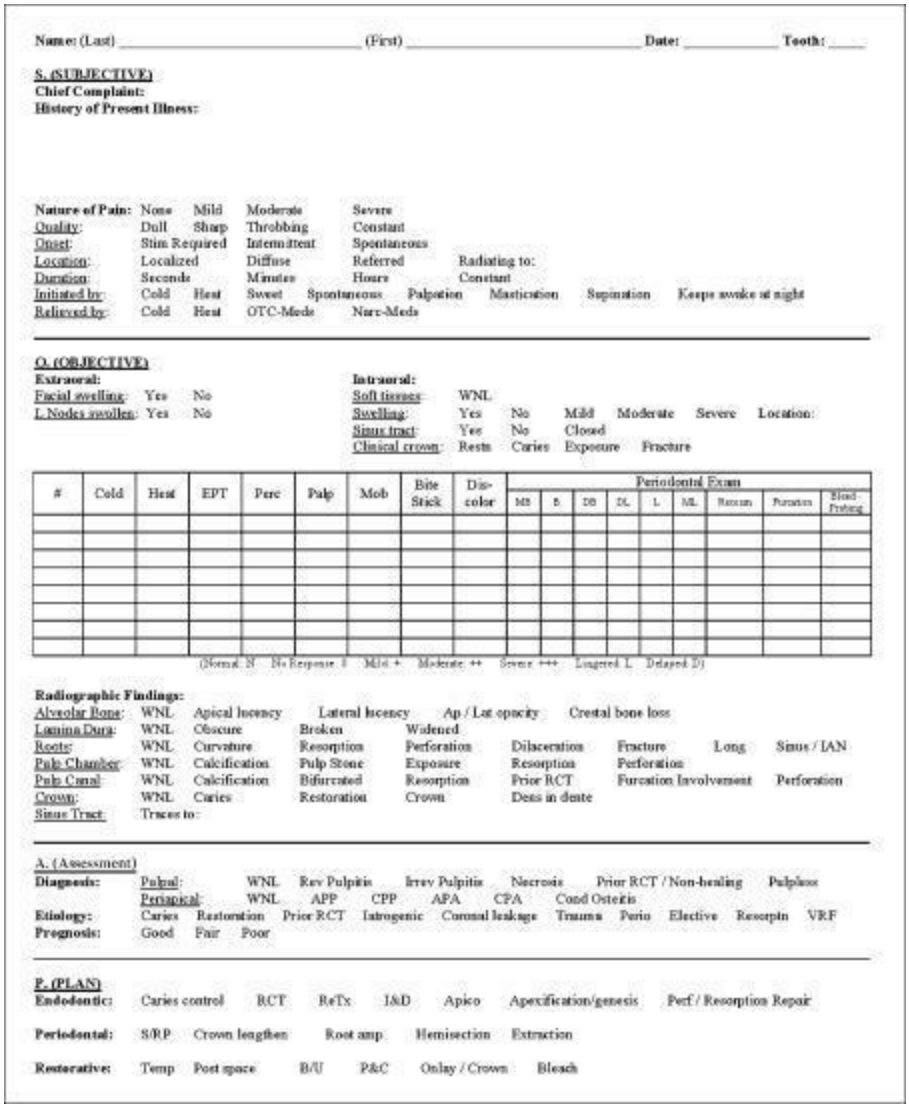

Name: (Last) _____ (First) _____ Date: _____ Tooth: _____

S. (SUBJECTIVE)
Chief Complaint:
History of Present Illness:

Nature of Pain: None Mild Moderate Severe
Quality: Dull Sharp Throbbing Constant
Onset: Stim Required Intermittent Spontaneous
Location: Localized Diffuse Referred Radiating to:
Duration: Seconds Minutes Hours Constant
Initiated by: Cold Heat Sweet Spontaneous Palpation Mastication Supination Keeps awake at night
Relieved by: Cold Heat OTC-Meds Narc-Meds

O. (OBJECTIVE)
Extraoral:
Facial swelling: Yes No
L Nodes swollen: Yes No

Intraoral:
Soft tissues: WNL
Swelling: Yes No Mild Moderate Severe Location:
Sinus tract: Yes No Closed
Clinical crown: Resto Caries Exposure Fracture

#	Cold	Heat	EPT	Perc	Palp	Mob	Bite Stick	Dis-color	Periodontal Exam								
									MB	B	DB	DL	L	ML	Recess	Furcation	Blood Probing

(Normal: N No Response: 0 Mild: + Moderate: ++ Severe: +++ Lingered: L Delayed: D)

Radiographic Findings:
Alveolar Bone: WNL Apical lucency Lateral lucency Ap / Lat opacity Crestal bone loss
Lamina Dura: WNL Obscure Broken Widened
Roots: WNL Curvature Resorption Perforation Dilaceration Fracture Long Sinus / IAN
Pulp Chamber: WNL Calcification Pulp Stone Exposure Resorption Perforation
Pulp Canal: WNL Calcification Bifurcated Resorption Prior RCT Furcation Involvement Perforation
Crown: WNL Caries Restoration Crown Dens in dente
Sinus Tract: Traces to:

A. (Assessment)
Diagnosis: Pulpal: WNL Rev Pulpitis Irrev Pulpitis Necrosis Prior RCT / Non-healing Pulpless
Periapical: WNL APP CPP APA CPA Cond Osteitis
Etiology: Caries Restoration Prior RCT Iatrogenic Coronal leakage Trauma Perio Elective Resorptn VRF
Prognosis: Good Fair Poor

P. (PLAN)
Endodontic: Caries control RCT ReTx I&D Apico Apexification/genesis Perf / Resorption Repair

Periodontal: S/RP Crown lengthen Root amp Hemisection Extraction

Restorative: Temp Post space B/U P&C Onlay / Crown Bleach

FIG. 1.3 When taking a dental history and *performing* a diagnostic examination, often a premade form can facilitate complete and accurate documentation.

A dental history form shows blank space for patient's last name and first name along with date and tooth. It is followed by chief complaint and history of present illness under the “S. (subjective)” heading. Thereafter, the different options for nature of pain are shown, followed by O. (Objective) heading that has extra oral and intraoral issues, which is further followed by radiographic findings. Next, two headings, A. (Assessment) and P. (Plan) are followed by different options.

Source: (Courtesy Dr. Ravi Koka, San Francisco, CA.)

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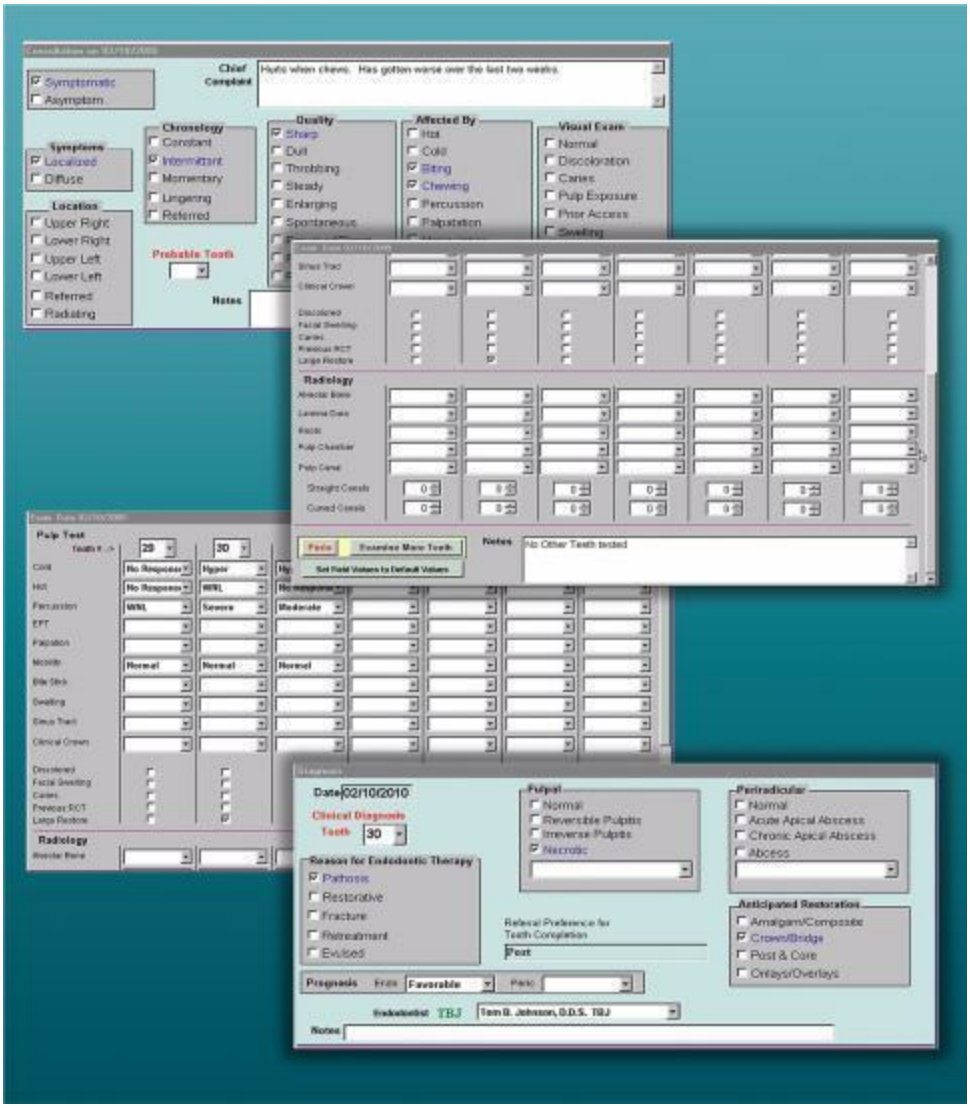

FIG. 1.4 Several practice management software packages have features for charting endodontic diagnoses using user-defined drop-down menus and areas for specific notations. Note that for legal purposes, it is desirable that all recorded documentation have the ability to be locked, or if any modifications are made after 24 hours, the transaction should be recorded with an automated time/date stamp. This is necessary so the data cannot be fraudulently manipulated.

Four panels are shown as follows:

1) Chief complaint is hurts when chews. Has gotten worse over the last two weeks. Tab on the upper left corner reads, symptomatic and asymptomatic. The headings are symptoms, location, chronology, quality, affected by, visual exam each with different options. Probable tooth is selected from dropdown option.

2) It has drop down lists for sinus test and clinical crown, followed by

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symptoms (discolored, facial swelling, caries, previous RCT, and large intestine each with blank check box) and radiology (alveolar bone, lamina dura, roots, pulp chamber, and pulp canal each with drop down options). It is further followed by options for straight canals and curved canals.

3)Different options (cold, hot, percussion, EPT, palpation, mobility, bite stick, swelling, sinus track, and clinical crown, each with drop down list) for pulp test are shown. It is further followed by options such as discolored, facial swelling, caries, previous RCT and large intestine, each with a blank check box. At the bottom, radiology has the option alveolar bone followed by drop down boxes.

4) It has date, followed by clinical diagnosis and tooth number with drop down list. The various options are given under the headings reason for endodontic therapy, pulpal, periradicular, anticipated restoration, and prognosis. At the bottom, note is followed by blank check box.

Source: (Courtesy PBS Endo, Cedar Park, TX.)

History of present dental problem

The dialogue between the patient and the clinician should encompass all of the details pertinent to the events that led to the chief complaint. The clinician should direct the conversation in a manner that produces a clear and concise narrative that chronologically depicts all of the necessary information about the patient's symptoms and the development of these symptoms. To help elucidate this information, the patient is first instructed to fill out a dental history form as part of the patient's office registration. This information will help the clinician decide which approach to use when asking the patient questions. The interview first determines *what is going on* in an effort to determine *why it is going on* for the purpose of eventually determining *what is necessary to resolve the chief complaint.*

Dental history interview

After starting the interview and determining the nature of the chief complaint, the clinician continues the conversation by documenting the sequence of events that initiated the request for an evaluation. The dental history is divided into five basic directions of questioning: localization, commencement, intensity, provocation or attenuation, and duration.

Localization. "Can you point to the offending tooth?" Often the patient can

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point to or tap the offending tooth. This is the most fortunate scenario for the clinician because it helps direct the interview toward the events that might have caused any particular pathosis in this tooth. In addition, localization allows subsequent diagnostic tests to focus more on this particular tooth. When the symptoms are not well localized, the diagnosis is a greater challenge.

Commencement. "When did the symptoms first occur?" A patient who is having symptoms often remembers when these symptoms started. Sometimes the patient will even remember the initiating event. It may have been spontaneous in nature; it may have begun after a dental visit for a restoration; trauma may be the etiology; biting on a hard object may have initially produced the symptoms; or the initiating event may have occurred concurrently with other symptoms (e.g., sinusitis, headache, chest pain). However, the clinician should resist the tendency to make a premature diagnosis based on these circumstances. The clinician should not simply assume "guilt by association" but instead should use this information to enhance the overall diagnostic process.

Intensity. "How intense is the pain?" It often helps to quantify how much pain the patient is actually having. The clinician might ask, "On a scale from 1 to 10, with 10 the most severe, how would you rate your symptoms?" Hypothetically, a patient could present with "an uncomfortable sensitivity to cold" or "an annoying pain when chewing" but might rate this "pain" only as a 2 or a 3. These symptoms certainly contrast with the type of symptoms that prevent a patient from sleeping at night. Often the intensity can be subjectively measured by what is necessary for the diminution of pain (e.g., acetaminophen versus a narcotic pain reliever). This intensity level may affect the decision to treat or not to treat with endodontic therapy. Pain is now considered a standard vital sign, and documenting pain intensity (scale of 0 to 10) provides a baseline for comparison after treatment.

Provocation or attenuation. "What produces or reduces the symptoms?" Mastication and locally applied temperature changes account for the majority of initiating factors that cause dental pain. The patient may relate that drinking something cold causes the pain or possibly that chewing or biting is the only stimulus that "makes it hurt." The patient might say that the pain is only reproduced on "release from biting." On occasion, a patient may present to the dental office with a cold drink in hand and state that the symptoms can

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only be *reduced* by bathing the tooth in cold water. Nonprescription pain relievers may relieve some symptoms, whereas narcotic medication may be required to reduce others. Note that patients who are using narcotic as well as non-narcotic (e.g., ibuprofen) analgesics may respond differently to questions and diagnostic tests, thereby altering the validity of diagnostic results. Thus, it is important to know what drugs patients have taken in the previous 4 to 6 hours. These provoking and relieving factors may help the clinician to determine which diagnostic tests should be performed to establish a more objective diagnosis.

Duration. "Do the symptoms subside shortly, or do they linger after they are provoked?" The difference between a cold sensitivity that subsides in a few seconds and one that subsides in minutes may determine whether a clinician repairs a defective restoration or provides endodontic treatment. The duration of symptoms after a stimulating event should be recorded to establish how long the patient felt the sensation in terms of seconds or minutes. Clinicians often first test control teeth (possibly including a contralateral "normal" tooth) to define a "normal" response for the patient; thus, "lingering" pain is apparent when comparing the duration between the control teeth and the suspected tooth.

With the dental history interview complete, the clinician has a better understanding of the patient's chief complaint and can concentrate on making an objective diagnostic evaluation, although the subjective (and artistic) phase of making a diagnosis is not yet complete and will continue after the more objective testing and scientific phase of the investigatory process.

Examination and testing

Extraoral examination

Basic diagnostic protocol suggests that a clinician observe patients as they enter the operatory. Signs of physical limitations may be present, as well as signs of facial asymmetry that result from facial swelling. Visual and palpation examinations of the face and neck are warranted to determine whether swelling is present. Many times a facial swelling can be determined only by palpation when a unilateral "lump or bump" is present. The presence of bilateral swellings may be a normal finding for any given patient;

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however, it may also be a sign of a systemic disease or the consequence of a developmental event. Palpation allows the clinician to determine whether the swelling is localized or diffuse, firm or fluctuant. These latter findings will play a significant role in determining the appropriate treatment.

Palpation of the cervical and submandibular lymph nodes is an integral part of the examination protocol. If the nodes are found to be firm and tender along with facial swelling and an elevated temperature, there is a high probability that an infection is present. The disease process has moved from a localized area immediately adjacent to the offending tooth to a more widespread systemic involvement.

Extraoral facial swelling of odontogenic origin typically is the result of endodontic etiology because diffuse facial swelling resulting from a periodontal abscess is rare. Swellings of nonodontogenic origin must always be considered in the differential diagnosis, especially if an obvious dental pathosis is not found.[77] This situation is discussed in subsequent chapters.

A subtle visual change such as loss of definition of the nasolabial fold on one side of the nose may be the earliest sign of a canine space infection (Fig. 1.5). Pulpal necrosis and periradicular disease associated with a maxillary canine should be suspected as the source of the problem. Extremely long maxillary central incisors also may be associated with a canine space infection, but most extraoral swellings associated with the maxillary centrals express themselves as a swelling of the upper lip and base of the nose.

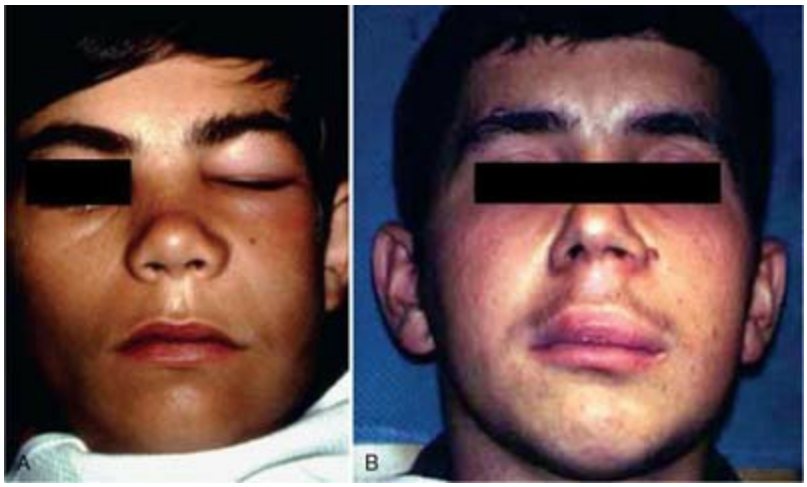

FIG. 1.5 **A,** Canine space swelling of the left side of the face extending into and involving the left eye. **B,** Swelling of the upper lip and the loss

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of definition of the nasolabial fold on the patient's left side, which indicates an early canine space infection.

A) Front view of patient's face shows inflammation on eye lid and under eye area.

B) Front view of patient's face shows bump on the right side of upper lip.

If the buccal space becomes involved, the swelling will be extraoral in the area of the posterior cheek (Fig. 1.6). These swellings are generally associated with infections originating from the buccal root apices of the maxillary premolar and molar teeth and the mandibular premolar (Fig. 1.7) and first molar teeth. The mandibular second and third molars also may be involved, but infections associated with these two teeth are just as likely to exit to the lingual where other spaces would be involved. For infections associated with these teeth, the root apices of the maxillary teeth must lie superior to the attachment of the buccinator muscle to the maxilla, and the apices of the mandibular teeth must be inferior to the buccinator muscle attachment to the mandible.[77]

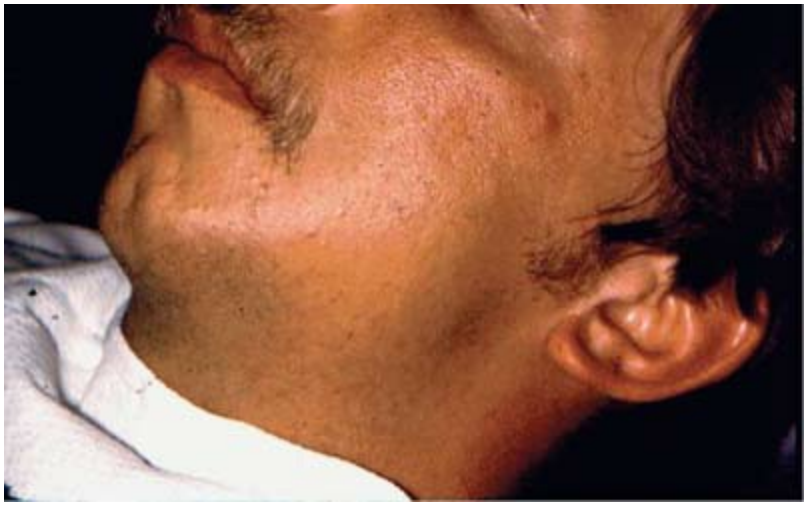

FIG. 1.6 Buccal space swelling associated with an acute periradicular abscess from the mandibular left second molar.

Close-up view of lateral view of patient's face shows swelling over the lining of left cheek and the mandible region.

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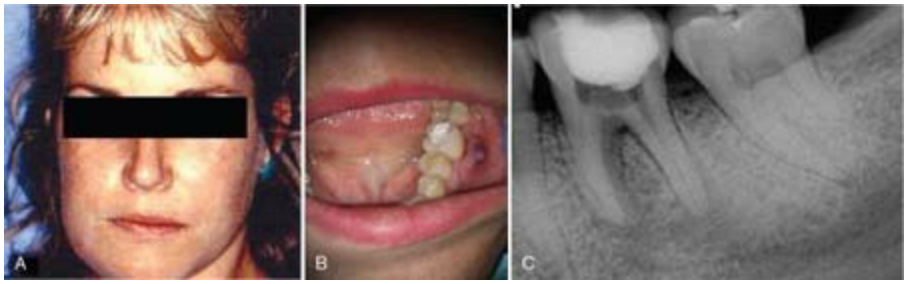

FIG. 1.7 **A,** Buccal space swelling of the left side of the patient's face. Note the asymmetry of the left side of the face. **B,** Intraoral view of another patient shows swelling present in the left posterior mucobuccal fold. **C,** This buccal space infection was associated with periradicular disease from the mandibular left first molar. Note on the radiograph the periradicular radiolucency and incomplete endodontic treatment.

A) Front view of patient's face shows inflammation in the mandible region on left.

B) Close-up view shows open mouth with a gray lesion near the molar teeth on the left.

C) Radiograph shows the second molar with less dense tissue between crown and root.

Source: (*B* and *C*, Courtesy Dr. Jaydeep S. Talim, Los Angeles, CA.)

Extraoral swelling associated with mandibular incisors will generally exhibit itself in the submental (Fig. 1.8) or submandibular space. Infections associated with any mandibular teeth, which exit the alveolar bone on the lingual and are inferior to the mylohyoid muscle attachment, will be noted as swelling in the submandibular space. Further discussions of fascial space infections may be found in Chapter 16.

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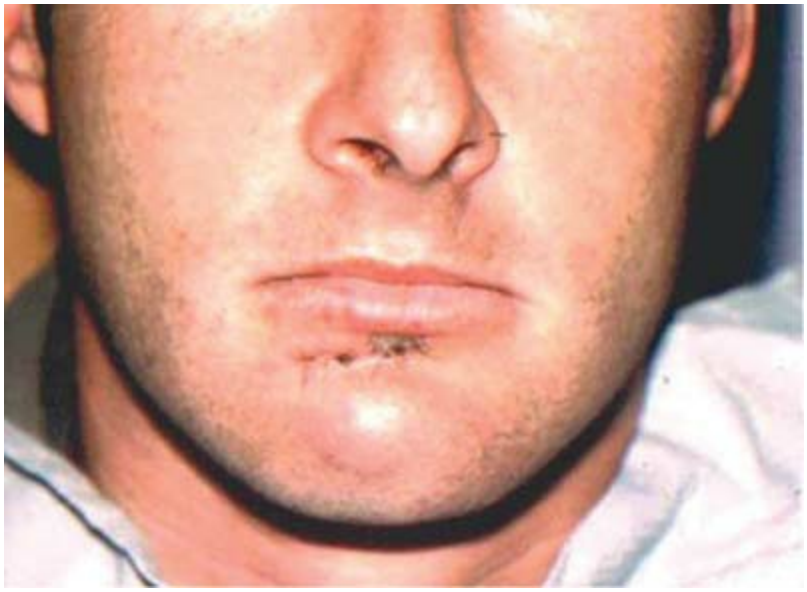

FIG. 1.8 Swelling of the submental space associated with periradicular disease from the mandibular incisors.

Close-up view shows the lower face of a person with a large round bump near the root canal of incisors on the chin.

Sinus tracts of odontogenic origin may also open through the skin of the face (Figs. 1.9 and 1.10).[2,56,64] These openings in the skin will generally close once the offending tooth is treated and healing occurs. A scar is more likely to be visible on the skin surface in the area of the sinus tract stoma than on the oral mucosal tissues (see Fig. 1.10, *C* and *D*). Many patients with extraoral sinus tracts give a history of being treated by general physicians, dermatologists, oncologists, or plastic surgeons with systemic or topical antibiotics or surgical procedures in attempts to heal the extraoral stoma. In these particular cases, after multiple treatment failures, the patients may finally be referred to a dental clinician to determine whether there is a dental cause. Raising the awareness of physicians to such cases will aid in more accurate diagnosis and faster referral to the dentist or endodontist.

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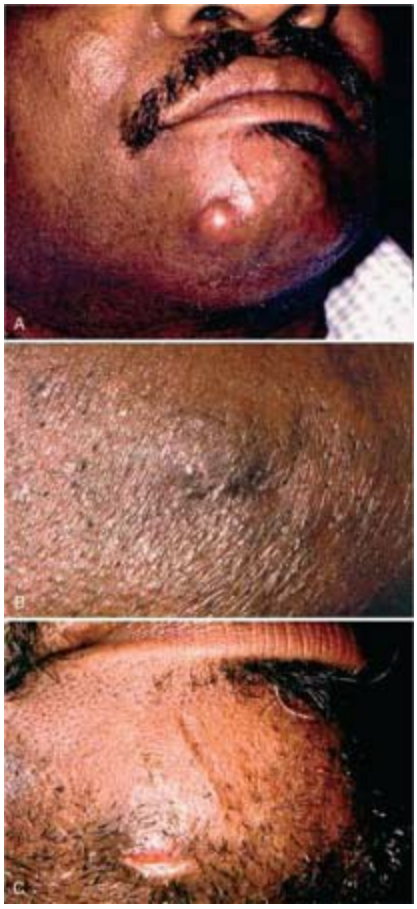

FIG. 1.9 **A,** Extraoral drainage associated with periradicular disease from the mandibular right canine. Note the parulis on the right anterior side of the face. **B,** Initial scar associated with the extraoral drainage incision after the parulis was drained and root canal therapy performed on the canine. **C,** Three-month follow-up shows healing of the incision area. Note the slight inversion of the scar tissue.

Set of three photographs marked A through C depict changes in the sinus tracts.

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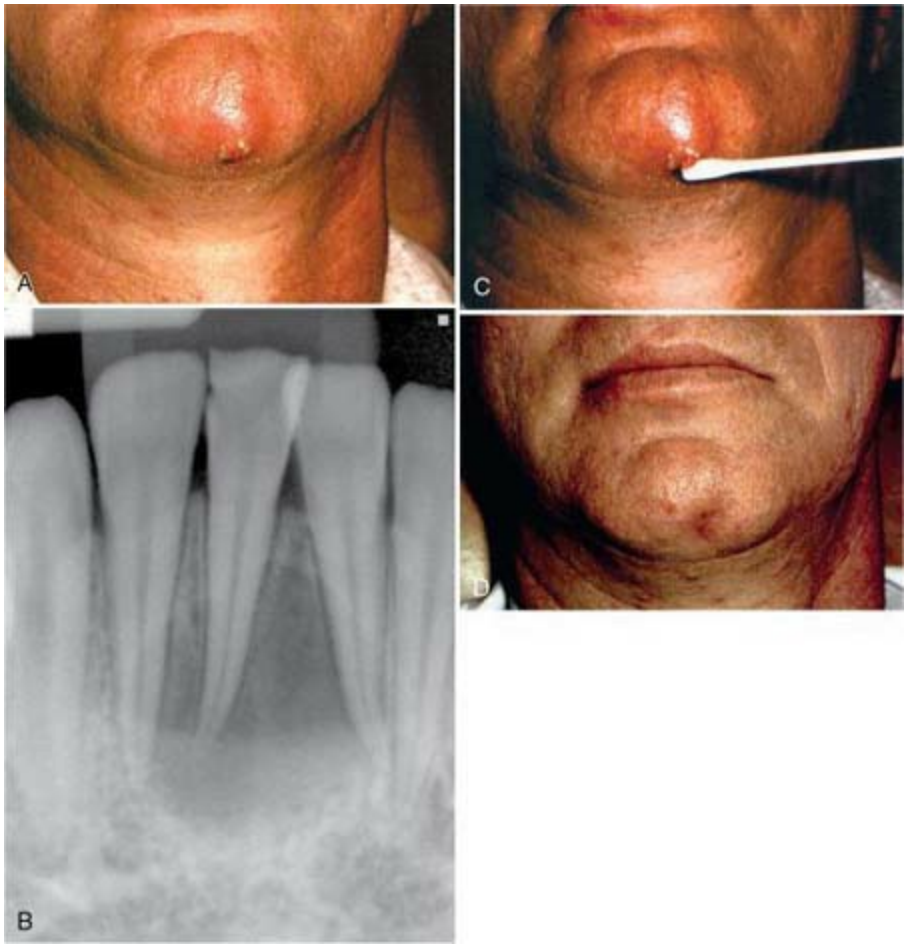

FIG. 1.10 **A,** Extraoral sinus tract opening onto the skin in the central chin area. **B,** Radiograph showing large radiolucency associated with the mandibular incisors. **C,** A culture is obtained from the drainage of the extraoral sinus tract. **D,** The healed opening of the extraoral sinus tract 1 month after root canal therapy was completed. Note the slight skin concavity in the area of the healed sinus tract.

A) Close-up view shows fluid-filled wounds on the chin.

B) Radiograph shows large dark patch in the root of incisors.

C) Close-up view shows a Q-tip removing the oozed out fluid from purulent drainage.

D) Close-up view shows a slight pit in place of the fluid-filled wounds.

Intraoral examination

The intraoral examination may give the clinician insight as to which intraoral areas may need a more focused evaluation. Any abnormality should be

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carefully examined for either prevention or early treatment of associated pathosis.[4,30,75,113,110,126] Swelling, localized lymphadenopathy, or a sinus tract should provoke a more detailed assessment of related and proximal intraoral structures.

Soft-tissue examination

As with any dental examination, there should be a routine evaluation of the intraoral soft tissues. The gingiva and mucosa should be dried with either a low-pressure air syringe or a 2-by-2-inch gauze pad. By retracting the tongue and cheek, all of the soft tissue should be examined for abnormalities in color or texture. Any raised lesions or ulcerations should be documented and, when necessary, evaluated with a biopsy or referral.[82]

Intraoral swelling

Intraoral swellings should be visualized and palpated to determine whether they are diffuse or localized and whether they are firm or fluctuant. These swellings may be present in the attached gingiva, alveolar mucosa, mucobuccal fold, palate, or sublingual tissues. Other testing methods are required to determine whether the origin is endodontic, periodontic, or a combination of these two or whether it is of nonodontogenic origin.

Swelling in the anterior part of the palate (Fig. 1.11) is most frequently associated with an infection present at the apex of the maxillary lateral incisor or the palatal root of the maxillary first premolar. More than 50% of the maxillary lateral incisor root apices deviate in the distal or palatal directions. A swelling in the posterior palate (Fig. 1.12) is most likely associated with the palatal root of one of the maxillary molars.[77]

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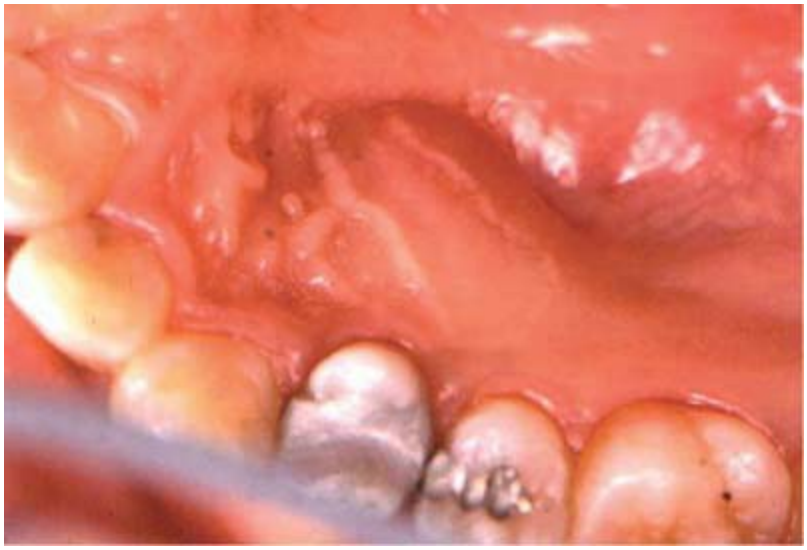

FIG. 1.11 Fluctuant swelling in the anterior palate associated with periradicular disease from the palatal root of the maxillary first premolar.

Close up view of interior of mouth shows silver fillings on two teeth and an extremely soft tissue with pus inside present near the premolars.

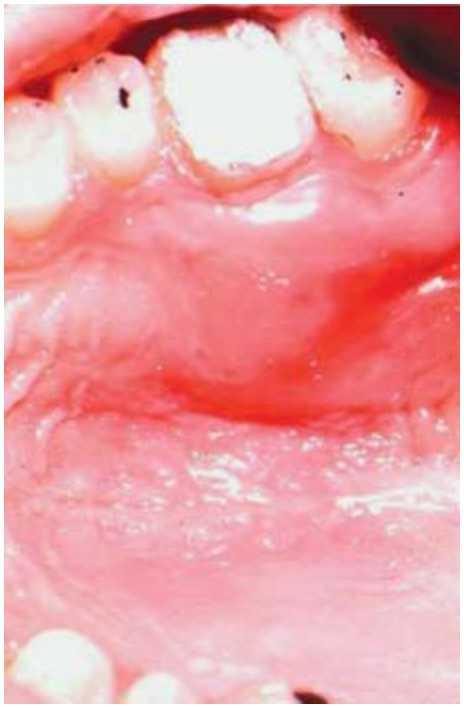

FIG. 1.12 Fluctuant swelling in the posterior palate associated with periradicular disease from the palatal root of the maxillary first molar.

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Close-up view of upper teeth shows a sore tissue in the soft palate.

Intraoral swelling present in the mucobuccal fold (Fig. 1.13) can result from an infection associated with the apex of the root of any maxillary tooth that exits the alveolar bone on the facial aspect and is inferior to the muscle attachment present in that area of the maxilla (see also Chapter 15). The same is true with the mandibular teeth if the root apices are superior to the level of the muscle attachments and the infection exits the bone on the facial. Intraoral swelling can also occur in the sublingual space if the infection from the root apex spreads to the lingual and exits the alveolar bone superior to the attachment for the mylohyoid muscle. The tongue will be elevated and the swelling will be bilateral because the sublingual space is contiguous with no midline separation. If the infection exits the alveolar bone to the lingual with mandibular molars and is inferior to the attachment of the mylohyoid muscle, the swelling will be noted in the submandibular space. Severe infections involving the maxillary and mandibular molars can extend into the parapharyngeal space, resulting in intraoral swelling of the tonsillar and pharyngeal areas. This can be life threatening if the patient's airway becomes obstructed.[77,80]

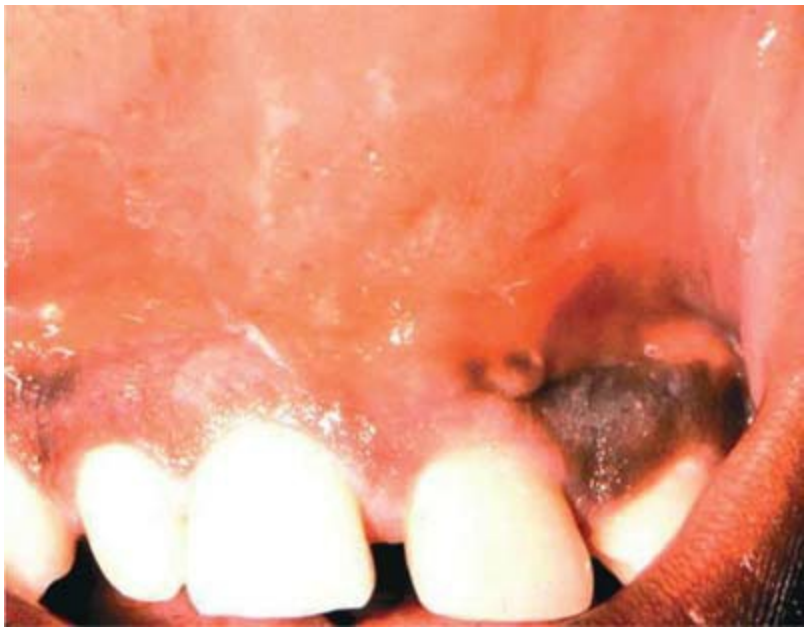

FIG. 1.13 Fluctuant swelling in the mucobuccal fold associated with periradicular disease from the maxillary central incisor.

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Close-up view shows an injured and black tissue in the root canal of central incisor.

Intraoral sinus tracts

On occasion, a chronic endodontic infection will drain through an intraoral communication to the gingival surface and is known as a *sinus tract.*[12] This pathway, which is sometimes lined with epithelium, extends directly from the source of the infection to a surface opening, or *stoma,* on the attached gingival surface. As previously described, it can also extend extraorally. The term *fistula* is often inappropriately used to describe this type of drainage. The fistula, by definition, is actually an abnormal communication pathway between two internal organs or from one epithelium-lined surface to another epithelium-lined surface.[6]

Histologic studies have found that most sinus tracts are not lined with epithelium throughout their entire length. One study found that only 1 out of the 10 sinus tracts examined were lined with epithelium, whereas the other 9 specimens were lined with granulation tissue.[55] Another study, with a larger sample size, found that two thirds of the specimens did not have epithelium extending beyond the level of the surface mucosa rete ridges.[12] The remaining specimens had some epithelium that extended from the oral mucosa surface to the periradicular lesion.[12] The presence or absence of an epithelial lining does not seem to prevent closure of the tract as long as the source of the problem is properly diagnosed and adequately treated and the endodontic lesion has healed. Failure of a sinus tract to heal after treatment will necessitate further diagnostic procedures to determine whether other sources of infection are present or whether a misdiagnosis occurred.

In general, a periapical infection that has an associated sinus tract is not painful, although often there is a history of varying magnitudes of discomfort before sinus tract development. Besides providing a conduit for the release of infectious exudate and the subsequent relief of pain, the sinus tract can also provide a useful aid in determining the source of a given infection. Sometimes objective evidence as to the origin of an odontogenic infection is lacking. The stoma of the sinus tract may be located directly adjacent to or at a distant site from the infection. Tracing the sinus tract will provide objectivity in diagnosing the location of the problematic tooth. To trace the

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sinus tract, a size #25 or #30 gutta-percha cone is threaded into the opening of the sinus tract. Although this may be slightly uncomfortable to the patient, the cone should be inserted until resistance is felt. After a periapical radiograph is exposed, the origin of the sinus tract is determined by following the path taken by the gutta-percha cone (Fig. 1.14). This will direct the clinician to the tooth involved and, more specifically, to the part of the root of the tooth that is the source of the pathosis. Once the causative factors related to the formation of the sinus tract are removed, the stoma and the sinus tract will close within several days.

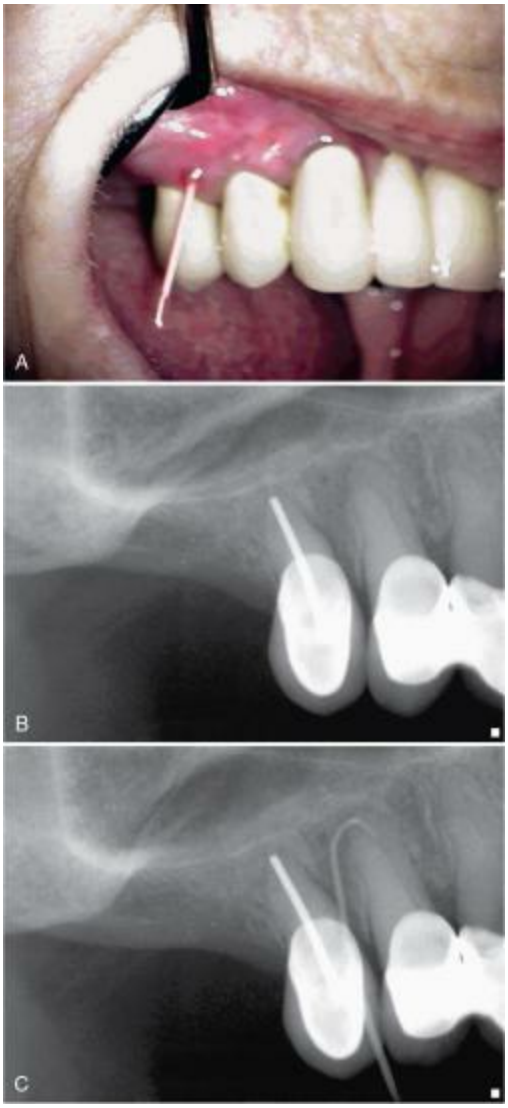

FIG. 1.14 **A,** To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. **B,**

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Radiograph of the area shows an old root canal in a maxillary second premolar and a questionable radiolucent area associated with the first premolar, with no clear indication of the etiology of the sinus tract. **C,** After tracing the sinus tract, the gutta-percha is seen to be directed to the source of pathosis, the apex of the maxillary first premolar.

A) Close-up view shows a gutta-percha cone being inserted in the root canal above first premolar.

B) Radiograph shows two teeth where a thread is inserted through the first tooth. The first tooth is radiopaque while the second tooth is partially radiolucent.

C) Radiograph shows two teeth with a thread passing from them.

The stomata of intraoral sinus tracts may open in the alveolar mucosa, in the attached gingiva, or through the furcation or gingival crevice. They may exit through either the facial or the lingual tissues depending on the proximity of the root apices to the cortical bone. If the opening is in the gingival crevice, it is normally present as a narrow defect in one or two isolated areas along the root surface. When a narrow defect is present, the differential diagnosis must include the opening of a periradicular endodontic lesion, a vertical root fracture, or the presence of a developmental groove on the root surface. This type of sinus tract can be differentiated from a primary periodontal lesion because the latter generally presents as a pocket with a broad coronal opening and more generalized alveolar bone loss around the root. Other pulp testing methods may assist in verifying the source of infection.[111,112,121]

Palpation

In the course of the soft-tissue examination, the alveolar hard tissues should also be palpated. Emphasis should be placed on detecting any soft-tissue swelling or bony expansion, especially noting how it compares with and relates to the adjacent and contralateral tissues. In addition to objective findings, the clinician should question the patient about any areas that feel unusually sensitive during this palpation part of the examination.

A palpation test is performed by applying firm digital pressure to the mucosa covering the roots and apices. The index finger is used to press the mucosa against the underlying cortical bone. This will detect the presence of

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periradicular abnormalities or specific areas that produce painful response to digital pressure. A positive response to palpation may indicate an active periradicular inflammatory process. However, this test does not indicate whether the inflammatory process is of endodontic or periodontal origin.

Percussion

Referring back to the patient's chief complaint may indicate the importance of percussion testing for this particular case. If the patient is experiencing acute sensitivity or pain on mastication, this response can typically be duplicated by individually percussing the teeth, which often isolates the symptoms to a particular tooth. Pain to percussion does not indicate that the tooth is vital or nonvital but is rather an indication of inflammation in the periodontal ligament (i.e., symptomatic apical periodontitis). This inflammation may be secondary to physical trauma, occlusal prematurities, periodontal disease, or the extension of pulpal disease into the periodontal ligament space. The indication of where the pain originates is interpreted by the mesencephalic nucleus, receiving its information from proprioceptive nerve receptors. Although subject to debate, the general consensus is that there are relatively few proprioceptors in the dental pulp; however, they are prevalent in the periodontal ligament spaces.[24] This is why it may be difficult for the patient to discriminate the location of dental pain in the earlier stages of pathosis, when only the C fibers are stimulated. Once the disease state extends into the periodontal ligament space, the pain may become more localized for the patient; therefore, the affected tooth will be more identifiable with percussion and mastication testing.

Before percussing any teeth, the clinician should tell the patient what will transpire during this test. Because the presence of acute symptoms may create anxiety and possibly alter the patient's response, properly preparing the patient will lead to more accurate results. The contralateral tooth should first be tested as a control, as should several adjacent teeth that are certain to respond normally. The clinician should advise the patient that the sensation from this tooth is normal and ask to be advised of any tenderness or pain from subsequent teeth.

Percussion is performed by tapping on the incisal or occlusal surfaces of the teeth either with the finger or with a blunt instrument. The testing should

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initially be done gently, with light pressure being applied digitally with a gloved finger tapping. If the patient cannot detect significant difference between any of the teeth, the test should be repeated using the blunt end of an instrument, like the back end of a mirror handle (Fig. 1.15). The tooth crown is tapped vertically and horizontally. The tooth should first be percussed occlusally, and if the patient discerns no difference, the test should be repeated, percussing the buccal and lingual aspects of the teeth. For any heightened responses, the test should be repeated as necessary to determine that it is accurate and reproducible, and the information should be documented.

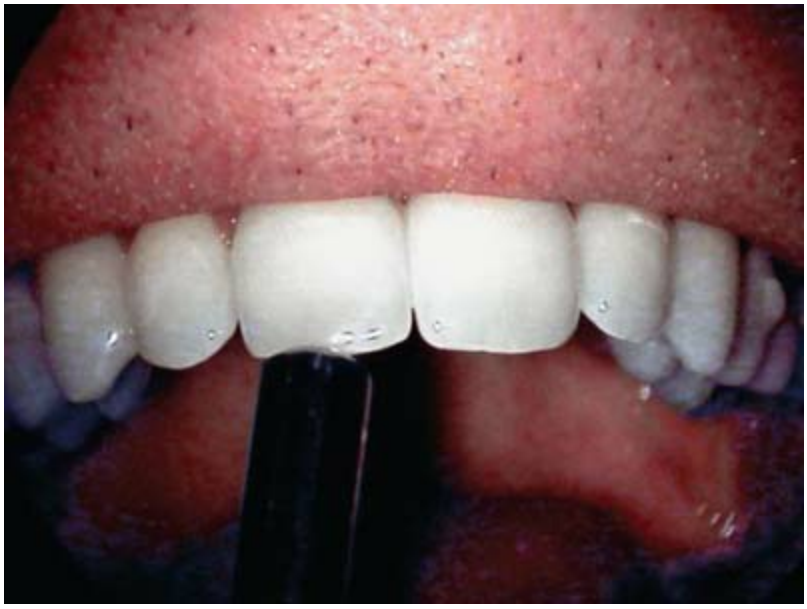

FIG. 1.15 Percussion testing of a tooth, using the back end of a mirror handle.

Close-up view shows a front tooth in the upper jaw being tapped with a mirror handle.

Although this test does not disclose the condition of the pulp, it indicates the presence of a periradicular inflammation. An abnormal positive response indicates inflammation of the periodontal ligament that may be of either pulpal or periodontal origin. The sensitivity of the proprioceptive fibers in an inflamed periodontal ligament will help identify the location of the pain. This test should be done gently, especially in highly sensitive teeth. It should be

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repeated several times and compared with control teeth.

Mobility

Like percussion testing, an increase in tooth mobility is not an indication of pulp vitality. It is merely an indication of a compromised periodontal attachment apparatus. This compromise could be the result of acute or chronic physical trauma, occlusal trauma, parafunctional habits, periodontal disease, root fractures, rapid orthodontic movement, or the extension of pulpal disease, specifically an infection, into the periodontal ligament space. Tooth mobility is directly proportional to the integrity of the attachment apparatus or to the extent of inflammation in the periodontal ligament. Often the mobility reverses to normal after the initiating factors are repaired or eliminated. Because determining mobility by simple finger pressure can be visually subjective, the back ends of two mirror handles should be used, one on the buccal aspect and one on the lingual aspect of the tooth (Fig. 1.16). Pressure is applied in a facial-lingual direction as well as in a vertical direction and the tooth mobility is scored (Box 1.2). Any mobility that exceeds +1 should be considered abnormal. However, the teeth should be evaluated on the basis of how mobile they are relative to the adjacent and contralateral teeth.

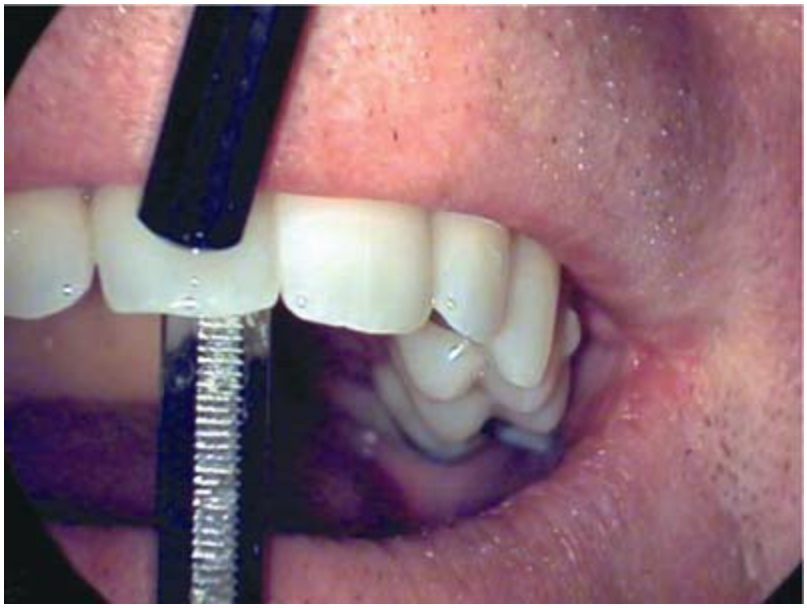

FIG. 1.16 Mobility testing of a tooth, using the back ends of two mirror

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handles.

Close-up view shows one of the upper incisors being examined through two mirror handles present on either side of the tooth.

Box 1.2

Recording Tooth Mobility

+1 mobility: The first distinguishable sign of movement greater than normal
+2 mobility: Horizontal tooth movement no greater than 1 mm
+3 mobility: Horizontal tooth movement greater than 1 mm, with or without the visualization of rotation or vertical depressability

Periodontal examination

Periodontal probing is an important part of any intraoral diagnosis. The measurement of periodontal pocket depth is an indication of the depth of the gingival sulcus, which corresponds to the distance between the height of the free gingival margin and the height of the attachment apparatus below. Using a calibrated periodontal probe, the clinician should record the periodontal pocket depths on the mesial, middle, and distal aspects of both the buccal and lingual sides of the tooth, noting the depths in millimeters. The periodontal probe is "stepped" around the long axis of the tooth, progressing in 1-mm increments. Periodontal bone loss that is wide, as determined by a wide span of deep periodontal probing, is generally considered to be of periodontal origin and is typically more generalized in other areas of the mouth. However, isolated areas of vertical bone loss may be of an endodontic origin, specifically from a nonvital tooth whose infection has extended from the periapex to the gingival sulcus. Again, proper pulp testing is imperative, not just for the determination of a diagnosis but also for the development of an accurate prognosis assessment. For example, a periodontal pocket of endodontic origin may resolve after endodontic treatment, but if the tooth was originally vital with an associated deep periodontal pocket, endodontic treatment will not improve the periodontal condition. In addition, as discussed in Chapter 22, a vertical root fracture may often cause a localized

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narrow periodontal pocket that extends deep down the root surface. Characteristically, the adjacent periodontium is usually within normal limits.

Furcation bone loss can be secondary to periodontal or pulpal disease. The amount of furcation bone loss, as observed both clinically and radiographically, should be documented (Box 1.3). Results of pulp tests (described later) will aid in diagnosis.

Box 1.3

Recording Furcation Defects

Class I furcation defect: The furcation can be probed but not to a significant depth.
Class II furcation defect: The furcation can be entered into but cannot be probed completely through to the opposite side.
Class III furcation defect: The furcation can be probed completely through to the opposite side.

Pulp tests

Pulp test (pulp sensibility test) is a diagnostic procedure to determine pulp status. It can be performed with electrical, mechanical, or thermal stimuli, or by the assessment of the blood supply to the tooth.[6] It involves attempting to make a determination of the responsiveness of pulpal sensory neurons.[62,63] It aims to obtain a subjective response from the patient (i.e., to determine whether the pulpal nerves are functional), or the tests may involve a more objective approach using devices that detect the integrity of the pulpal vasculature. Unfortunately, the quantitative evaluation of the status of pulp tissue can only be determined histologically, as it has been shown that there is not necessarily a good correlation between the objective clinical signs and symptoms and the pulpal histology.[122,123]

Thermal

Various methods and materials have been used to test the pulp's response to thermal stimuli. The baseline or normal response to either cold or hot is a patient's report that a sensation is felt but disappears immediately upon

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removal of the thermal stimulus. Abnormal responses include a lack of response to the stimulus, a lingering or intensification of a painful sensation after the stimulus is removed, or an immediate, excruciatingly painful sensation as soon as the stimulus is placed on the tooth.

Cold testing is the primary pulp testing method used by many clinicians today. It is especially useful for patients presenting with porcelain jacket crowns or porcelain-fused-to-metal crowns where no natural tooth surface (or much metal) is accessible. If a clinician chooses to perform this test with sticks of ice, then the use of a rubber dam is recommended, because melting ice will run onto adjacent teeth and gingiva, yielding potentially false-positive responses.

Frozen carbon dioxide (CO_2), also known as *dry ice* or *carbon dioxide snow*, or *CO_2 stick*, has been found to be reliable in eliciting a positive response if vital pulp tissue is present in the tooth.[46,98,99] One study found that vital teeth would respond to both frozen CO_2 and skin refrigerant, with skin refrigerant producing a slightly quicker response.[66] Frozen carbon dioxide has also been found to be effective in evaluating the pulpal response in teeth with full coverage crowns for which other tests such as electric pulp testing is not possible.[11] For testing purposes, a solid stick of CO_2 is prepared by delivering CO_2 gas into a specially designed plastic cylinder (Fig. 1.17). The resulting CO_2 stick is applied to the facial surface of either the natural tooth structure or crown. Several teeth can be tested with a single CO_2 stick. The teeth should be isolated and the oral soft tissues should be protected with a 2-by-2-inch gauze or cotton roll so the frozen CO_2 will not come into contact with these structures. Because of the extremely cold temperature of the frozen CO_2 (−69°F to −119°F; −56°C to −98°C), burns of the soft tissues can occur. It has been demonstrated on extracted teeth that frozen CO_2 application has resulted in a significantly greater intrapulpal temperature decrease than either skin refrigerant or ice.[11] Also, it appears that the application of CO_2 to teeth does not result in any irreversible damage to the pulp tissues or cause any significant enamel crazing.[61,104]

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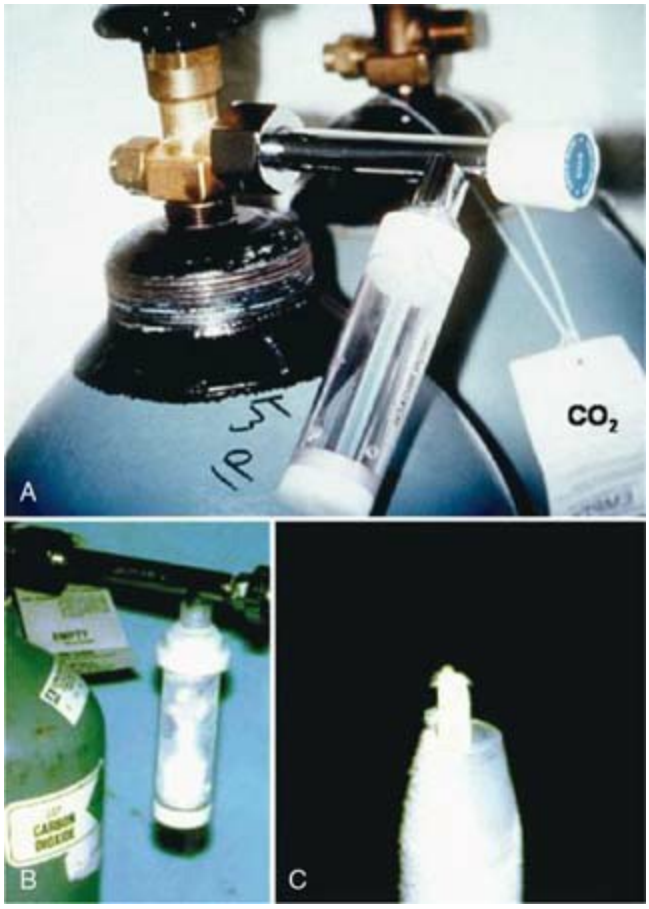

FIG. 1.17 **A,** Carbon dioxide tank with apparatus attached to form solid CO_2 stick/pencil. **B,** CO_2 gas being transformed into a solid stick/pencil. **C,** CO_2 stick/pencil extruded from end of a plastic carrier and ready for use.

Set of three photographs marked A through C depict transformation of carbon dioxide gas from a cylinder into a carbon dioxide stick or pencil.

The most popular method of performing cold testing is with a refrigerant spray. It is readily available, easy to use, and provides test results that are reproducible, reliable, and equivalent to that of frozen CO_2.[46,66,96,141] One of the current products contains 1,1,1,2-tetrafluoroethane, which has zero ozone depletion potential and is environmentally safe. It has a temperature of −26.2°C.[66] The spray is most effective for testing purposes when it is applied to the tooth on a large #2 cotton pellet (Fig. 1.18). In one study,[65] a significantly lower intrapulpal temperature was achieved when a #2 cotton pellet was dipped or sprayed with the refrigerant compared with the result when a small #4 cotton pellet or cotton applicator was used. The sprayed

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cotton pellet should be applied to the midfacial area of the tooth or crown. As with any other pulp testing method, adjacent or contralateral "normal" teeth should also be tested to establish a baseline response. It appears that frozen CO_2 and refrigerant spray are superior to other cold testing methods and equivalent or superior to the electric pulp tester for assessing pulp vitality.[11,46] However, one study found that periodontal attachment loss and gingival recession may influence the reported pain response with cold stimuli.[116]

FIG. 1.18 **A,** Refrigerant spray container. **B,** A large cotton pellet made of a cotton roll, or a ready-made size #2 (large) cotton pellet, can be used to apply the refrigerant spray to the tooth surface. The small #4 cotton pellet does not provide as much surface area as the #2 cotton pellet, and therefore should not be used to deliver the refrigerant to the tooth surface. **C,** A large cotton pellet sprayed with the refrigerant and ready to be applied to the tooth surface.

A) A spray bottle, with text on the bottle reading, "Green Endo Ice spray."

B) An applicator with a large cotton ball on its one end.

C) A bundle of cotton with two small cotton pellets.

Source: (*A,* Courtesy Coltène/Whaledent, Cuyahoga Falls, OH.)

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To be most reliable, cold testing should be used in conjunction with an electric pulp tester (described later in this chapter) so that the results from one test will verify the findings of the other test. If a mature, nontraumatized tooth does not respond to both cold testing and electric pulp testing, then the pulp can be considered necrotic.[23,98,141] However, a multirooted tooth, with at least one root containing vital pulp tissue, may respond to a cold test and electric pulp test even if one or more of the roots contain necrotic pulp tissue.[98]

Another thermal testing method involves the use of heat. Heat testing is most useful when a patient's chief complaint is intense dental pain on contact with any hot liquid or food. When a patient is unable to identify which tooth is sensitive, a heat test is appropriate. Starting with the most posterior tooth in that area of the mouth, each tooth is individually isolated with a dental dam. An irrigating syringe is filled with a liquid (most commonly plain water) that has a temperature similar to that which would cause the painful sensation. The liquid is then expressed from the syringe onto the isolated tooth to determine whether the response is normal or abnormal. The clinician moves forward in the quadrant, isolating each individual tooth until the offending tooth is located. That tooth will exhibit an immediate, intense painful response to the heat. With heat testing, a delayed response may occur, so waiting 10 seconds between each heat test will allow sufficient time for the onset of symptoms. This method can also be used to apply cold water to the entire crown for cases in which cold is the precipitating stimulus.

Another method for heat testing is to apply heated gutta-percha or compound stick to the surface of the tooth. If this method is used, a light layer of lubricant should be placed onto the tooth surface before applying the heated material to prevent the hot gutta-percha or compound from adhering to the dry tooth surface. Heat can also be generated by the friction created when a dry rubber-polishing wheel is run at a high speed against the dry surface of a tooth. However, this latter method is not recommended. Another approach is the use of electronic heat-testing instruments.[20]

If the heat test confirms the results of other pulp testing procedures, emergency care can then be provided. Often a tooth that is sensitive to heat may also be responsible for some spontaneous pain. The patient may present with cold liquids in hand just to minimize the pain (Fig. 1.19). In such cases,

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the application of cold to a specific tooth may eliminate the pain and greatly assist in the diagnosis. Typically, a tooth that responds to heat and then is relieved by cold is found to be necrotic.

FIG. 1.19 Irreversible pulpitis associated with the mandibular right second molar. Patient has found that the only way to alleviate the pain is to place a jar filled with ice water against the right side of his face.

A patient places an ice jar at the right corner of lips.

Electric

Assessment of pulp neural responses *(sensibility)* can also be accomplished by electric pulp testing.[79] Electric pulp testers of different designs and manufacturers have been used for this purpose. Electric pulp testers should be an integral part of any dental practice. It should be noted that the sensibility of the pulp is determined by the intactness and health of the vascular supply, not by the status of the pulpal nerve fibers. Even though advances are being made with regard to determining the status of the pulp on the basis of the blood supply, this technology has not been perfected enough at this time to be

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used on a routine basis in a clinical setting.

The electric pulp tester has some limitations in providing predictable information about the status of the pulp. The response of the pulp to electric testing does not reflect the histologic health or disease status of the pulp.[122,123] A response by the pulp to the electric current only denotes that some viable nerve fibers are present in the pulp and are capable of responding. Numeric readings on the pulp tester have significance only if the number differs significantly from the readings obtained from a control tooth tested on the same patient with the electrode positioned at a similar area on both teeth. However, in most cases, the response is scored as either present or absent. Studies[122,123] have shown that electric pulp test results are most accurate when no response is obtained to any amount of electric current. This lack of response has been found most frequently when a necrotic pulp is present. In addition, false-positive and false-negative responses can occur (Box 1.4), and the clinician must take it into account when formulating the final diagnosis.

Box 1.4

Potential Common Interpretation Errors of Responses Obtained From Electric Pulp Testing

False-positive responses

- Partial pulp necrosis
- Patient's high anxiety
- Ineffective tooth isolation
- Contact with metal restorations

False-negative responses

- Calcific obliterations in the root canals
- Recently traumatized teeth
- Immature apex
- Drugs that increase patient's threshold for pain
- Poor contact of pulp tester to tooth

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The electric pulp tester will not work unless the probe can be placed in contact with or be bridged to the natural tooth structure.[95] With the advent of universal precautions for infection control, the use of rubber gloves prevents the clinician from completing the circuit.[7] Some pulp testers may require the patient to place a finger, or fingers, on the tester probe to complete the electric circuit; however, the use of lip clips is an alternative to having patients hold the tester. Proper use of the electric pulp tester requires the evaluated teeth to be carefully isolated and dried. A control tooth of similar tooth type and location in the arch should be tested first in order to establish a baseline response and to inform the patient as to what a "normal" sensation is. The suspected tooth should be tested at least twice to confirm the results. The tip of the testing probe that will be placed in contact with the tooth structure must be coated with a water- or petroleum-based medium.[86] The most commonly used medium is toothpaste. The coated probe tip is placed in the incisal third of the facial or buccal area of the tooth to be tested.[15] Once the probe is in contact with the tooth, the patient is asked to touch or grasp the tester probe, unless a lip clip is used (Fig. 1.20, *A*). This completes the circuit and initiates the delivery of an electric current to the tooth. The patient is instructed to remove his or her finger(s) from the probe when a "tingling" or "warming" sensation is felt in the tooth. The readings from the pulp tester are recorded (Fig. 1.20, *B*) and will be evaluated once all the appropriate teeth have been tested by the electric pulp tester and the other pulp testing methods.

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FIG. 1.20 **A,** Electric pulp tester with probe. The probe tip will be coated with a conducive medium, such as toothpaste, and placed in contact with the tooth surface. The patient will activate the unit by placing a finger on the metal shaft of the probe. **B,** View of the electric pulp tester control panel; the knob on the front right of the unit controls the rate at which the electric current is delivered to the tooth. The plastic panel on the left front displays the digital numerical reading obtained from the pulp test. The digital scale runs from 0 to 80.

Set of two photographs marked A and B depict components of an electric pulp tester with a probe.

Source: (Courtesy SybronEndo, Orange, CA.)

If a complete coverage crown or extensive restoration is present, a bridging technique can be attempted to deliver the electric current to any exposed natural tooth structure.[95] The tip of an endodontic explorer is coated with toothpaste or other appropriate medium and placed in contact with the natural tooth structure. The tip of the electric pulp tester probe is coated with a small amount of toothpaste and placed in contact with the side of the explorer. The

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patient completes the circuit and the testing proceeds as described previously. If no natural tooth structure is available, then an alternative pulp testing method, such as cold, should be used.

One study compared the ability of thermal and electric pulp testing methods to register the presence of vital pulp tissue.[99] The *sensibility*, which is the ability of a test to identify teeth that are diseased, was 0.83 for the cold test, 0.86 for heat test, and 0.72 for the electric test. This means the cold test correctly identified 83% of the teeth that had a necrotic pulp, whereas heat tests were correct 86% of the time and electric pulp tests were correct only 72% of the time. This same study evaluated the *specificity* of these three tests. Specificity relates to the ability of a test to identify teeth without disease. Ninety-three percent of teeth with healthy pulps were correctly identified by both the cold and electric pulp tests, whereas only 41% of the teeth with healthy pulps were identified correctly by the heat test. From the results of the testing, it was found that the cold test had an accuracy of 86%, the electric pulp test 81%, and the heat test 71%.

Some studies have indicated there might not be a significant difference between pulp testing results obtained by electric pulp tester and those obtained by the thermal methods.[46,98,99] However, cold tests have been shown to be more reliable than electric pulp tests in younger patients with less developed root apices.[5,42,98] This is the reason to verify the results obtained by one testing method and compare them with results obtained by other methods. Until such time that the testing methods used to assess the vascular supply of the pulp become less time consuming and technique sensitive, thermal and electric pulp testing will continue to be the primary methods for determining pulp sensibility.

Laser doppler flowmetry

Laser Doppler flowmetry (LDF) is a method used to assess blood flow in microvascular systems. Attempts are being made to adapt this technology to assess pulpal blood flow. A diode is used to project an infrared light beam through the crown and pulp chamber of a tooth. The infrared light beam is scattered as it passes through the pulp tissue. The Doppler principle states that the light beam's frequency will shift when hitting moving red blood cells but will remain unshifted as it passes through static tissue. The average

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Doppler frequency shift will measure the velocity at which the red blood cells are moving.[114]

Several studies[40,60,69,84,114,115,117] have found LDF to be an accurate, reliable, and reproducible method of assessing pulpal blood flow. One of the great advantages of pulp testing with devices such as the LDF is that the collected data are based on objective findings rather than subjective patient responses. As is discussed in Chapter 21, certain luxation injuries will cause inaccuracies in the results of electric and thermal pulp testing. LDF has been shown to be a great indicator for pulpal vitality in these cases.[130] However, this technology is not being used routinely in the dental practice.

Pulse oximetry

The pulse oximeter is another noninvasive device (Fig. 1.21). Widely used in medicine, it is designed to measure the oxygen concentration in the blood and the pulse rate. A pulse oximeter works by transmitting two wavelengths of light, red and infrared, through a translucent portion of a patient's body (e.g., a finger, earlobe, or tooth). Some of the light is absorbed as it passes through the tissue; the amount absorbed depends on the ratio of oxygenated to deoxygenated hemoglobin in the blood. On the opposite side of the targeted tissue, a sensor detects the absorbed light. On the basis of the difference between the light emitted and the light received, a microprocessor calculates the pulse rate and oxygen concentration in the blood.[118] The transmission of light to the sensor requires that there be no obstruction from restorations, which can sometimes limit the usefulness of pulse oximetry to test the status of the pulp tissue.

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FIG. 1.21 Nellcor OxiMax N-600x pulse oximeter.

A digital pulse oximeter is shown. The plastic panel records pulse with a highest reading of 99 and lowest reading of 65 on the left.

Source: (Courtesy Nellcor Puritan Bennett, Boulder, CO; now part of Covidien.)

Custom-made sensors have been developed and were found to be more accurate than electric and thermal pulp tests.[31,54] This sensor has been especially useful in evaluating teeth that have been subjected to traumatic injuries, as such teeth tend to present, especially in the short term, with questionable neural response using conventional pulp testing methods.[8,31,53]

Studies regarding the ability of pulse oximetry to diagnose pulp health have drawn various conclusions. Several studies have found pulse oximetry to be a reliable method for assessing pulp health.[69,70,118,125,140] Others have stated that in its present form the pulse oximeter may not be predictable for such diagnoses.[140] Most of the problems appear to be related to the currently available technology. Some investigators have concluded that the devices used for pulp testing are too cumbersome and complicated to be used on a routine basis in a dental practice.[68,118,140]

Special tests

Bite test

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Bite tests and percussion tests are indicated when a patient presents with pain while biting. On occasion, the patient may not know which tooth is sensitive to biting pressure, and percussion and bite tests may help to localize the tooth involved. The tooth may be sensitive to biting when the pulpal pathosis has extended into the periodontal ligament space, creating a *symptomatic apical periodontitis,* or the sensitivity may be present secondary to a crack in the tooth. The clinician can often differentiate between periradicular periodontitis and a cracked tooth or fractured cusp. If periradicular periodontitis is present, the tooth will respond with pain to percussion and biting tests regardless of where the pressure is applied to the coronal part of the tooth. A cracked tooth or fractured cusp will typically elicit pain only when the bite or percussion test is applied in a certain direction to one cusp or section of the tooth.[22,108]

For the bite test to be meaningful, a device should be used that allows the clinician to apply pressure to individual cusps or areas of the tooth. A variety of devices have been used for bite tests, including cotton tip applicators, toothpicks, orangewood sticks, and rubber polishing wheels. There are several devices specifically designed to perform this test. The Tooth Slooth (Professional Results, Laguna Niguel, CA) (Fig. 1.22) and FracFinder (Hu-Friedy, Oakbrook, IL) are just two of the commercially available devices used for the bite test. As with all pulp tests, adjacent and contralateral teeth should be used as controls so that the patient is aware of the “normal” response to these tests. The small cupped-out area on these instruments is placed in contact with the cusp to be tested. The patient is then asked to apply biting pressure with the opposing teeth to the flat surface on the opposite side of the device. The biting pressure should be applied slowly until full closure is achieved. Firm pressure should be applied for a few seconds; the patient is then asked to release the pressure quickly. Each individual cusp on a tooth can be tested in a similar manner. The clinician should note whether the pain is elicited during the pressure phase or on quick release of the pressure. A common finding with a fractured cusp or cracked tooth is the frequent presence of pain upon release of biting pressure.

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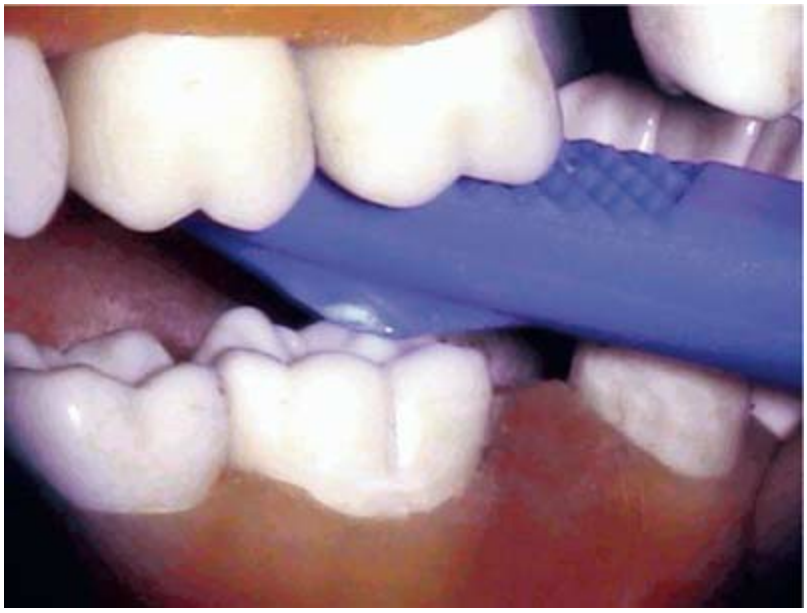

FIG. 1.22 To determine which tooth, or tooth part, is sensitive to mastication, having the patient bite on a specially designed bite stick is often helpful.

Close-up view shows a plastic bite stick between patient's teeth.

Test cavity

The test cavity method for assessing pulpal response is not routinely used since, by definition, it is an invasive irreversible test. This method is used only when all other test methods are deemed impossible or the results of the other tests are inconclusive. An example of a situation in which this method can be used is when the tooth suspected of having pulpal disease has a full coverage crown. If no sound tooth structure is available to use a bridging technique with the electric pulp tester, and the cold test results are inconclusive, a small class I cavity preparation is made through the occlusal surface of the crown. This is accomplished with a high-speed #1 or #2 round bur with proper air and water coolant. The patient is not anesthetized while this procedure is performed, and the patient is asked to respond if any painful sensation is felt during the drilling procedure. If the patient feels pain once the bur contacts sound dentin, the procedure is terminated and the class I cavity preparation is restored. This sensation signifies only that there is some viable nerve tissue remaining in the pulp, not that the pulp is totally healthy. If the patient fails to feel any sensation when the bur reaches the dentin, this

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is a good indication that the pulp is necrotic and root canal therapy is indicated. The patient should be given a full explanation and reassurance of the procedure before the test cavity is attempted.

Staining and transillumination

To determine the presence of a crack in the surface of a tooth, the application of a stain to the area is often of great assistance. It may be necessary to remove the restoration in the tooth to better visualize a crack or fracture. Methylene blue dye, when painted on the tooth surface with a cotton tip applicator, will penetrate into cracked areas. The excess dye may be removed with a moist application of 70% isopropyl alcohol. The remaining dye will indicate the possible location of the crack.

Transillumination using a bright fiberoptic light probe to the surface of the tooth may be very helpful (Fig. 1.23). Directing a high-intensity light directly on the exterior surface of the tooth at the cementum-enamel junction (CEJ) may reveal the extent of the fracture. Teeth with fractures block transilluminated light. The part of the tooth that is proximal to the light source will absorb this light and glow, whereas the area beyond this fracture will not have light transmitted to it and will show dark by comparison.[101] Although the presence of a fracture may be evident using dyes and transillumination, the full extent of the fracture cannot always be determined by these tests alone.

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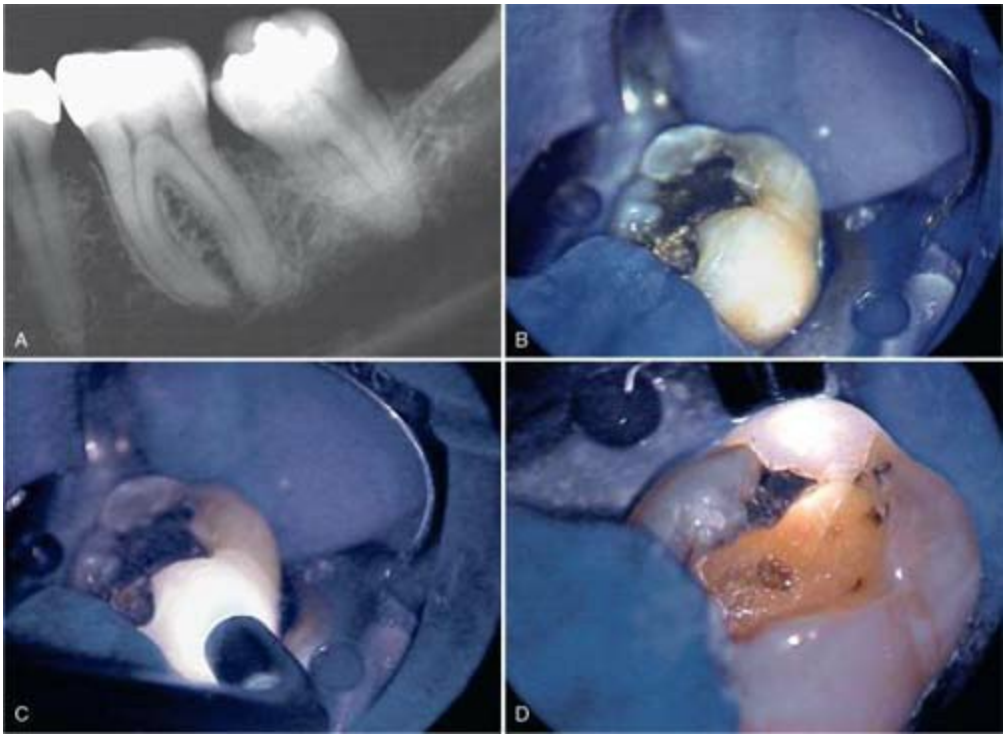

FIG. 1.23 Sometimes there is no clear indication of why a tooth is symptomatic. This radiograph shows a mandibular second molar with a moderately deep restoration **(A);** the pulp tests nonvital. Without any transillumination, a fracture cannot be detected **(B).** However, by placing a high-intensity light source on the tooth surface, a root fracture can be observed on the buccal surface **(C)** and the distal-lingual surface **(D).**

A) Radiograph shows a radiolucent region in and around the root of second molar.

B) Close-up view of tooth isolated by rubber dam clamp shows a filling in the cavity.

C) Close-up view shows the isolated tooth being examined under a light source.

D) Close-up view shows yellow and soft tissue of the fractured tooth. The enamel layer is peeled off at the edge.

Selective anesthesia

When symptoms are not localized or referred, the diagnosis may be challenging. Sometimes the patient may not even be able to specify whether the symptoms are emanating from the maxillary or mandibular arch. In these

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instances, when pulp testing is inconclusive, *selective anesthesia* may be helpful.

If the patient cannot determine which arch the pain is coming from, then the clinician should first selectively anesthetize the maxillary arch. This should be accomplished by using a periodontal ligament (intraligamentary) injection. The injection is administered to the most posterior tooth in the quadrant of the arch that may be suspected, starting from the distal sulcus. The anesthesia is subsequently administered in an anterior direction, one tooth at a time, until the pain is eliminated. If the pain is not eliminated after an appropriate period of time, then the clinician should similarly repeat this technique on the mandibular teeth below. It should be understood that periodontal ligament injections may anesthetize an adjacent tooth and thus are more useful for identifying the arch rather than the specific tooth.

Radiographic examination and interpretation

Intraoral radiographs

The radiographic interpretation of a potential endodontic pathosis is an integral part of endodontic diagnosis and prognosis assessment. Few diagnostic tests provide as much useful information as dental radiography. For this reason, the clinician is sometimes tempted to prematurely make a definitive diagnosis based solely on radiographic interpretation. However, the image should be used only as one sign, providing important clues in the diagnostic investigation. When not coupled with a proper history and clinical examination and testing, the radiograph alone can lead to a misinterpretation of normality and pathosis (Fig. 1.24). Because treatment planning will ultimately be based on the diagnosis, the potential for inappropriate treatment may frequently exist if the radiograph alone is used for making final diagnosis. The clinician should not subject the patient to unnecessary multiple radiation exposures; two pretreatment images from different angulations are often sufficient. However, under extenuating circumstances—especially when the diagnosis is difficult—additional exposures may be necessary to determine the presence of multiple roots, multiple canals, resorptive defects, caries, restoration defects, root fractures, and the extent of root maturation and apical development.

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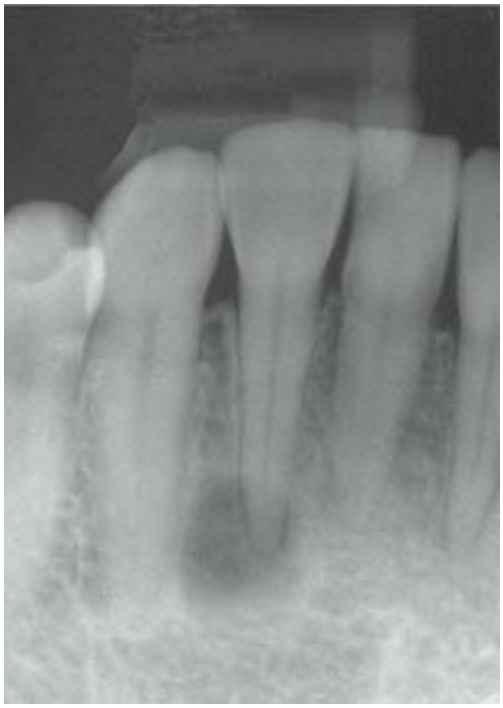

FIG. 1.24 Radiograph showing what appears to be a mandibular lateral incisor associated with periapical lesion of a nonvital tooth. Although pulp necrosis can be suspected, the tooth tested vital. In this case, the appearance of apical bone loss is secondary to a cementoma.

Radiograph shows a dark and roughly triangular patch at the root canal of incisor, with blur image of two tooth to the left of patch.

The radiographic appearance of endodontic pathosis can sometimes be highly subjective. In a study by Goldman and colleagues, there was only 50% agreement among interpreters for the radiographic presence of pathosis.[49] When the cases were reevaluated several months later, the same evaluators agreed with their own original diagnosis less than 85% of the time.[50] This further emphasizes the necessity for additional objective diagnostic tests, as well as the importance of obtaining and comparing older radiographs of the same area of the patient.

For standard two-dimensional radiography, clinicians basically project x-radiation through an object and capture the image on a recording medium, either x-ray film or a digital sensor. Much like casting a shadow from a light source, the image appearance may vary greatly depending on how the radiographic source is directed. Thus, the three-dimensional interpretation of the resulting two-dimensional image requires not only knowledge of

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normality and pathosis but also advanced knowledge of how the radiograph was exposed. By virtue of "casting a shadow," the anatomic features that are closest to the film (or sensor) will move the least when there is a change in the horizontal or vertical angulation of the radiation source (Fig. 1.25). This may be helpful in determining the existence of additional roots, the location of pathosis, and the unmasking of anatomic structures. Changes in the horizontal or vertical angulation may help elucidate valuable anatomic and pathologic information; it also has the potential to hide important information. An incorrect vertical angulation may cause the buccal roots of a maxillary molar to be masked by the zygomatic arch. An incorrect horizontal angulation may cause roots to overlap with the roots of adjacent teeth, or it may incorrectly create the appearance of a one-rooted tooth, when two roots are actually present.

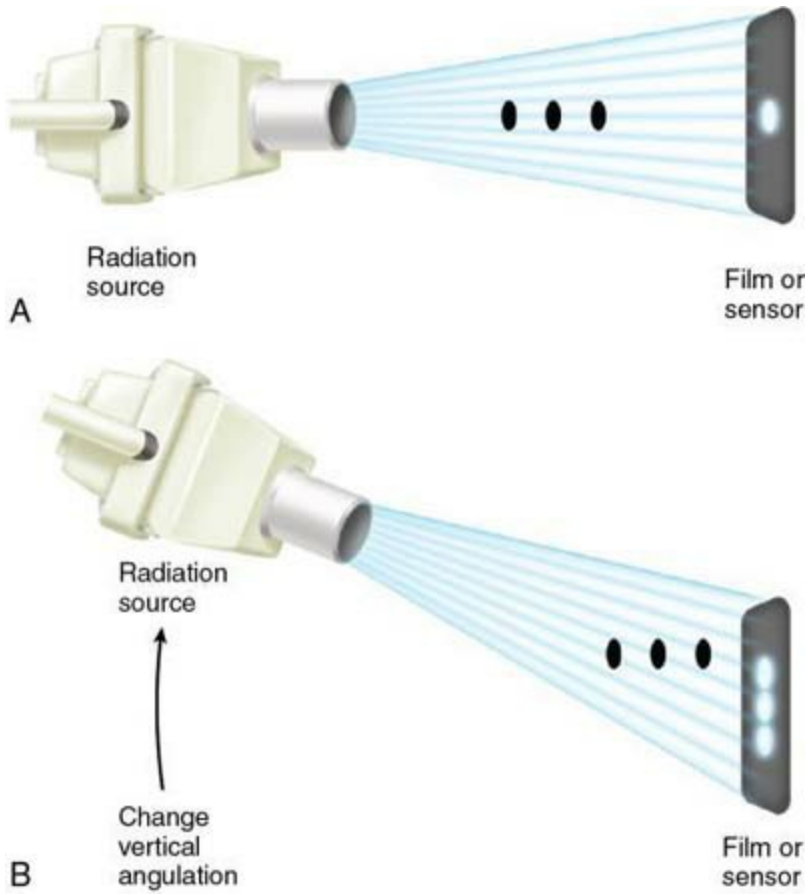

FIG. 1.25 Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. **A,** When the source of radiation is directly perpendicular to overlapping

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objects, the image is captured without much separation of the objects. However, when the radiation source is at an angle to offset the overlapping objects, the image is captured with the objects being viewed as separated. **B,** The object that is closest to the film (or sensor) will move the least, with the object closest to the radiation source appearing farthest away.

Set of two illustrations marked A and B depict ways that may lead to casting of shadow resulting in changes in inaccurate diagnosis.

In general, when endodontic pathosis appears radiographically, it appears as a radiolucency in the area of the periapex. The pathosis may present merely as a widening or break in the lamina dura—the most consistent radiographic finding when a tooth is nonvital[67]—or it may present as a radiolucent area at the apex of the root or in the alveolar bone adjacent to the exit of a lateral or furcation accessory canal. On occasion no radiographic change can be seen at all, even in the presence of a disease process in the alveolar bone. This is mainly due to the fact that the disease process did not reach the cortical plate of the bone.

Two-dimensional dental radiography has two basic shortcomings: the lack of early detection of pathosis in the cancellous bone, because of the density of the cortical plates, and the influence of the superimposition of anatomic structures. Variability in the radiographic expression of an osseous pathosis has much to do with the relative location of the root of the tooth and how it is oriented with respect to the cortical and cancellous bone. Radiographic changes from bone loss will not be detected if the loss is only in cancellous bone.[16] However, the radiographic evidence of pathosis will be observed once this bone loss extends to the junction of the cortical and cancellous bone. In addition, certain teeth are more prone to exhibit radiographic changes than others, depending on their anatomic location.[17] The radiographic appearance of endodontic pathosis is correlated with the relationship of the periapex of the tooth and its juxtaposition to the cortical-cancellous bone junction. The apices of most anterior and premolar teeth are located close to the cortical-cancellous bone junction. Therefore, periapical pathosis from these teeth is exhibited sooner on the radiograph. By comparison, the distal roots of mandibular first molars and both roots of mandibular second molars are generally positioned more centrally within the cancellous bone, as are maxillary molars, especially the palatal roots.

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Periapical lesions from these roots must expand more before they reach the cortical-cancellous bone junction and are recognized as radiographic pathosis. For these reasons, it is important not to exclude the possibility of pulpal pathosis in situations in which there are no radiographic changes.

Many factors can influence the quality of the radiographic interpretation, including the ability of the person exposing the radiograph, the quality of the radiographic film, the quality of the exposure source, the quality of the film processing, and the skill with which the film is viewed. Controlling all of these variables can be a difficult challenge but is paramount for obtaining an accurate radiographic interpretation.

Digital radiography

Digital radiography has been available since the late 1980s and has recently been refined and popularized with better hardware and a more user-friendly interface. It has the ability to capture, view, magnify, enhance, and store radiographic images in an easily reproducible format that does not degrade over time. Significant advantages of digital radiographs over conventional radiographs include lower radiation doses, instant viewing, convenient manipulation, efficient transmission of an image via the Internet, simple duplication, and easy archiving.

Digital radiography uses no x-ray film and requires no chemical processing. Instead, a *sensor* is used to capture the image created by the radiation source. This sensor is either directly or wirelessly attached to a local computer, which interprets this signal and, using specialized software, translates the signal into a two-dimensional digital image that can be displayed, enhanced, and analyzed. The image is stored in the patient's file, typically in a dedicated network server, and can be recalled as needed. Further information about digital radiography may be found in Chapter 2.

The viewing of a digital radiographic image on a high-resolution monitor allows for rapid and easy interpretation for both the clinician and the patient. The image appears almost instantly, with no potential for image distortion from improper chemical processing. The clinician can magnify different areas on the radiograph and then digitally enhance the image in order to better visualize certain anatomic structures; in some cases, the image can even be colorized, which is a useful tool for patient education (Fig. 1.26).

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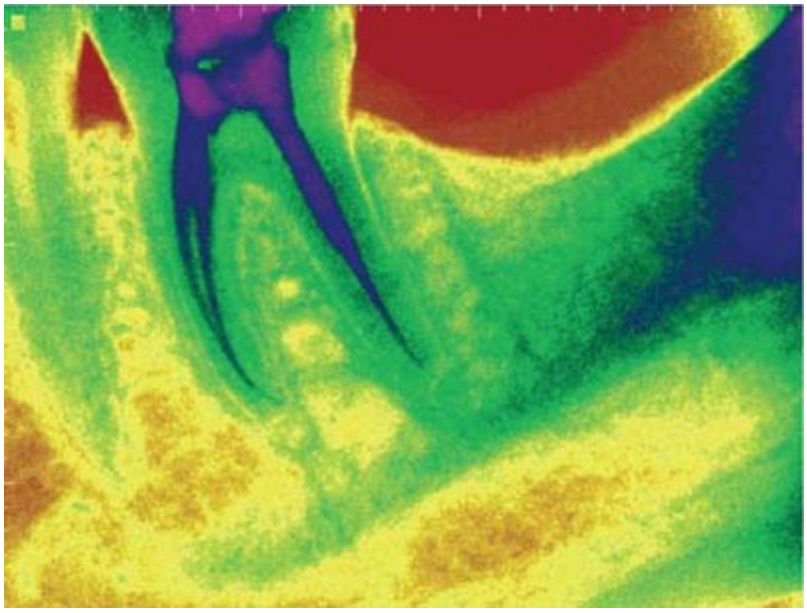

FIG. 1.26 Digital radiography has an advantage over conventional film in that the image can be enhanced and colorized—a useful tool for patient education.

Multi-colored radiograph shows blue colored thin-density tissue in the root and a little space between root and neck present on a greenish-yellow background.

In the past, x-ray film has had a slightly better resolution than most digital radiography images, at about 16 line pairs per millimeter (lp/mm).[87] However, current sensors offer resolutions beyond that of conventional film. Under the best of circumstances, the human eye can see only about 10 lp/mm, which is the lowest resolution for most dental digital radiography systems. Digital sensors are much more sensitive to radiation than conventional x-ray film and thus require 50% to 90% less radiation in order to acquire an image, an important feature for generating greater patient acceptance of dental radiographs.

The diagnostic quality of this technology has been shown to be comparable to conventional film-based radiography.[39,73,97] The interpretation of a digital radiograph can be subjective, similar to that of the conventional film.[134] Factors that appear to have the most impact on the interpretation of the image are the years of experience of the examiner and familiarity of the operator with the given digital system.[134]

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Cone-beam computerized tomography

Limitations in conventional two-dimensional radiography promulgated a need for three-dimensional imaging, known as *cone-beam computerized tomography* (CBCT) (also known as *cone-beam volumetric tomography* [CBVT]) or as *cone-beam volumetric imaging* [CBVI]. Although a form of this technology has existed since the early 1980s,[106] specific devices for dental use first appeared almost two decades later.[90] Most of these machines are similar to a dental panoramic radiographic device, whereby the patient stands or sits as a cone-shaped radiographic beam is directed to the target area with a reciprocating capturing sensor on the opposite side (Fig. 1.27). The resulting information is digitally reconstructed and interpreted to create an interface whereby the clinician can three-dimensionally interpret "slices" of the patient's tissues in a multitude of planes (Figs. 1.28 and 1.29).[37,33] The survey of the scans can be interpreted immediately after the scan. Various software applications have been used to enable the images to be sent to other clinicians. This is accomplished either in printed format or with portable and transferable software that can be used interactively by another clinician.

FIG. 1.27 Cone-beam volumetric tomography, using the 3D Accuitomo 80.

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A 3D-Accuitomo 80 CBCT appearing as rectangular framework with a chair inside.

Source: (Courtesy J. Morita USA, Irvine, CA.)

FIG. 1.28 Cone-beam volumetric tomography has the ability to capture, store, and present radiographic images in various horizontal and vertical planes.

Lateral view of human shows X-ray beams interpreting the mandible tissues by forming a multitude of planes.

Source: (Courtesy J. Morita USA, Irvine, CA.)

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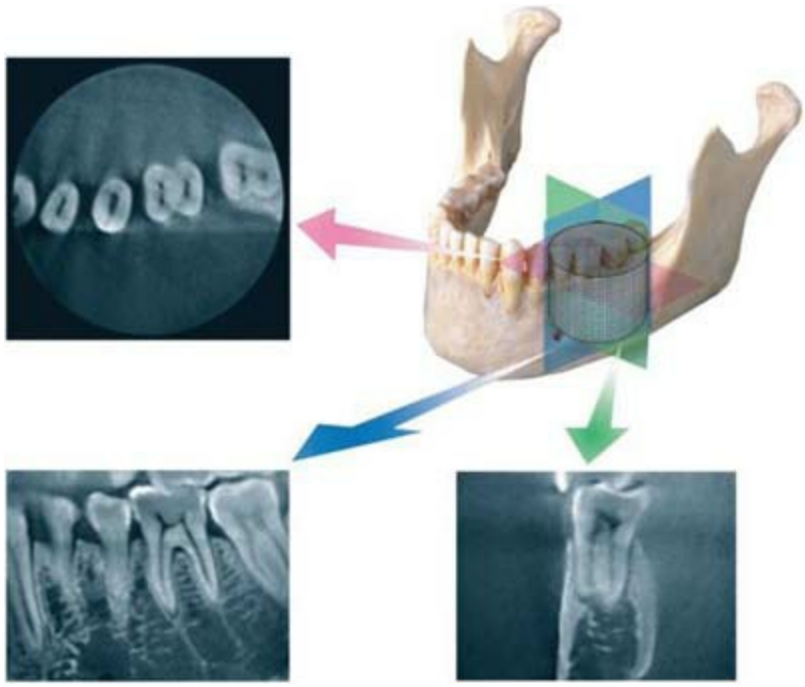

FIG. 1.29 Cone-beam volumetric tomography has the advantage of being able to detect pathosis in the bone or associated with the teeth without the obstruction of anatomic structures. The planes of vision may be axial, sagittal, or coronal.

Three insets marked from the lower jaw bone leads to radiographs as follows: 1) An occlusal view of premolar or canine with cavities inside. 2) Close-up view of teeth with radiolucent pulp and root canals. 3) A molar teeth with less-density tissue between crown and root.

Source: (Courtesy J. Morita USA, Irvine, CA.)

In general, many dental applications only require a limited field of vision, confining the study to the maxilla and mandible. However, many devices have the ability to provide a full field of vision for viewing more regional structures. Clinicians should thoroughly understand the ethical and medical-legal ramifications of doing scans with full fields of view. Incidental nondental findings have been seen from these scans, such as intracranial aneurysms, which, when undetected, could be life threatening.[91]

The radiation source of CBCT is different from that of conventional two-dimensional dental imaging in that the radiation beam created is conical in shape. Also, conventional digital dental radiography is captured and interpreted as *pixels*, a series of dots that collectively produces an image of the scanned structure. For CBCT, the image is instead captured as a series of

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three-dimensional pixels, known as *voxels.* Combining these voxels gives a three-dimensional image that can be "sliced" into various planes, allowing for specific evaluations without a necropsy (Fig. 1.30). One of the advantages of using a device that has a limited field of vision is that the voxel size can be less than half that of a device using a full field of vision, thereby increasing the resolution of the resulting image and providing for a more accurate interpretation of anatomic structures and pathologic conditions. The development of limited field of vision devices has also contributed to decreasing the radiation and cost of these machines, making them more practical for endodontic use.[41]

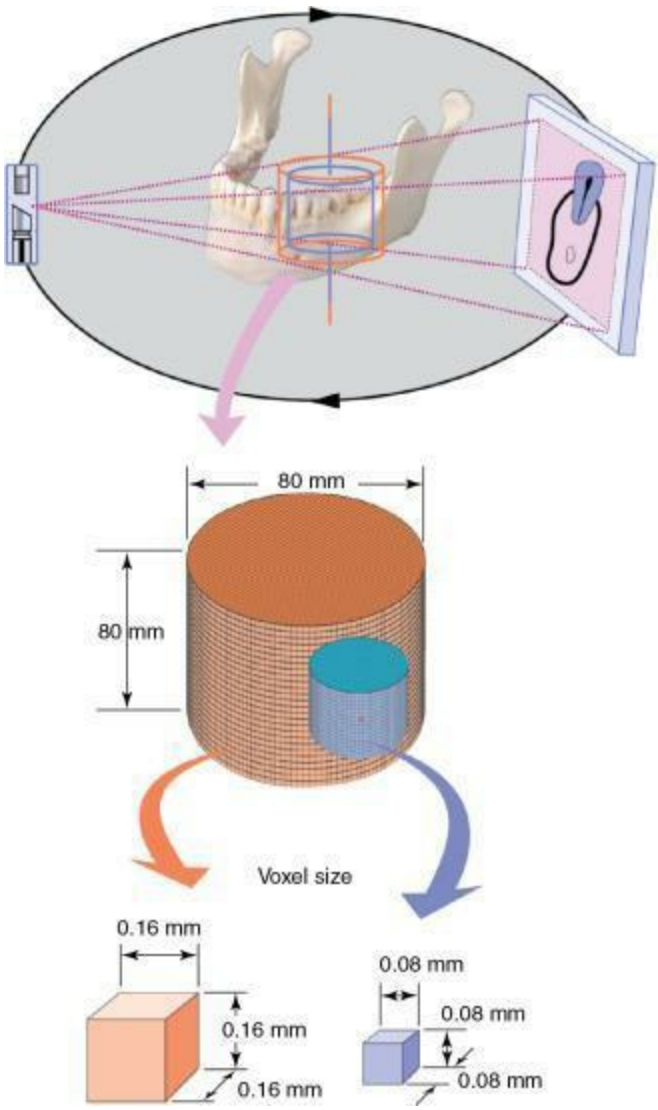

FIG. 1.30 The radiation source in cone-beam volumetric tomography

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is conical. The receiving sensor captures the image as “voxels,” or three-dimensional pixels of information, allowing digital interpretation.

Side view of lower jaw bone shows the analysis of mandible quadrant using CBCT. Cone shaped X-ray beams produce the image of tooth with its root canal on the left. An arrow from the lower jaw bone on the left leads to a large cylindrical structure with a smaller cylindrical structure inside indicating 3D voxel size. The height and breadth of the large cylindrical structure are each 80 millimeters. Two arrows from the 3D voxel size leads to a larger and smaller cuboid. The larger cuboid has dimensions of 0.16 times 0.16 times 0.16 millimeters. The smaller cuboid has dimensions of 0.8 times 0.8 times 0.8 millimeters.

Compared with two-dimensional radiographs, CBCT can clearly visualize the interior of the cancellous bone without the superimposition of the cortical bone. Studies show that CBCT is much more predictable and efficient in demonstrating anatomic landmarks, bone density, bone loss, periapical lesions, root fractures, root perforations, and root resorptions.[1,21,26,27,38,47,71,78,81,85,92,94,128,131,142]

The superimposition of anatomic structures can also mask the interpretation of alveolar defects. Specifically, the maxillary sinus, zygoma, incisive canal and foramen, nasal bone, orbit, mandibular oblique ridge, mental foramen, mandibular mentalis, sublingual salivary glands, tori, and the overlap of adjacent roots may either obscure bone loss or mimic bone loss, making an accurate interpretation of conventional radiography sometimes difficult or impossible. Several studies have demonstrated the advantages of CBCT in the differential diagnosis of such structures from pathologic conditions.[21,29,71,137]

CBCT should not be seen as a replacement for conventional dental radiography, but rather as a diagnostic adjunct. The advantage of conventional dental radiography is that it can visualize most of the structures in one image. CBCT can show great detail in many planes of vision but can also leave out important details if the “slice” is not in the area of existing pathosis (Fig. 1.31). There is a promising future for the use of CBCT for endodontic diagnosis and treatment. It has already proven invaluable in the detection of dental and nondental pathoses (Fig. 1.32). For a further review of CBCT and radiography, see Chapter 2.

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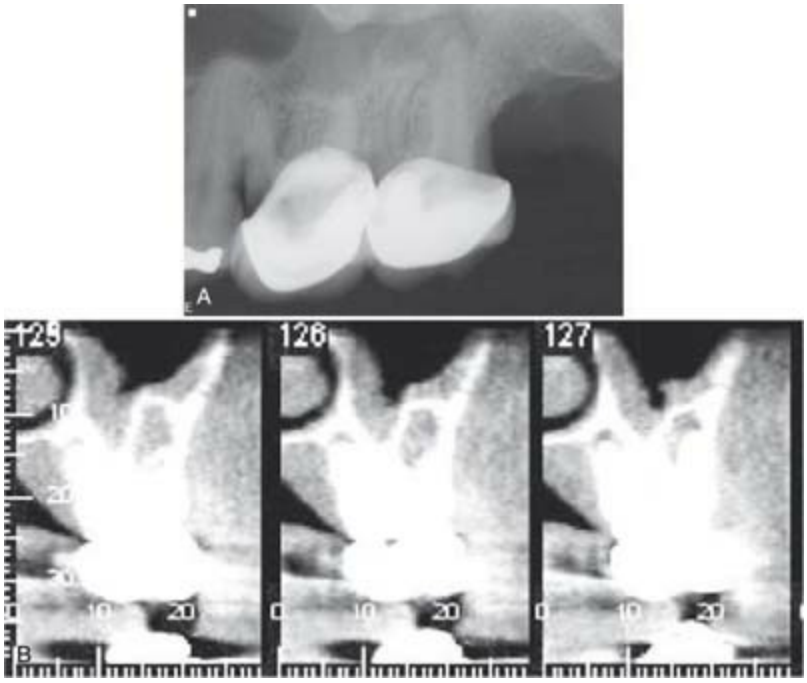

FIG. 1.31 **A,** This standard two-dimensional radiographic image reveals recurrent caries under the mesial margin of the maxillary first molar. However, the patient localized pain to mastication on the maxillary second molar. **B,** Cone-beam volumetric tomography revealed an apical radiolucency associated with the maxillary second molar. The bone loss was obscured on the two-dimensional radiograph by the maxillary sinus, zygoma, and cortical bone.

A) Radiograph shows two teeth with loss of tissue under the mesial margin.

B) Three radiograph show gradual changes in the tissue-density near the root apex. The third radiograph has smaller radiolucent patch.

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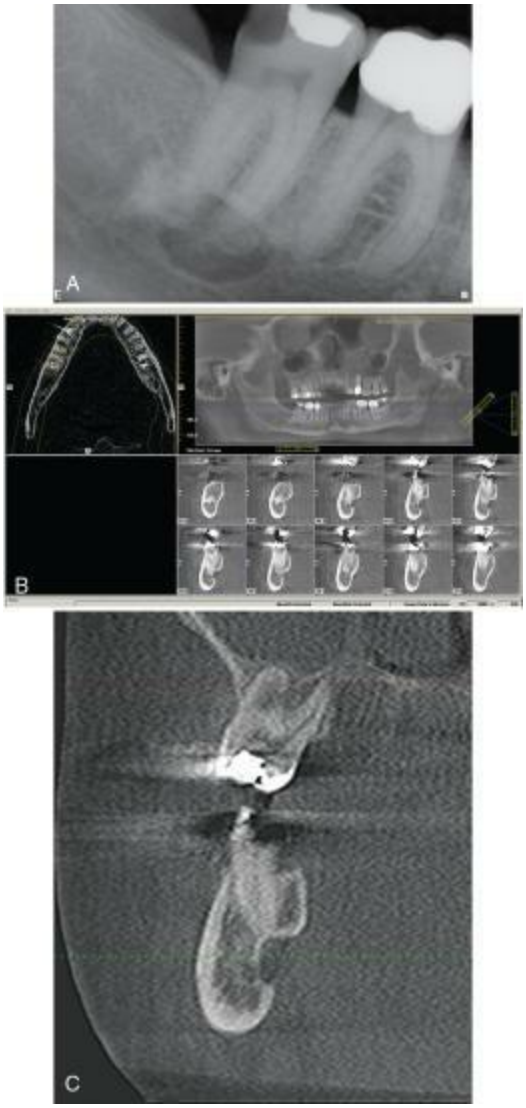

FIG. 1.32 **A,** Periapical radiograph showing a large apical radiolucency associated with the mandibular second molar. Apical pathosis should be ruled out. **B,** Cone-beam volumetric tomography revealed salivary indentation of the mandible in the area apical and lingual to the mandibular second molar, consistent with a Stafne defect. **C,** Enlargement of coronal section in the area of the mandibular second molar and the Stafne defect located on the lingual aspect of the mandible.

Set of three radiographs marked A through C compares periapical radiograph with cone beam computed tomograph for mandibular second molar.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) has also been suggested for dental

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diagnosis. It may offer simultaneous three-dimensional hard- and soft-tissue imaging of teeth without ionizing radiation.[58] The use of MRI in endodontics is still limited.

Cracks and fractures

The wide variety of types of cracks and fractures in teeth and their associated signs and symptoms often make their diagnosis difficult. The extensiveness of the crack or fracture line may directly alter the prognosis assessment for a given tooth and should be examined before treatment decision making. Certain types of cracks may be as innocent as a superficial enamel craze line, or they may be as prominent as a fractured cusp. The crack may progress into the root system to involve the pulp, or it may split the entire tooth into two separate segments. The crack may be oblique, extending cervically, such that once the coronal segment is removed the tooth may or may not be restorable. Any of these situations may present with mild, moderate, or severe symptoms or possibly no symptoms at all.

Crack types

There have been many suggestions in the literature of how to classify cracks in teeth. By defining the type of crack present, an assessment of the prognosis may be determined and treatment alternatives can be planned. Unfortunately, it is often extremely difficult to determine how extensive a crack is until the tooth is extracted.

Cracks in teeth can be divided into three basic categories:

- Craze lines
- Fractures (also referred to as *cracks*)
- Split tooth/roots

Craze lines are merely cracks in the enamel that do not extend into the dentin and either occur naturally or develop after trauma. They are more prevalent in adult teeth and usually occur more in the posterior teeth. If light is transilluminated through the crown of such a tooth, these craze lines may show up as fine lines in the enamel with light being able to transmit through them, indicating that the crack is only superficial. The use of optical

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coherence tomography (OCT) has also been suggested for the detection of enamel cracks.[59] Craze lines typically will not manifest with symptoms. No treatment is necessary for craze lines unless they create a cosmetic issue.

Fractures extend deeper into the dentin than superficial craze lines and primarily extend mesially to distally, involving the marginal ridges. Dyes and transillumination are helpful for visualizing potential root fractures.

Symptoms from a fractured tooth range from none to severe pain. A fracture in the tooth does not necessarily indicate that the tooth has split into two pieces; however, left alone or especially with provocations such as occlusal prematurities, the fracture may progress into a split root. A fractured tooth may be treated by a simple restoration, endodontics (nonsurgical or surgical), or even extraction, depending on the extent and orientation of the fracture, the degree of symptoms, and whether the symptoms can be eliminated. This makes the clinical management of fractured teeth difficult and sometimes unpredictable.

A definitive combination of factors, signs, and symptoms that, when collectively observed, allows the clinician to conclude the existence of a specific disease state is termed a *syndrome*. However, given the multitude of signs and symptoms that fractured roots can present with, it is often difficult to achieve an objective definitive diagnosis. For this reason, the terminology of *cracked tooth syndrome*[22,108] should be avoided.[6] The subjective and objective factors seen in cases of fractured teeth will generally be diverse; therefore, a tentative diagnosis of a fractured tooth will most likely be more of a prediction. Once this prediction is made, the patient must be properly informed as to any potential decrease in prognosis of the pending dental treatment. Because treatment options for repairing fractured teeth have only a limited degree of success, early detection and prevention, and proper informed consent, are crucial.[9,10,72,119,120,124,132]

Split tooth/roots occur when a fracture extends from one separating them into two segments. If the split is more oblique, it is possible that once the smaller separated segment is removed, the tooth might still be restorable—for example, a fractured cusp. However, if the split extends below the osseous level, the tooth may not be restorable and endodontic treatment may not result in a favorable prognosis.

Proper prognosis assessment is imperative before any dental treatment but

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is often difficult in cases of cracked teeth. Because of the questionable long-term success from treating cases of suspected or known fractures, the clinician should be cautious in making the decision to continue with treatment and should avoid endodontic treatment in cases of a definitive diagnosis of a split tooth or root.

Vertical root fractures

One of the more common reasons for recurrent endodontic pathosis is the *vertical root fracture*, a severe crack in the tooth that extends longitudinally down the long axis of the root (Figs. 1.33 and 1.34). Often it extends through the pulp and to the periodontium. It tends to be more centrally located within the tooth, as opposed to being more oblique, and typically traverses through the marginal ridges. These fractures may be present before endodontic treatment, secondary to endodontic treatment, or may develop after endodontic treatment has been completed. Because diagnosing these vertical root fractures may be difficult, they often go unrecognized. Therefore, diagnosing the existence and extent of a vertical root fracture is imperative before any restorative or endodontic treatment is done, as it can dramatically affect the overall success of treatment.

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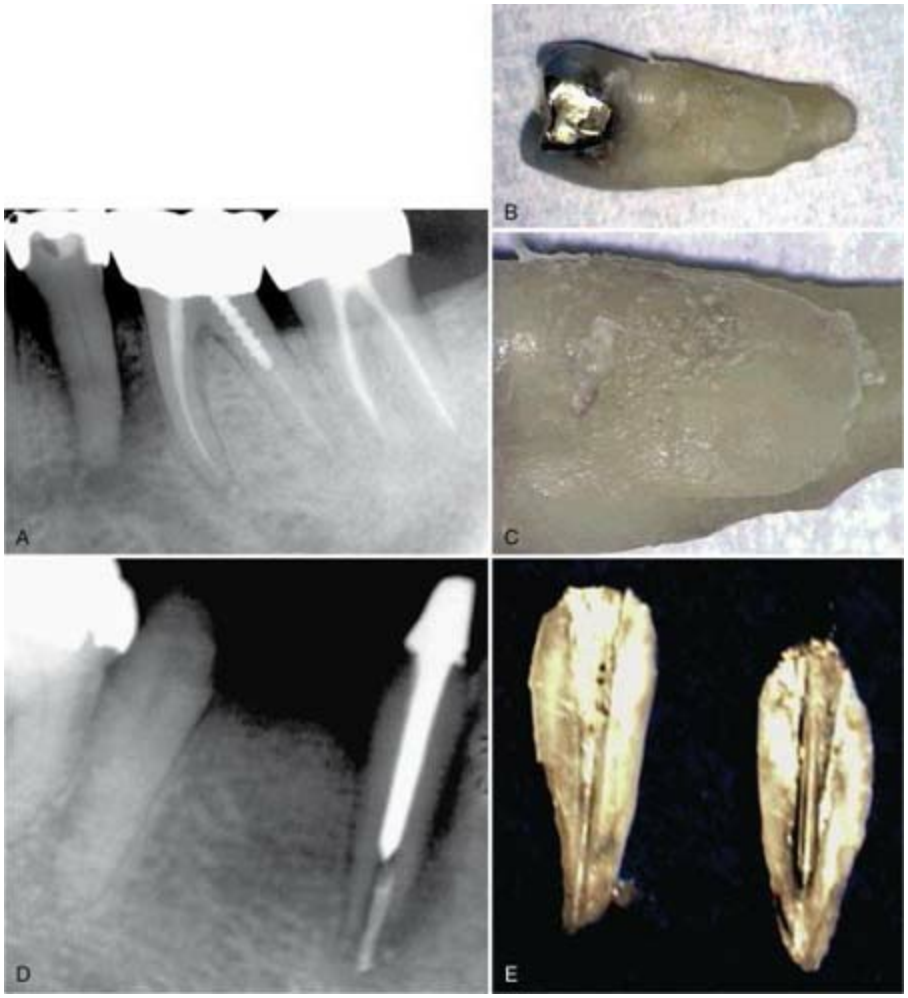

FIG. 1.33 Poorly fitting intracoronal restorations can place stresses within the tooth that can cause a vertical root fracture. **A,** This radiograph of a mandibular second premolar (with a gold inlay) reveals extensive periapical and periradicular bone loss, especially on the distal aspect. **B,** The tooth pulp tested nonvital, and there was an associated 12-mm-deep, narrow, isolated periodontal pocket on the buccal aspect of the tooth. After the tooth was extracted, the distal aspect was examined. **C,** On magnification (×16) the distal aspect of the root revealed an oblique vertical root fracture. Similarly, the placement of an ill-fitting post may exert intraradicular stresses on a root that can cause a fracture to occur vertically. **D,** This radiograph depicts a symmetrical space between the obturation and the canal wall, suggesting a vertical root fracture. **E,** After the tooth is extracted, the root fracture can be easily observed.

Set of five radiographs marked A through E depicts stress of poorly fitted intracoronal restorations resulting in a vertical root fracture.

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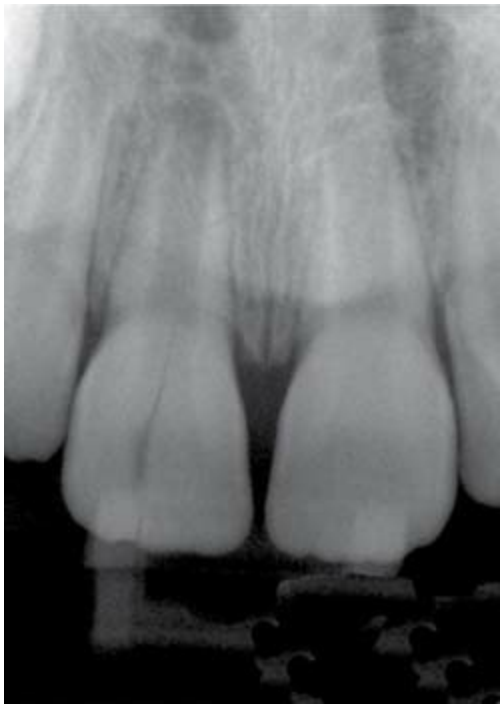

FIG. 1.34 Physical trauma from sports-related injuries or seizure-induced trauma, if directed accordingly, may cause a vertical root fracture in a tooth. This fracture occurred in a 7-year-old child secondary to trauma from a grand mal seizure.

Radiograph of two teeth shows teeth on the left having a slant line of fracture extended toward the apex.

A patient who consents to endodontic treatment must be informed if the tooth has a questionable prognosis. The clinician must be able to interpret the subjective and objective findings that suggest a vertical root fracture or split tooth, be able to make a prediction as to the eventual potential of healing, and convey this information to the patient. A more detailed discussion on vertical root fractures is described in Chapter 22.

Perforations

Root perforations are clinical complications that may lead to treatment failure. When root perforation occurs, communications between the root canal system and either periradicular tissues or the oral cavity may reduce the prognosis of treatment. Root perforations may result from extensive carious lesions, resorption, or operator error occurring during root canal instrumentation or post preparation.

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The treatment prognosis of root perforations depends on the size, location, time of diagnosis and treatment, degree of periodontal damage, as well as the sealing ability and biocompatibility of the repair material.[45] It has been recognized that treatment success depends mainly on immediate sealing of the perforation and appropriate infection control. Among the materials that are commonly used to seal root perforations are mineral trioxide aggregate (MTA), super ethoxybenzoic acid (EBA) cement, intermediate restorative material (IRM), glass ionomer cements, and composites. The topic of perforations is further discussed in Chapter 20.

Clinical classification of pulpal and periapical diseases

Many attempts have been made over the years to develop classifications of pulpal and periapical disease. However, studies have shown that making a correlation between clinical signs and symptoms and the histopathology of a given clinical condition is challenging.[122,123] Therefore *clinical* classifications have been developed in order to formulate treatment plan options. In the most general terms, the objective and subjective findings are used to classify the suspected pathosis, with the assigned designations merely representing the presence of healthy or diseased tissue.

The terminology and classifications that follow are based on those suggested by the American Association of Endodontists in 2016.[6]

Pulpal disease

Normal pulp

This is a clinical diagnostic category in which the pulp is symptom-free and normally responsive to pulp testing.[6] Teeth with normal pulp do not usually exhibit any spontaneous symptoms. The symptoms produced from pulp tests are mild, do not cause the patient distress, and result in a transient sensation that resolves in seconds. Radiographically, there may be varying degrees of pulpal calcification but no evidence of resorption, caries, or mechanical pulp exposure. No endodontic treatment is indicated for these teeth.

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Pulpitis

This is a clinical and histologic term denoting inflammation of the dental pulp, clinically described as reversible or irreversible and histologically described as acute, chronic, or hyperplastic.[6]

Reversible pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the inflammation should resolve and the pulp return to normal.[6] When the pulp within the tooth is irritated so that the stimulation is uncomfortable to the patient but reverses quickly after irritation, it is classified as *reversible pulpitis*. Causative factors include caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of the irritant will resolve the symptoms. Confusion can occur when there is exposed dentin, without evidence of pulp pathosis, which can sometimes respond with sharp, quickly reversible pain when subjected to thermal, evaporative, tactile, mechanical, osmotic, or chemical stimuli. This is known as *dentin* (or *dentinal*) *sensitivity* (or *hypersensitivity*). Exposed dentin in the cervical area of the tooth accounts for most of the cases diagnosed as dentin sensitivity.[103]

As described in Chapter 14, fluid movement within dentinal tubules can stimulate the odontoblasts and associated fast-conducting A-delta nerve fibers in the pulp, which in turn produce sharp, quickly reversible dental pain (Fig. 1.35). The more open these tubules are (e.g., from a newly exposed preparation, dentin decalcification, periodontal scaling, tooth-bleaching materials, or coronal tooth fractures), the more the tubule fluid will move and, subsequently, the more the tooth will display dentin sensitivity when stimulated. When making a diagnosis, it is important to discriminate this dentin sensitivity sensation from that of reversible pulpitis, which would be secondary to caries, trauma, or new or defective restorations. Detailed questioning about recent dental treatment and a thorough clinical and radiographic examination will help to separate dentin sensitivity from other pulpal pathosis, as the treatment modalities for each are completely different.[18]

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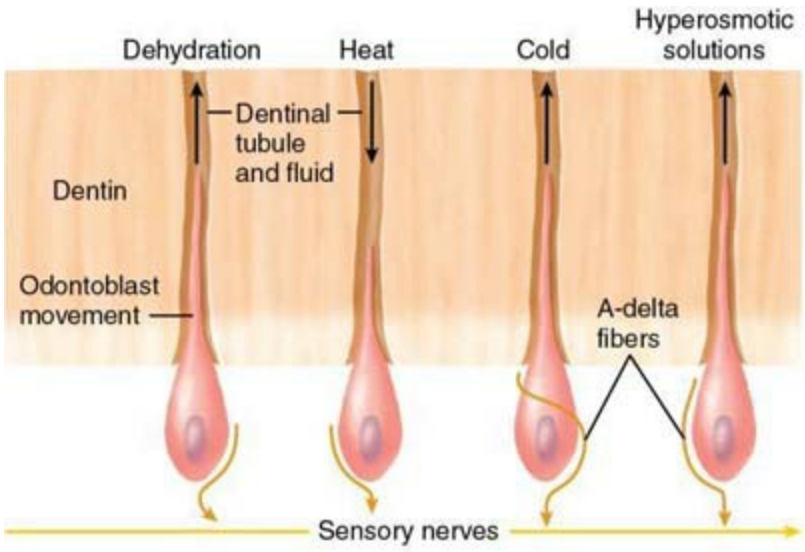

FIG. 1.35 Dentinal tubules are filled with fluid that, when stimulated, will cause sensation. Temperature changes, air, and osmotic changes can provoke the odontoblastic process to induce the stimulation of underlying A-delta fibers.

An illustration shows tooth stimulated by dehydration, heat, cold, and Hyper-osmotic solutions. Dehydration: An arrow, labeled dentinal tubule and fluid, points up toward the root and corresponding odontoblast movement leads to dental pain. Heat: Dentinal tubule and fluid go toward pulp at the bottom. In cold and hyper-osmotic solutions, dentinal tubule and fluid go up that stimulate A-delta fibers. A rightward arrow below all illustrations is labeled sensory nerves.

Irreversible pulpitis

As the disease state of the pulp progresses, the inflammatory condition of the pulp can change to *irreversible pulpitis.* At this stage, treatment to remove the diseased pulp will be necessary. This condition can be divided into the subcategories of *symptomatic* and *asymptomatic* irreversible pulpitis.

Symptomatic irreversible pulpitis.

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] Teeth that are classified as having *symptomatic irreversible pulpitis* exhibit intermittent or spontaneous pain. Rapid exposure to dramatic temperature changes (especially to cold stimuli) will elicit heightened and prolonged episodes of

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pain even after the thermal stimulus has been removed. The pain in these cases may be sharp or dull, localized, diffuse, or referred. Typically, there are minimal or no changes in the radiographic appearance of the periradicular bone. With advanced irreversible pulpitis, a thickening of the periodontal ligament may become apparent on the radiograph, and there may be some evidence of pulpal irritation by virtue of extensive pulp chamber or root canal space calcification. Deep restorations, caries, pulp exposure, or any other direct or indirect insult to the pulp, recently or historically, may be present. It may be seen radiographically or clinically or may be suggested from a complete dental history. Patients who present with symptomatic anterior teeth for which there are no obvious etiologic factors also should be questioned regarding past general anesthesia or endotracheal intubation procedures.[3,127,138] In addition, patients should be questioned about a history of orthodontic treatment. Typically, when symptomatic irreversible pulpitis remains untreated, the pulp will eventually become necrotic.[109,139]

Asymptomatic irreversible pulpitis.

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] However, the patient does not complain of any symptoms. On occasion, deep caries will not produce any symptoms, even though clinically or radiographically the caries may extend well into the pulp. Left untreated, the tooth may become symptomatic or the pulp will become necrotic. In cases of *asymptomatic irreversible pulpitis,* endodontic treatment should be performed as soon as possible so that symptomatic irreversible pulpitis or necrosis does not develop and cause the patient severe pain and distress.

Pulp necrosis

This is a clinical diagnostic category indicating death of the dental pulp. The pulp is usually nonresponsive to pulp testing.[6] When pulpal *necrosis* (or *nonvital pulp*) occurs, the pulpal blood supply is nonexistent and the pulpal nerves are nonfunctional. It is the only clinical classification that directly attempts to describe the histologic status of the pulp (or lack thereof). This condition is subsequent to symptomatic or asymptomatic irreversible pulpitis. After the pulp becomes completely necrotic, the tooth will typically become

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asymptomatic until such time when there is an extension of the disease process into the periradicular tissues. With pulp necrosis, the tooth will usually not respond to electric pulp tests or to cold stimulation. However, if heat is applied for an extended period of time, the tooth may respond to this stimulus. This response could possibly be related to remnants of fluid or gases in the pulp canal space expanding and extending into the periapical tissues.

Pulpal necrosis may be partial or complete and it may not involve all of the canals in a multirooted tooth. For this reason, the tooth may present with confusing symptoms. Pulp testing over one root may give no response, whereas over another root it may give a positive response. The tooth may also exhibit symptoms of symptomatic irreversible pulpitis. Pulp necrosis, in the absence of restorations, caries, or luxation injuries, is likely caused by a longitudinal fracture extending from the occlusal surface and into the pulp.[19]

After the pulp becomes necrotic, microbial growth can be sustained within the canal. When this infection (or its microbial byproducts) extends into the periodontal ligament space, the tooth may become symptomatic to percussion or exhibit spontaneous pain. Radiographic changes may occur, ranging from a thickening of the periodontal ligament space to the appearance of a periapical radiolucent lesion. The tooth may become hypersensitive to heat, even to the warmth of the oral cavity, and is often relieved by applications of cold. As previously discussed, this may be helpful in attempting to localize a necrotic tooth (i.e., by the application of cold one tooth at a time) when the pain is referred or not well localized.

Previously treated

This is a clinical diagnostic category indicating that the tooth has been endodontically treated and the canals are obturated with various filling materials other than intracanal medicaments.[6] In this situation, the tooth may or may not present with signs or symptoms but will require additional nonsurgical or surgical endodontic procedures to retain the tooth. In most such situations, there will no longer be any vital or necrotic pulp tissue present to respond to pulp testing procedures.

Previously initiated therapy

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This is a clinical diagnostic category indicating that the tooth has been previously treated by partial endodontic therapy (e.g., pulpotomy, pulpectomy).[6] In most instances, the partial endodontic therapy was performed as an emergency procedure for symptomatic or asymptomatic irreversible pulpitis cases. In other situations, these procedures may have been performed as part of vital pulp therapy procedures, traumatic tooth injuries, apexification, or apexogenesis therapy. At the time these cases present for root canal therapy it would not be possible to make an accurate pulpal diagnosis because all, or part, of the pulp tissue has already been removed.

Apical (periapical) disease

Normal apical tissues

This classification is the standard against which all of the other apical disease processes are compared. In this category the patient is asymptomatic and the tooth responds normally to percussion and palpation testing. The radiograph reveals an intact lamina dura and periodontal ligament space around all the root apices.

Periodontitis

This classification refers to an inflammation of the periodontium.[6] When located in the periapical tissues it is referred to as apical periodontitis. Apical periodontitis can be subclassified to symptomatic apical periodontitis and asymptomatic apical periodontitis.

Symptomatic apical periodontitis

This condition is defined as an inflammation, usually of the apical periodontium, producing clinical symptoms including a painful response to biting or percussion or palpation. It might or might not be associated with an apical radiolucent area.[6] This tooth may or may not respond to pulp sensibility tests, and the radiograph or image of the tooth will typically exhibit at least a widened periodontal ligament space and may or may not show an apical radiolucency associated with one or all of the roots.

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Asymptomatic apical periodontitis

This condition is defined as inflammation and destruction of apical periodontium that is of pulpal origin; it appears as an apical radiolucent area, and does not produce clinical symptoms.[6] This tooth does not usually respond to pulp sensibility tests, and the radiograph or image of the tooth will exhibit an apical radiolucency. The tooth is generally not sensitive to biting pressure but may "feel different" to the patient on percussion. Manifestation of persistent apical periodontitis may vary among patients.[89]

Acute apical abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *rapid onset*, spontaneous pain, tenderness of the tooth to pressure, pus formation, and swelling of associated tissues.[6] A tooth with an *acute apical abscess* will be acutely painful to biting pressure, percussion, and palpation. This tooth will not respond to any pulp sensibility tests and will exhibit varying degrees of mobility. The radiograph or image can exhibit anything from a widened periodontal ligament space to an apical radiolucency. Swelling will be present intraorally and the facial tissues adjacent to the tooth will almost always present with some degree of swelling. The patient will frequently be febrile, and the cervical and submandibular lymph nodes may exhibit tenderness to palpation.

Chronic apical abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *gradual onset*, little or no discomfort, and the intermittent discharge of pus through an associated sinus tract.[6] The sinus tract can appear intraorally or extraorally. In general, a tooth with a *chronic apical abscess* will not present with clinical symptoms. The tooth will not respond to pulp sensibility tests, and the radiograph or image will exhibit an apical radiolucency. Usually the tooth is not sensitive to biting pressure but can "feel different" to the patient on percussion. This entity is distinguished from asymptomatic apical periodontitis because it will exhibit intermittent drainage through an associated sinus tract.

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Referred pain

The perception of pain in one part of the body that is distant from the actual source of the pain is known as *referred pain*. Whereas pain of nonodontogenic origin can refer pain to the teeth, teeth may also refer pain to other teeth as well as to other anatomic areas of the head and neck. This may create a diagnostic challenge in that the patient may insist that the pain is from a certain tooth or even from an ear when, in fact, it is originating from a distant tooth with pulpal pathosis. Using electronic pulp testers, investigators found that patients could localize *which* tooth was being stimulated only 37.2% of the time and could narrow the location to three teeth only 79.5% of the time, illustrating that patients may have a difficult time discriminating the exact location of pulpal pain.[44]

Referred pain from a tooth is usually provoked by an intense stimulation of pulpal C fibers, the slow conducting nerves that, when stimulated, cause an intense, slow, dull pain. Anterior teeth seldom refer pain to other teeth or to opposite arches, whereas posterior teeth may refer pain to the opposite arch or to the periauricular area but seldom to the anterior teeth.[14] Mandibular posterior teeth tend to transmit referred pain to the periauricular area more often than maxillary posterior teeth. One study showed that when second molars were stimulated with an electric pulp tester, patients could discriminate accurately which arch the sensation was coming from only 85% of the time, compared with an accuracy level of 95% with first molars and 100% with anterior teeth.[136] The investigators also pointed out that when patients first feel the sensation of pain, they are more likely to accurately discriminate the origin of the pain. With higher levels of discomfort, patients have less ability to accurately determine the source of the pain. Therefore, in cases of diffuse or referred pain, the history of where the patient first felt the pain may be significant.

Because referred pain can complicate a dental diagnosis, the clinician must be sure to make an accurate diagnosis to protect the patient from unnecessary dental or medical treatment. After all the testing procedures are complete and if it is determined that the pain is not of odontogenic origin, then the patient should be referred to an orofacial pain clinic for further testing. For further information on pain of nonodontogenic origin, see Chapter 4.

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Summary

Endodontics is a multifaceted specialty, with much emphasis on how cases are clinically treated. Clinicians have increased their ability to more accurately perform endodontic procedures by way of increased visualization using the operating microscope, precise apical foramen detection using electronic apex locators, enhanced imaging techniques using digital radiography, three-dimensional imaging, and more. Practices have incorporated more refined canal cleaning and shaping techniques by using ultrasonics and rotary-driven nickel titanium files facilitated with computer-assisted electronic handpieces. Many other advancements also have been introduced with the objective of achieving an optimal result during endodontic treatment. However, these advancements are useless if an incorrect diagnosis is made. Before the clinician ever considers performing any endodontic treatment, the following questions must be answered:

- Is the existing problem of dental origin?
- Are the pulpal tissues within the tooth pathologically involved?
- Why is the pulpal pathosis present?
- What is the prognosis?
- What is the appropriate form of treatment?

Testing, questioning, and reasoning are combined to achieve an accurate diagnosis and to ultimately form an appropriate treatment plan. The art and science of making this diagnosis are the first steps that must be taken before initiating any endodontic treatment.

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References

1. Abella F, Patel S, Duran-Sindreu F, et al: *Evaluating the periapical status of teeth with irreversible pulpitis by using cone-beam computed tomography scanning and periapical radiographs*, J Endod 2012;38: 1588.
2. Abuabara A, Zielak JC, Schramm CA, et al: *Dental infection simulating skin lesion*, An Bras Dermatol 2012;87: 619.
3. Adolphs N, Kessler B, von Heymann C, et al: *Dentoalveolar injury related to general anaesthesia*, a 14 years review and a statement from the surgical point of view based on a retrospective analysis of the documentation of a university hospital Dent Traumatol 2011;27: 10.
4. Al-Hezaimi K, Naghshbandi J, Simon JH, et al: *Successful treatment of a radicular groove by intentional replantation and emdogain therapy*, four years follow-up Oral Surg Oral Med Oral Pathol Oral Radiol Endodon 2009;107: e82.
5. Alomari FA, Al-Habahbeh R, Alsakarna BK. *Responses of pulp sensibility tests during orthodontic treatment and retention*, Int Endod J 2011;44: 635.
6. American Association of Endodontists. *Glossary of endodontic terms*, ed 9 2016; American Association of Endodontists Chicago.
7. Anderson RW, Pantera EA. *Influence of a barrier technique on electric pulp testing*, J Endod 1988;14: 179.
8. Andreasen JD, Andreasen FM, Andersson L. *Textbook and color atlas of traumatic injuries to the teeth*, ed 5 2019; Wiley Blackwell Philadelphia.

Telegram: @dental_k

www.konkur.in

9. Andreasen JO, Ahrensburg SS, Tsillingaridis G. *Root fractures*, the influence of type of healing and location of fracture on tooth survival ratesan analysis of 492 cases Dent Traumatol 2012;28: 404.
10. Arakawa S, Cobb CM, Rapley JW, et al: *Treatment of root fracture by CO_2 and Nd:YAG lasers*, an in vitro study J Endod 1996;22: 662.
11. Augsburger RA, Peters DD. *In vitro effects of ice, skin refrigerant, and CO_2 snow on intrapulpal temperature*, J Endod 1981;7: 110.
12. Baumgartner JC, Picket AB, Muller JT. *Microscopic examination of oral sinus tracts and their associated periapical lesions*, J Endod 1984;10: 146.
13. Beltes C, Zachou E. *Endodontic management in a patient with vitamin D-resistant rickets*, J Endod 2012;38: 255.
14. Bender IB. *Pulpal pain diagnosis*, a review J Endod 2000;26: 175.
15. Bender IB, Landau MA, Fonsecca S, et al: *The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth*, J Am Dent Assoc 1989;118: 305.
16. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone. Part I*, J Am Dent Assoc 1961;62: 152.
17. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone. Part II*, J Am Dent Assoc 1961;62: 708.
18. Berman LH. *Dentinal sensation and hypersensitivity*, a review of mechanisms and treatment alternatives J Periodontol 1984;56: 216.
19. Berman LH, Kuttler S. *Fracture necrosis*, diagnosis, prognosis assessment, and treatment recommendations J

Telegram: @dental_k

www.konkur.in

Endod 2010;36: 442.
20. Bierma MK, McClanahan S, Baisden MK, et al: *Comparison of heat-testing methodology*, J Endod 2012;38: 1106.
21. Bornstein MM, Lauber R, Sendi P, et al: *Comparison of periapical radiography and limited cone-beam computed tomography in mandibular molars for analysis of anatomical landmarks before apical surgery*, J Endod 2011;37: 151.
22. Cameron CE. *The cracked tooth syndrome*, additional findings J Am Dent Assoc 1981;93: 971.
23. Chen E, Abbottt PV. *Evaluation of accuracy, reliability, and repeatability of five dental pulp tests*, J Endod 2011;37: 1619.
24. Chiego DJ, Cox CF, Avery JK. *H-3 HRP analysis of the nerve supply to primate teeth*, Dent Res 1980;59: 736.
25. Cleveland JL, Gooch BF, Shearer BG, et al: *Risk and prevention of hepatitis C virus infection*, J Am Dent Assoc 1999;130: 641.
26. Costa FF, Gaia BF, Umetsubo OS, et al: *Detection of horizontal root fracture with small-volume cone-beam computed tomography in the presence and absence of intracanal metallic post*, J Endod 2011;37: 1456.
27. Costa FF, Gaia BF, Umetsubo OS, et al: *Use of large-volume cone-beam computed tomography in identification and localization of horizontal root fracture in the presence and absence of intracanal metallic post*, J Endod 2012;38: 856.
28. Costa FWG, Rodrigues RR, Batista ACB. *Multiple radiopaque mandibular lesions in a patient with Apert syndrome*, J Endod 2012;38: 1639.
29. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic*

Telegram: @dental_k

www.konkur.in

applications of cone-beam volumetric tomography, J Endod 2007;33: 1121.

30. Dankner E, Harari D, Rotstein I. *Dens evaginatus of anterior teeth*, literature review and radiographic survey of 15,000 teeth Oral Surg Oral Med Oral Pathol 1996;81: 472.
31. Dastmalchi N, Jafarzadeh H, Moradi S. *Comparison of the efficacy of a custom-made pulse oximeter probe with digital electric pulp tester, cold spray, and rubber cup for assessing pulp vitality*, J Endod 2012;38: 1182.
32. Davido N, Rigolet A, Kerner S, et al: *Case of Ewing's sarcoma misdiagnosed as a periapical lesion of maxillary incisor*, J Endod 2011;37: 259.
33. Deepak BS, Subash TS, Narmatha VJ, et al: *Imaging techniques in endodontics*, an overview J Clin Imaging Sci 2012;2: 13.
34. DeRossi SS, Glick M. *Dental considerations for the patient with renal disease receiving hemodialysis*, J Am Dent Assoc 1996;127: 211.
35. DeRossi SS, Glick M. *Lupus erythematosus*, considerations for dentistry J Am Dent Assoc 1998;129: 330.
36. Dirks SJ, Paunovich ED, Terezhalmy GT, et al: *The patient with Parkinson's disease*, Quint Int 2003;34: 379.
37. Durack C, Patel S. *Cone beam computed tomography in endodontics*, Braz Dent J 2012;23: 179.
38. Edlund M, Nair MK, Nair UP. *Detection of vertical root fractures by using cone-beam computed tomography*, a clinical study J Endod 2011;37: 768.
39. Eikenerg S, Vandre R. *Comparison of digital dental x-ray systems with self-developing film and manual processing for endodontic file length determination*, J Endod 2000;26:

Telegram: @dental_k

www.konkur.in

65.
40. Evans D, Reid J, Strang R, et al: *A comparison of laser Doppler flowmetry with other methods of assessing the vitality of traumatized anterior teeth*, Endod Dent Traumatol 1999;15: 284.
41. Farman AG, Levato CM, Scarfe WC. *A primer on cone beam CT*, Inside Dentistry 2007;1: 90.
42. Filippatos CG, Tsatsoulis IN, Floratos S, et al: *A variability of electric pulp response threshold in premolars*, a clinical study J Endod 2012;38: 144.
43. Fouad AF. *Diabetes mellitus as a modulating factor of endodontic infections*, J Dent Educ 2003;67: 459.
44. Friend LA, Glenwright HD. *An experimental investigation into the localization of pain from the dental pulp*, Oral Surg Oral Med Oral Pathol 1968;25: 765.
45. Fuss Z, Trope M. *Root perforations*, classification and treatment choices based on prognostic factors Endod Dent Traumatol 1996;12: 255.
46. Fuss Z, Trowbridge H, Bender IB, et al: *Assessment of reliability of electrical and thermal pulp testing agents*, J Endod 1986;12: 301.
47. Ganz SD. *Cone beam computed tomography-assisted treatment planning concepts*, Dent Clin North Am 2011;55: 515.
48. Gillcrist JA. *Hepatitis viruses A, B, C, D, E and G*, implications for dental personnel J Am Dent Assoc 1999;130: 509.
49. Goldman M, Pearson A, Darzenta N. *Endodontic success*, who is reading the radiograph Oral Surg Oral Med Oral Pathol 1972;33: 432.
50. Goldman M, Pearson A, Darzenta N. *Reliability of radiographic interpretations*, Oral Surg Oral Med Oral

Telegram: @dental_k

www.konkur.in

Pathol 1974;38: 287.
51. Goodchild JH, Glick M. *A different approach to medical risk assessment*, Endod Topics 2003;4: 1.
52. Goon WW, Jacobsen PL. *Prodromal odontalgia and multiple devitalized teeth caused by a herpes zoster infection of the trigeminal nerve*, report of case J Am Dent Assoc 1988;116: 500.
53. Gopikrishna V, Tinagupta K, Kandaswamy D. *Comparison of electrical, thermal and pulse oximetry methods for assessing pulp vitality in recently traumatized teeth*, J Endod 2007;33: 531.
54. Gopikrishna V, Tinagupta K, Kandaswamy D. *Evaluation of efficacy of a new custom-made pulse oximeter dental probe in comparison with electrical and thermal tests for assessing pulp vitality*, J Endod 2007;33: 411.
55. Harrison JW, Larson WJ. *The epithelized oral sinus tract*, Oral Surg Oral Med Oral Pathol 1976;42: 511.
56. Heling I, Rotstein I. *A persistent oronasal sinus tract of endodontic origin*, J Endod 1989;15: 132.
57. Herman WW, Konzelman JL, Prisant LM. *New national guidelines on hypertension*, J Am Dent Assoc 2004;135: 576.
58. Idiyatullin D, Corum C, Moeller S, et al: *Dental magnetic resonance imaging*, making the invisible visible J Endod 2011;37: 745.
59. Imai K, Shimada Y, Sadr A, et al: *Nonivasive cross-sectional visualization of enamel cracks by optical coherence tomography*, in vitro, J Endod 2012;38: 1269.
60. Ingolfsson AER, Tronstad L, Riva CE. *Reliability of laser Doppler flowmetry in testing vitality of human teeth*, Endod Dent Traumatol 1994;10: 185.
61. Ingram TA, Peters DD. *Evaluation of the effects of carbon*

Telegram: @dental_k

www.konkur.in

dioxide used as a pulp test. Part 2, in vivo effect on canine enamel and pulpal tissues J Endod 1983;9: 296.

62. Jafarzadeh H, Abbott PV. *Review of pulp sensibility tests. Part I*, general information and thermal tests Int Endod J 2010;43: 738.
63. Jafarzadeh H, Abbott PV. *Review of pulp sensibility tests. Part II*, electric pulp tests and test cavities Int Endod J 2010;43: 945.
64. Johnson BR, Remeikis NA, Van Cura JE. *Diagnosis and treatment of cutaneous facial sinus tracts of dental origin*, J Am Dent Assoc 1999;130: 832.
65. Jones DM. *Effect of the type carrier used on the results of dichlorodifluoromethane application to teeth*, J Endod 1999;25: 692.
66. Jones VR, Rivera EM, Walton RE. *Comparison of carbon dioxide versus refrigerant spray to determine pulpal responsiveness*, J Endod 2002;28: 531.
67. Kaffe I, Gratt BM. *Variations in the radiographic interpretation of the periapical dental region*, J Endod 1988;14: 330.
68. Kahan RS, Gulabivala K, Snook M, et al: *Evaluation of a pulse oximeter and customized probe for pulp vitality testing*, J Endod 1996;22: 105.
69. Karayilmaz H, Kirzioglu Z. *Comparison of the reliability of laser Doppler flowmetry, pulse oximetry and electric pulp tester in assessing the pulp vitality of human teeth*, J Oral Rehabil 2011;38: 340.
70. Kataoka SH, Setzer FC, Gondim-Junior E, et al: *Pulp vitality in patients with intraoral and oropharyngeal malignant tumors undergoing radiation therapy assessed by pulse oximetry*, J Endod 2011;37: 1197.
71. Katz J, Chaushu G, Rotstein I. *Stafne's bone cavity in the*

Telegram: @dental_k

www.konkur.in

anterior mandible, a possible diagnosis challenge J Endod 2001;27: 304.

72. Kawai K, Masaka N. *Vertical root fracture treated by bonding fragments and rotational replantation*, Dent Traumatol 2002;18: 42.
73. Khocht A, Janal M, Harasty L, et al: *Comparison of direct digital and conventional intraoral radiographs in detecting alveolar bone loss*, J Am Dent Assoc 2003;134: 1468.
74. Koivisto T, Bowles WR, Rohrer M. *Frequency and distribution of radiolucent jaw lesions*, a retrospective analysis of 9,723 cases J Endod 2012;38: 729.
75. Kusgoz A, Yildirim T, Kayipmaz S, et al: *Nonsurgical endodontic treatment of type III dens invaginatus in maxillary canine*, an 18-month follow up Oral Surg Oral Med Oral Pathol Oral Radiol Endodon 2009;107: e103.
76. Lalla RV, D'Ambrosio JA. *Dental management considerations for the patient with diabetes mellitus*, J Am Dent Assoc 2001;132: 1425.
77. Laskin DM. *Anatomic considerations in diagnosis and treatment of odontogenic infections*, J Am Dent Assoc 1964;69: 308.
78. Liang YH, Li G, Wesselink PR, et al: *Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans*, J Endod 2011;37: 326.
79. Lin J, Chandler NP. *Electric pulp testing*, a review Int Endod J 2008;41: 365.
80. Little JW, Miller CS, Rhodus NL. *Dental management of the medically compromised patient*, ed 9 2018; Elsevier St. Louis.
81. Lofthag-Hansen S, Huumonen S, Gröndahl K, et al:

Telegram: @dental_k

www.konkur.in

Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 114.

82. Marder MZ. *The standard of care for oral diagnosis as it relates to oral cancer*, Compend Contin Educ Dent 1998;19: 569.
83. Mattson JS, Cerutis DR. *Diabetes mellitus*, a review of the literature and dental implications Comp Cont Educ Dent 2001;22: 757.
84. Mesaros S, Trope M, Maixner W, et al: *Comparison of two laser Doppler systems on the measurement of blood flow of premolar teeth under different pulpal conditions*, Int Endod J 1997;30: 167.
85. Metska ME, Aartman IHA, Wesselink PR, et al: *Detection of vertical root fractures in vivo in endodontically treated teeth by cone-beam computed tomography scans*, J Endod 2012;38: 1344.
86. Michaelson RE, Seidberg BH, Guttuso J. *An in vivo evaluation of interface media used with the electric pulp tester*, J Am Dent Assoc 1975;91: 118.
87. Miles DA, VanDis ML. *Advances in dental imaging*, Dent Clin North Am 1993;37: 531.
88. Miller CS, Little JW, Falace DA. *Supplemental corticosteroids for dental patients with adrenal insufficiency*, reconsideration of the problem J Am Dent Assoc 2001;132: 1570.
89. Morsani JM, Aminoshariae A, Han YW. *Genetic predisposition to persistent apical periodontitis*, J Endod 2011;37: 455.
90. Mozzo P, Proccacci A, Tacconi, et al: *A new volumetric CT machine for dental imaging based on the cone-beam technique*, preliminary results Eur Radiol 1998;8: 1558.

Telegram: @dental_k

www.konkur.in

91. Nair M, Pettigrew J, Mancuso A. *Intracranial aneurysm as an incidental finding*, Dentomaxillofac Radiol 2007;36: 107.
92. Nakata K, Naitob M, Izumi M, et al: *Effectiveness of dental computed tomography in diagnostic imaging of periradicular lesion of each root of a multirooted tooth*, a case report J Endod 2007;32: 583.
93. Nalliab RP, Allareddy V, Elangovan S, et al: *Hospital emergency department visits attributed to pulpal and periapical disease in the United States in 2006*, J Endod 2011;37: 6.
94. Özer SY. *Detection of vertical root fractures by using cone beam computed tomography with variable voxel sizes in an in vitro model*, J Endod 2011;37: 75.
95. Pantera EA, Anderson RW, Pantera CT. *Use of dental instruments for bridging during electric pulp testing*, J Endod 1992;18: 37.
96. Pantera EA, Anderson RW, Pantera CT. *Reliability of electric pulp testing after pulpal testing with dichlorodifluoromethane*, J Endod 1993;19: 312.
97. Paurazas SM, Geist JR, Pink FE. *Comparison of diagnostic accuracy of digital imaging using CCD and CMOS-APS sensors with E-speed film in the detection of periapical bony lesions*, Oral Surg Oral Med Oral Pathol Oral Radiology Endodon 2000;44: 249.
98. Peters DD, Baumgartner JC, Lorton L. *Adult pulpal diagnosis. 1. Evaluation of the positive and negative responses to cold and electric pulp tests*, J Endod 1994;20: 506.
99. Petersson K, Soderstrom C, Kiani-Anaraki M, et al: *Evaluation of the ability of thermal and electric tests to register pulp vitality*, Endod Dent Traumatol 1999;15: 127.

Telegram: @dental_k

www.konkur.in

100. Pinto A, Glick M. *Management of patients with thyroid disease*, oral health considerations J Am Dent Assoc 2002;133: 849.
101. Pitts DL, Natkin E. *Diagnosis and treatment of vertical root fractures*, J Endod 1983;9: 338.
102. Poeschl PW, Crepaz V, Russmueller G, et al: *Endodontic pathogens causing deep neck space infections*, clinical impact of different sampling techniques and antibiotic susceptibility J Endod 2011;37: 1201.
103. Rees JS, Addy M. *A cross-sectional study of dentine hypersensitivity*, J Clin Periodontol 2002;29: 997.
104. Rickoff B, Trowbridge H, Baker J, et al: *Effects of thermal vitality tests on human dental pulp*, J Endod 1988;14: 482.
105. Riley CK, Terezhalmy GT. *The patient with hypertension*, Quint Int 2001;32: 671.
106. Robb RA, Sinak LJ, Hoffman EA, et al: *Dynamic volume imaging of moving organs*, J Med Syst 1982;6: 539.
107. Rodrigues CD, Villar-Neto MJC, Sobral APV, et al: *Lymphangioma mimicking apical periodontitis*, J Endod 2011;37: 91.
108. Rosen H. *Cracked tooth syndrome*, J Prosthet Dent 1982;47: 36.
109. Rotstein I, Engel G. *Conservative management of a combined endodontic-orthodontic lesion*, Endod Dent Traumatol 1991;7: 266.
110. Rotstein I, Moshonov J, Cohenca N. *Endodontic therapy for a fused mandibular molar*, Endod Dent Traumatol 1997;13: 149.
111. Rotstein I. *Interaction between endodontics and periodontics*, Periodontol 2000 2017;74: 11.
112. Rotstein I, Ingle JI. *Endodontic-periodontal*

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www.konkur.in

interrelationship, Rotstein I Zabalegui B Al-Hezaimi K Ingle's endodontics, ed 7, Raleigh, North Carolina 2019; PMPH USA 1111.

113. Rotstein I, Stabholz A, Heling I, et al: *Clinical considerations in the treatment of dens invaginatus*, Endod Dent Traumatol 1987;3: 249.
114. Roykens H, Van Maele G, DeMoor R, et al: *Reliability of laser Doppler flowmetry in a 2-probe assessment of pulpal blood flow*, Oral Surg Oral Med Oral Pathol Oral Radiol Endodon 1999;87: 742.
115. Rud J, Omnell KA. *Root fractures due to corrosion*, diagnostic aspects Scand J Dent Res 1970;78: 397.
116. Rutsatz C, Baumhardt SG, Feldens CA, et al: *Response of pulp sensibility test is strongly influenced by periodontal attachment loss and gingival recession*, J Endod 2012;38: 580.
117. Sasano T, Nakajima I, Shohi N, et al: *Possible application of transmitted laser light for the assessment of human pulpal vitality*, Endod Dent Traumatol 1997;13: 88.
118. Schnettler JM, Wallace JA. *Pulse oximetry as a diagnostic tool of pulp vitality*, J Endod 1991;17: 488.
119. Schwartz RS. *Mineral trioxide aggregate*, a new material for endodontics J Am Dent Assoc 1999;130: 967.
120. Selden HS. *Repair of incomplete vertical root fractures in endodontically treated teeth*, in vivo trials J Endod 1996;22: 426.
121. Seltzer S, Bender IB, Nazimov H. *Differential diagnosis of pulp conditions*, Oral Surg Oral Med Oral Pathol 1965;19: 383.
122. Seltzer S, Bender IB, Ziontz M. *The dynamics of pulp inflammation*, correlations between diagnostic data and actual histologic findings in the pulp. Part I Oral Surg Oral

Telegram: @dental_k

www.konkur.in

Med Oral Pathol 1963;16: 846.
123. Seltzer S, Bender IB, Ziontz M. *The dynamics of pulp inflammation*, correlations between diagnostic data and actual histologic findings in the pulp. Part II Oral Surg Oral Med Oral Pathol 1963;16: 969.
124. Seo DG, Yi YA, Shin AJ, et al: *Analysis of factors associated with cracked teeth*, J Endod 2012;38: 288.
125. Setzer FC, Kataoka SH, Natrielli F, et al: *Clinical diagnosis of pulp inflammation based on pulp oxygenation rates measured by pulse oximetry*, J Endod 2012;38: 880.
126. Simon JHS, Dogan H, Ceresa LM, et al: *The radicular groove*, it's potential clinical significance J Endod 2000;26: 295.
127. Simon JHS, Lies J. *Silent trauma*, Endod Dent Traumatol 1999;15: 145.
128. Shemesh H, Cristescu RC, Wesselink PR, et al: *The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations*, J Endod 2011;37: 513.
129. Steinbacher DM, Glick M. *The dental patient with asthma*, an update and oral health considerations J Am Dent Assoc 2001;132: 1229.
130. Stroblitt H, Gojer G, Norer B, et al: *Assessing revascularization of avulsed permanent maxillary incisors by laser Doppler flowmetry*, J Am Dent Assoc 2003;134: 1597.
131. Suebnukarn S, Rhienmora P, Haddawy P. *The use of cone-beam computed tomography and virtual reality simulation for pre-surgical practice in endodontic microsurgery*, Int Endod J 2012;45: 627.
132. Sugaya T, Kawanami M, Noguchi H, et al: *Periodontal healing after bonding treatment of vertical root fracture*,

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www.konkur.in

Dent Traumatol 2001;17: 174.
133. Tatlidil R, Gözübüyük MM. *Mucinous adenocarcinoma of lung presenting as oral metastases*, a case report and literature review J Endod 37, 2011;110:.
134. Tewary S, Luzzo J, Hartwell G. *Endodontic radiography*, who is reading the digital radiograph J Endod 2011;37: 919.
135. Treister N, Glick M. *Rheumatoid arthritis*, a review and suggested dental care considerations J Am Dent Assoc 1999;130: 689.
136. Van Hassel HJ, Harrington GW. *Localization of pulpal sensation*, Oral Surg Oral Med Oral Pathol 1969;28: 753.
137. Velvart P, Hecker H, Tillinger G. *Detection of the apical lesion and the mandibular canal in conventional radiography and computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol Endodon 2001;92: 682.
138. Vogel J, Stubinger S, Kaufmann M. *Dental injuries resulting from tracheal intubation*, a retrospective study Dent Traumatol 2009;25: 73.
139. Von Böhl M, Ren Y, Fudalej PS, et al: *Pulpal reactions to orthodontic force application in humans*, a systematic review J Endod 2012;38: 1463.
140. Wallace JA, Schnettler JM. *Pulse oximetry as a diagnostic tool of pulpal vitality*, J Endod 1993;17: 488.
141. Weisleder R, Yamauchi S, Caplan DJ, et al: *The validity of pulp testing*, a clinical study J Am Dent Assoc 2009;140: 1013.
142. Zou X, Liu D, Yue L, et al: *The ability of cone-beam computerized tomography to detect vertical root fractures in endodontically treated and nonendodontically treated teeth*, a report of 3 cases Oral Surg Oral Med Oral Pathol Oral Radiol Endodon 2011;111: 797.

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2: Radiographic interpretation

Madhu K. Nair, Martin D. Levin, Umadevi P. Nair

CHAPTER OUTLINE

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Radiographic interpretation

Interpretation of information captured by radiographic imaging modalities is central to the diagnostic process. It is very important to capture a diagnostically useful image using appropriate exposure parameters and view it with interactive manipulation of brightness and contrast or window/level (for cone-beam computed tomography [CBCT] studies) in an optimal environment to adequately evaluate anatomy and diagnose pathoses. Accurate interpretation of root and canal morphology, determination of

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radiographic canal length, diagnosis of radicular and periradicular disease (Fig. 2.1), and postsurgical and long-term evaluation of the outcome of endodontic treatment are some of the routine diagnostic imaging tasks in endodontics.[218] Systematic and methodical interpretation processes must be followed for all images. Recognition of anatomy, anatomic variants, and pathologic conditions or deviations from normal is important. Various imaging modalities exist in radiology. Some use ionizing radiation, whereas others use ultrasonic waves (ultrasonography [US]) or powerful external magnetic fields (magnetic resonance imaging [MRI]). Interventional and noninterventional imaging modalities are also available. Imaging modalities using ionizing radiation are most frequently used in endodontic diagnoses. The different image capture modalities include digital receptors using different technologies.

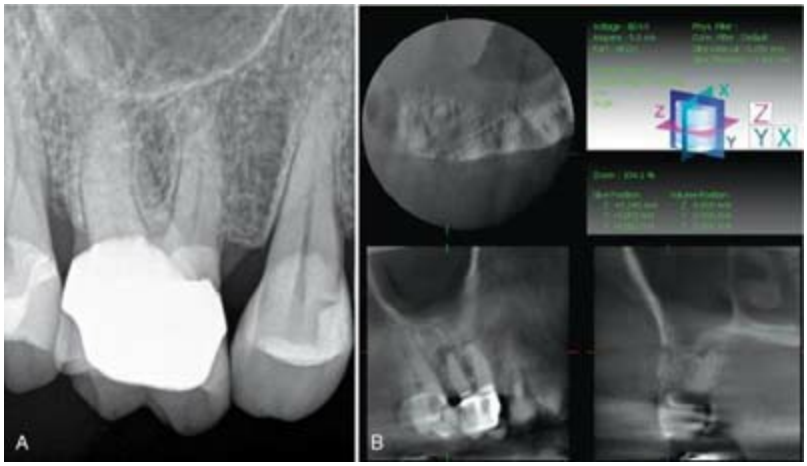

FIG. 2.1 **A,** A well-angulated periapical radiograph of the maxillary right first molar taken during a diagnostic appointment for endodontic evaluation of the maxillary right quadrant. At first glance, there is little radiographic evidence of significant or periradicular change. **B,** Contemporaneous CBCT image of same tooth gives an entirely different perspective; periapical changes are visible on all three roots in all three anatomic planes of section.

A) Radiograph shows right first molar with extremely radiopaque crown. Its adjacent tooth has wedge-shaped radiolucency.

B) Four panels show three radiographs depicting axial, coronal, and sagittal views of teeth, whereas the fourth panel shows a multitude of planes with X, Y, and Z planes. First radiograph: Teeth arranged horizontally with obscure view. Second radiograph: Three teeth with partially radiolucent region at the

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crown of right firsts molar. The tooth on the left shows fractured enamel, and there is significant space between adjacent teeth. Third radiograph: close-up view of tooth with inflammation at the root is shown.

Source: (*B* taken with J. Morita Veraviewepocs 3D [J. Morita, Osaka, Japan].)

Imaging modalities

Digital radiography using electronic sensors or photostimulable phosphor (PSP) plates is widely used in endodontics. Digital imaging modalities in endodontics use different image capture technologies, which include a charge-coupled device (CCD), a complementary metal oxide semiconductor (CMOS), or a PSP (also sometimes referred to as an indirect acquisition modality).

CCD-based solid-state sensors were used extensively in endodontics initially. However, the earlier generation sensors had a smaller active area and limited x-ray absorption and conversion efficiency, in addition to being bulky. Sensors use an array of radiation-sensitive elements that generate electric charges proportionate to the amount of incident radiation. To reduce the amount of radiation needed to capture an image, a light-sensitive array was developed that uses a scintillation layer laid on top of the CCD chip or added with a fiberoptic coupling. The generated charge is read out in a "bucket brigade" fashion and transferred to an analog-to-digital converter in the frame grabber assembly of the workstation. The digital information is processed, and an image is formed. CMOS-based sensors, on the other hand, have an active transistor at each element location. The area available for signal generation is relatively less, and there is a fixed pattern noise. These sensors are less expensive to manufacture and have been shown to be equally useful for specific diagnostic tasks.[114] Unlike the CCD, the CMOS chip requires very little electrical energy; therefore, no external power supply is needed to support USB utilization, and wireless applications are feasible. Wireless sensors are available (Fig. 2.2). However, radiofrequency interference may be a problem with these sensors. The current Wi-Fi sensor is less bulky and has a wire attached to it that enables transmission via 802.11 b/g standard. It uses a lithium-ion polymer battery that can last for approximately 100 exposures.

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FIG. 2.2 **A** and **B,** High-resolution complementary metal oxide semiconductor (CMOS) sensors are available from many manufacturers. Note that Figure In **B,** wireless CMOS sensors transmit images to the chairside workstation by 2.4 GHz radiofrequency.

A) Three digital dental sensors, named Schick 33, varying in size are placed on a white background.

B) Schick Wi-Fi wireless sensor on a white background.

Source: (Courtesy SIRONA DENTAL SYSTEMS, Long Island City, NY.)

Yet another type of sensor uses PSPs for image capture. PSP technology is also referred to as *computed radiography* (CR).[114,199] Unlike the CCD and CMOS sensors, PSP sensors are wireless. The phosphor is activated by a process called *doping*, which enables charges to be generated and stored when exposed to radiation. A latent image is stored in the sensor, and a PSP reader with a laser beam of specific wavelength is used to read out the image. Previously captured images can be erased by exposing the PSP sensor to white light. PSP plates can be damaged easily by scratching, but they are not as expensive as CCD or CMOS sensors. Incomplete erasure of the image can lead to ghost images when the plate is reused, and delayed processing can result in a decrease in image clarity.[6] PSP-based sensors are used in high-volume scenarios. Spatial resolution is lower with this type of sensor, but it has a wider dynamic range. These sensors can tolerate a wider range of exposures to produce a diagnostically useful image.

Radiation dose continues to be a concern with all imaging studies. The lowest possible dose must be delivered for each study. Most dental offices would not be in compliance with the latest recommendations of the National Council for Radiation Protection (NCRP) on reducing the radiation dose from

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intraoral radiographs (Box 2.1). Two terms have been specifically defined in the NCRP's report. The terms *shall* and *shall not* indicate that adherence to the recommendation would be in compliance with the standards of radiation safety. The terms *should* and *should not* indicate prudent practice and acknowledge that exceptions may be made in certain circumstances. In addition, the report establishes nine new recommendations for image processing of conventional film.

Box 2.1

Recommendations of the National Council on Radiation Protection

1. Dentists must examine their patients before ordering or prescribing x-ray images (this is not a new guideline).
2. The use of leaded aprons on patients shall not be required if all other recommendations in this report are rigorously followed (read full Report #145).
3. Thyroid shielding shall be used for children and should be provided for adults when it will not interfere with the examination (e.g., panoramic imaging).
4. Rectangular collimation of the beam, which has been recommended for years, shall be routinely used for periapical radiographs. Each dimension of the beam, measured in the plane of the image receptor, should not exceed the dimension of the image receptor by more than 2% of the source-to-image receptor distance. Similar collimation should be used, when feasible, for bitewing radiographs.
5. Image receptors of speeds slower than ANSI speed Group E films shall not be used for intraoral radiography. Faster receptors should be evaluated and adopted if found acceptable. For extraoral radiography, high-speed (400 or greater) rare earth screen-film systems or digital-imaging systems of equivalent or greater speed shall be used.
6. Dental radiographic films shall be developed according to the film manufacturer's instructions using the time-temperature method. In practical application, this means that sight development (reading wet x-ray films at the time of the procedure) shall not be used.

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7. Radiographic techniques for digital imaging shall be adjusted for the minimum patient dose required to produce a signal-to-noise ratio sufficient to provide image quality to meet the purpose of the examination.
8. Clinicians designing new offices or remodeling existing locations will need shield protection to be provided by a qualified expert.

Modified from the National Council on Radiation Protection and Measurements: Radiation protection in dentistry, Report #145, Bethesda, MD, 2003. Available at www.ncrppublications.org/Reports/145.

A strong argument can be made for clinicians to switch to a direct digital radiography (DDR) system to avoid all the drastic changes necessary to ensure compliance with the new recommendations. Even though restriction of an intraoral dental x-ray beam is mandated by federal law to a circle no greater than 7 cm, rectangular collimation has been proven to significantly reduce the radiation dose to the patient and is strongly recommended in the NCRP Report #145.

The American Dental Association (ADA) Council on Scientific Affairs has made the following statement:

> *Tissue area exposed to the primary x-ray beam should not exceed the minimum coverage consistent with meeting diagnostic requirements and clinical feasibility. For periapical (PA) and bitewing radiography, rectangular collimation should be used whenever possible because a round field beam used with a rectangular image receptor produces segments of the beam circle that are not used in receptor exposure, which causes unnecessary radiation exposure to the patient.*[4]

Image characteristics and processing

Spatial resolution achieved with current generation digital sensors is equally good or better than that of conventional intraoral radiographic film. Intraoral film has a resolution of 16-line pairs per millimeter (lp/mm) as measured

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using a resolution tool, and it increases to 20 to 24 lp/mm with magnification. *Spatial resolution* is defined as the ability to display two objects that are close to each other as two separate entities. *Contrast resolution* is defined as the ability to differentiate between areas on the image based on density. Most diagnostic tasks in endodontics require a high-contrast resolution.[142] However, image quality is not only a function of spatial resolution. The choice of appropriate exposure parameters, sensor properties, the image processing used, and viewing conditions and modalities directly affect diagnostic accuracy.

Postprocessing of images may be carried out to alter image characteristics. Radiographs need not be reexposed if image quality is not adequate. Diagnostic information can be teased out of the image if appropriate image processing is used. However, the original image must be acquired with optimal exposure parameters to accomplish meaningful image processing.[212] Suboptimally exposed images cannot be processed to yield diagnostic information, which may lead to a reduction in the diagnostic accuracy of the image. Image enhancement must be task specific. Signal-to-noise ratio (SNR) must be optimized to extract necessary information from the image. The bit-depth of images also has a direct relationship to image quality. It indicates the number of shades of gray that the sensor can capture for display. For example, an 8-bit image can depict 256 shades of gray. Most sensors are 12 or 14 bits in depth, capturing 4096 or 65,536 shades of gray, respectively. If the sensor captures several thousand shades of gray, the image can be manipulated through enhancement techniques to display those shades of gray that best depict the anatomy of interest. The human visual system is limited in the number of shades of gray that can be read at any point in time. Therefore, image enhancement is a must for all images, so as to delineate signals of interest through manipulation of the grayscale. Most endodontic tasks require a high contrast and thus a shorter grayscale.

Digital radiographs can be saved in different file formats. Several file formats are available: DICOM (*D*igital *I*maging and *C*ommunications in *M*edicine); tiff (*t*agged *i*mage *f*ile *f*ormat); jpeg (*j*oint *p*hotographic *e*xperts' *g*roup); gif (*g*raphics *i*nterchange *f*ormat); BMP (Windows' bitmap image file); PNG (*p*ortable *n*etwork *g*raphics); and so on. There also are several proprietary formats. "Lossy" and "lossless" compression schemes can be

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used for saving images, although lossless compression is preferred.[72]

Digital imaging and communications in medicine

DICOM is a set of international standards established in 1985 by the American College of Radiology (ACR) and the National Electrical Manufacturers Association (NEMA)[60,205] to address the issue of vendor-independent data formats and data transfers for digital medical images. The ADA has promoted the interoperability of dental images through the efforts of its Working Group 12.1.26. DICOM serves as a standard for the transferal of radiologic images and other medical information between computers, allowing digital communication between systems from various manufacturers and across different platforms (e.g., Apple iOS or Microsoft Windows).[60] The DICOM standard provides for several hundred attribute fields in the record header, which contains information about the image (e.g., pixel density, dimensions, and number of bits per pixel), in addition to relevant patient data and medical information. Although earlier versions did not specify the exact order and definition of the header fields, each vendor is required to publish a DICOM conformance statement, which gives the location of pertinent data. The big hurdle is to support medical and dental consultations between two or more locations with different imaging software.[60] With DICOM in place, dental clinicians can change vendors and maintain database interoperability. Most software vendors are striving to achieve full DICOM compliance, and some have achieved at least partial compliance. However, proprietary DICOM images are still produced in different systems, with the capability to export in universal DICOM format as needed. Diagnostic images are best saved as DICOM files to preserve image fidelity or as tiff files with no compression. Diagnosis suffers when images undergo lossy compression.[64,123,217]

Based on the DICOM model, the ADA Standards Committee on Dental Informatics has identified four basic goals for electronic standards in dentistry: (1) interoperability, (2) electronic health record design, (3) clinical workstation architecture, and (4) electronic dissemination of dental information.[10] The dental profession must continue to promote DICOM compatibility so that proprietary software and file types do not hinder communication and risk making data obsolete.

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Diagnostic tasks in endodontics

It is important to analyze the type of sensor used, software, processing, video card and monitor, and viewing conditions to determine whether the sensor is good for a specific diagnostic task. Calibration improves diagnostic accuracy.[128] Likewise, the use of optimal processing parameters improves image quality to the extent of making a significant difference in the diagnostic outcome. For instance, density plot analysis was shown to help with endodontic file measurements.[172] The major advantage of direct digital radiography (CCD, CMOS) is that the dose is significantly less compared with that required for film. The use of DDR, therefore, is justified when its performance is comparable to that of film with no statistically significant differences.[120]

The three types of measurement generally available with digital imaging software are (1) linear measurement, the distance between two points in millimeters (Fig. 2.3); (2) angle measurement, the angle between two lines; and (3) area measurement, the area of the image or a segment of the image. Because magnification and distortion errors play a significant role in the accuracy of two-dimensional (2D) radiographic measurement, both film and digital systems are subject to parallax error. However, a study that compared endodontic file length images of human teeth taken with a custom jig suggested that “measurement error was significantly less for the digital images than the film-based images.”[63] This was true even though, as the authors pointed out, the measurement differences may not have been clinically significant. Sophisticated calibration algorithms are under development, and accurate measurement of parallel images should be more feasible in the future.[41]

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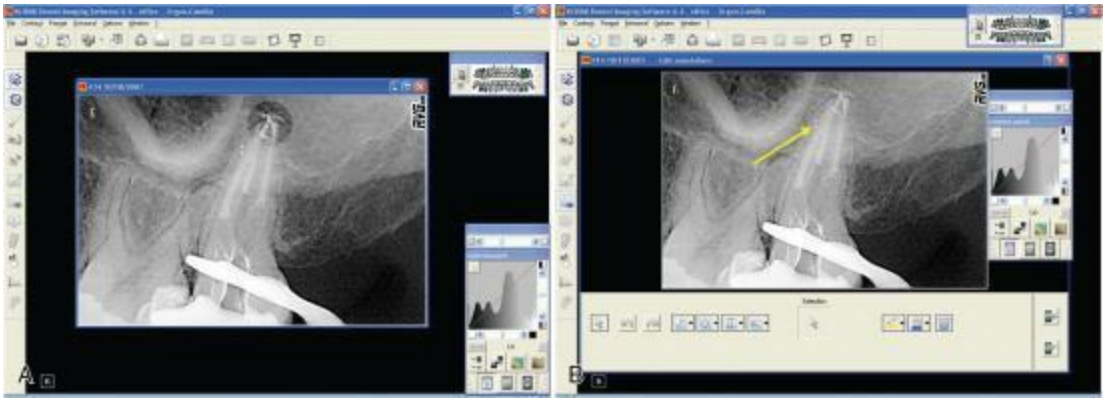

FIG. 2.3 **A,** Certain regions of interest (ROI) can be highlighted with a preset contrast tool that can be moved around the image. **B,** Preprogramed filters that enhance sharpness and contrast can be selected to optimize the image acquired.

A) Radiograph on computer screen with corresponding graph on right is shown. The radiograph has a circular patch with extremely less density at the root of tooth. The root of this tooth is longer than that of adjacent tooth.

B) Radiograph on computer screen with corresponding graph on right is shown. An arrow points toward the root of tooth. The root is denser comparatively to A radiograph.

Source: (Courtesy Carestream Dental LLC. Atlanta, GA.)

Diagnosis and healing

Image enhancement of direct and indirect digital radiographs based on the diagnostic task at hand has been shown to increase diagnostic accuracy compared to film-based images, which cannot be enhanced.[6,226] Posttreatment endodontic evaluation of healing of apical radiolucent areas is a challenge. Early changes indicating healing and bone fill are difficult to detect on conventional or digital radiographs. However, bone fill can be detected using more sensitive techniques, such as digital subtraction radiography (DSR), in which two images, separated in time but acquired with the exact same projection geometry and technique factors, can be subtracted from one another to tease out subtle changes in the periodontium and surrounding bone. Subtraction techniques are difficult to carry out in routine clinical practice because they are technique sensitive and can yield incorrect information if not performed accurately. Several studies have shown the usefulness of subtraction radiography using digital sensors.[138,150,231]

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Three-dimensional imaging

Computed tomography (CT) was introduced by Sir Godfrey Hounsfield in the 1970s. *Tomography* refers to "slice imaging," in which thin slices of the anatomy of interest are captured and synthesized manually or using an algorithm. CT makes use of automated reconstruction. Medical-grade CT used a translate-rotate image acquisition scheme as the technology developed, but the modality always resulted in higher radiation dose delivery because of redundancy of data capture, in addition to longer scan times with the potential for motion artifact. Multiple detectors and x-ray sources were used in later generations of CT units to reduce scanning times. The increased radiation dose, artifacts from metallic restorations, cost of scanning, long acquisition times, and lack of adequate dental-specific software have been drawbacks limiting the use of the technology in dentistry until recently. The advent of cone-beam volumetric computed tomography (CBVCT) introduced a faster, low-dose, low-cost, high-contrast imaging modality that could capture information in three dimensions using a limited field of view (FOV).

CBVCT, or cone beam CT (CBCT), is a relatively new diagnostic imaging modality that has been recently added to the endodontic imaging armamentarium. This modality uses a cone beam instead of a fan-shaped beam in multidetector computed tomography (MDCT), acquiring images of the entire volume as it rotates around the anatomy of interest. Compared with MDCT images, CBCT offers relatively high-resolution, isotropic images, allowing effective evaluation of root canal morphology and other subtle changes within the root canal system. Even though the resolution is not as high as that of conventional radiographs (18 microns), the availability of three-dimensional (3D) information, the relatively higher resolution, and a significantly lower dose compared to MDCT make CBCT the imaging modality of choice in challenging situations demanding localization and characterization of root canals.

The adoption of advanced imaging modalities such as CBCT for select diagnostic tasks is becoming popular with clinicians performing endodontic procedures. Two-dimensional grayscale images, whether conventional film-based or digital, cannot accurately depict the full 3D representation of the teeth and supporting structures. In fact, traditional images are poor representations of even the pulpal anatomy. They grossly underestimate canal

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structure and often cannot accurately visualize PA changes, especially where there is thick cortical bone, as in the presence of anatomic obstructions (Fig. 2.4, *A*). CBCT, however, allows the clinician to view the tooth and pulpal structures in thin slices in all three anatomic planes: axial, sagittal, and coronal. This capability alone allows visualization of PA pathoses and root morphology that was previously impossible to assess (see Fig. 2.4, *B* and *C*). Several tools available in CBCT, such as the ability to change the vertical or horizontal angulation of the image in real time, in addition to thin-slice, grayscale data of varying thicknesses, will never be available for conventional or even digital radiography. Furthermore, the use of CBCT data to view the region of interest in three anatomic planes of section at very low x-ray doses has never been as easy or accessible as it is today.

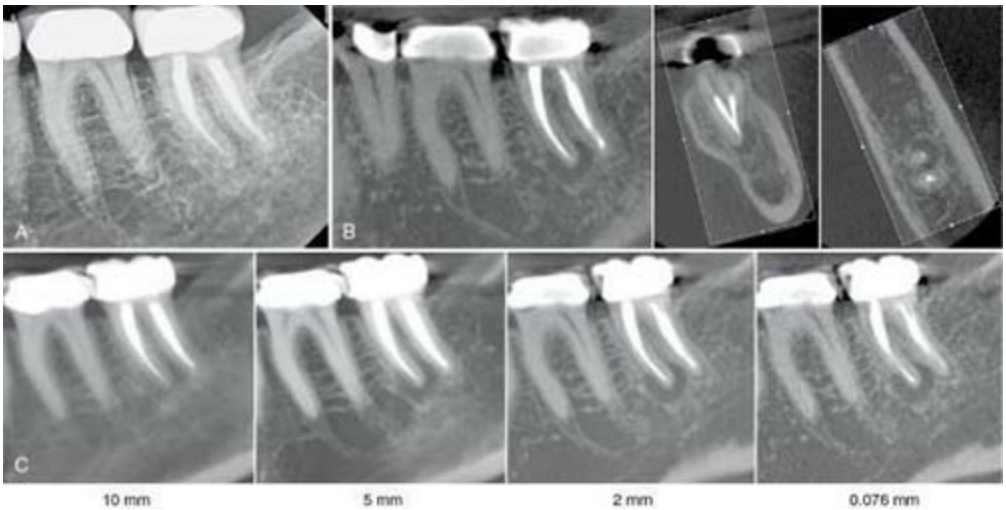

FIG. 2.4 This case demonstrates the difficulty in assessing lesions in the mandibular posterior region when there is a dense cortex. **A,** This well-angulated periapical radiograph shows a normal periapical region associated with the mandibular left second molar, for which the patient has presented for evaluation and possible retreatment. **B,** The CBCT corrected sagittal, coronal, and axial reconstructed multiplanar views, (*left to right,* sagittal, coronal, and axial views) show previous endodontic treatment, with a 6 mm diameter radiolucency with a well-defined, mildly corticated border, centered over a point on the buccal aspect of the root, 2 mm coronal to the apex; these are features consistent with an apical rarefying osteitis. **C,** The ray sum images of the sagittal view, where the image is "thinned" by decreasing the number of adjacent voxels using postprocessing software, simulates a curvilinear projection, showing diminishing superimposition (*left to right,* image layer of 10 mm, 5 mm, 2 mm, and 0.076 mm).

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Set of three radiographs marked A through C compares findings of lesion in mandibular posterior region through an angulated periapical radiograph and a corrected CBCT response to the treatment.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Microcomputed tomography (micro-CT) has also been evaluated in endodontic imaging.[103,169,170] Comparison of the effects of biomechanical preparation on the canal volume of reconstructed root canals in extracted teeth using micro-CT data was shown to assist with characterization of morphologic changes associated with these techniques.[170] Peters et al.[169] used micro-CT to evaluate the relative performance of nickel-titanium (Ni-Ti) instruments after the shaping of root canals of varying preoperative canal geometry. A study to examine the potential and accuracy of micro-CT for imaging of filled root canals showed it to be a highly accurate and nondestructive method for the evaluation of root canal fillings and its constituents. The qualitative and quantitative correlations between histologic and micro-CT examination of root canal fillings were high.[103] However, it is important to note that micro-CT remains a research tool and cannot be used for human imaging in vivo.

This chapter discusses the principles, applications, imaging attributes, image artifacts, and potential liability of adopting CBCT technology for endodontic procedures. Given this information, the student of endodontics will begin to realize the significant advantages, limitations, and diagnostic and treatment planning capabilities of this radiographic imaging modality.

Principles of cone beam computed tomography

Two important parameters of cone beam imaging are described in the following sections:

- Voxel size
- Field of view (FOV)

Voxels and voxel sizes

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Voxels are cuboidal elements that constitute a 3D volume, unlike pixels, which are 2D. Data are acquired and represented in three dimensions using voxels. Unlike with MDCT, typically used in medicine, cone beam units acquire x-ray information using low kV and low mA exposure parameters in a single pass from 180 to 360 degrees of rotation around the anatomy of interest. Medical scanners use higher voltages of 120 kV or more and a current of about 400 mA. Several units used in maxillofacial imaging use significantly lower exposure parameters (Figs. 2.5 to 2.7). The x-ray dose for all cone beam units is significantly lower than the dose received from a MDCT unit. Image attributes are also different in that volumes are reconstructed from isotropic voxels; that is, the images are constructed from volumetric detector elements that are cubical in nature and have the same dimensions of length, width, and depth. These voxel sizes can be as small as 0.076 to 0.6 mm.[139] By comparison, MDCT slice data are 0.5 mm to 1 cm thick. Fig. 2.8 illustrates the difference between a pixel and a voxel, the difference between an anisotropic pixel of MDCT and an isotropic pixel (voxel) of CBCT, and how the pixel data are acquired from both modalities.

FIG. 2.5 i-CAT unit.

Front view of a dental scanner, i-CAT unit that with a chair kept in a three-

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walled unit.

Source: (Courtesy Imaging Sciences International, Hatfield, PA.)

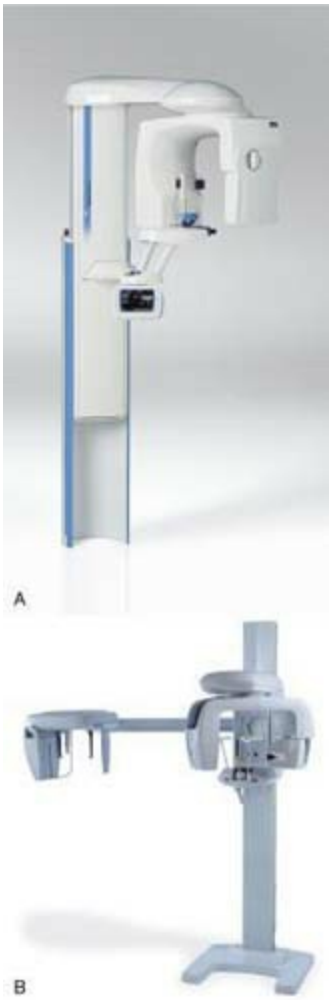

FIG. 2.6 **A,** Planmeca ProMax 3D. **B,** J. Morita Veraviewepocs 3D.

Set of two photographs marked A and B depict two types of 3 dimensional dental imaging devices.

Source: (*A,* Courtesy Planmeca Oy, Helsinki, Finland. *B,* Courtesy, JMorita USA, Irvine, CA).

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FIG. 2.7 **A,** CS 9000 3D and CS 8100 extraoral imaging systems. **B,** Morita Accu-i-Tomo 170.

Set of two photographs marked A and B depict two types of extraoral imaging systems.

Source: (A, Courtesy, Carestream Dental LLC, Atlanta, GA;. *B,* Courtesy J Morita USA, Irvine, CA).

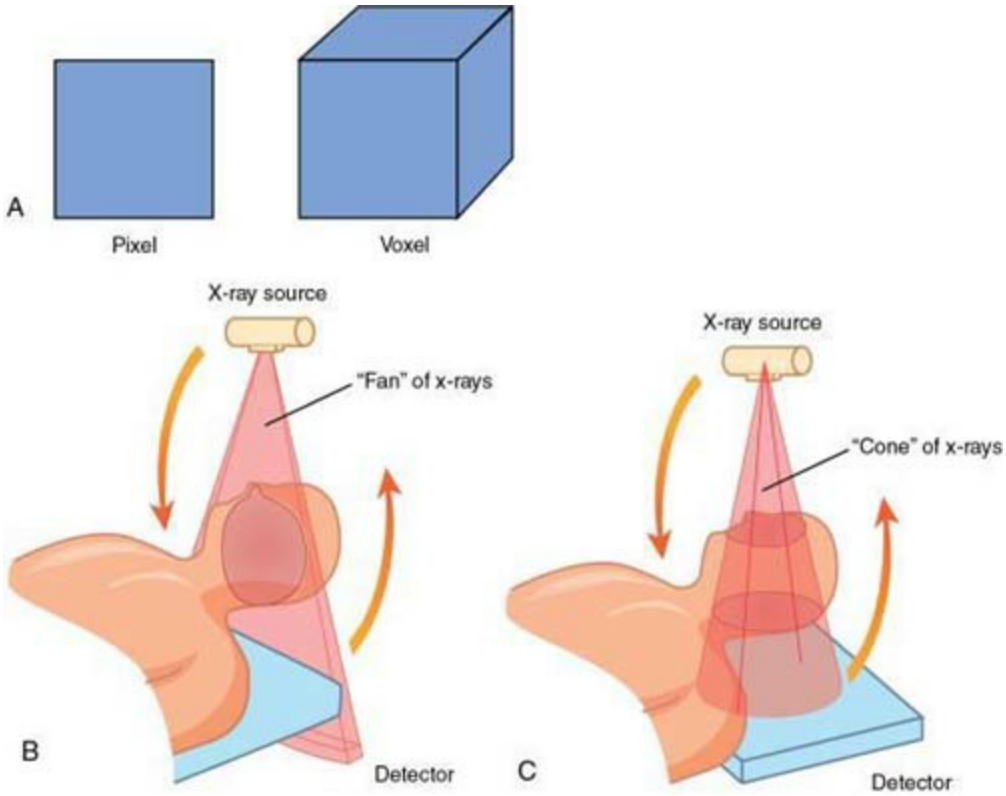

FIG. 2.8 **A,** *Left,* Drawing represents a *pixel* (picture element), the image capture and display element of any traditional digital image displayed on the computer. Shades of gray or color are displayed in these pixels to represent a 2D image. *Right,* Drawing represents a *voxel* (volume element). Voxels in CBCT are isometric and have the same dimension or length on all sides. They are very small (from 0.076 to 0.6 millimeters) and are the capture elements for cone beam imaging

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devices. Principles of conventional fan beam and cone beam computed tomography are presented in **B** and **C**, respectively.

A) A square represents pixel and a cuboid represents voxel.

B) Beam emitting from X-ray source is labeled “Fan” of x-rays.

C) Beam emitting from X-ray source is labeled “Cone” of x-rays.

Source: (**B** and **C,** From Babbush CA: *Dental implants: the art and science,* ed 2, St Louis, 2011, Elsevier/Saunders.)

The patient is positioned on a gantry in an MDCT unit, and images are acquired multiple slices at a time, which prolongs the acquisition time. The number of slices acquired is a direct function of the sensor array configuration. Spiral CT uses continuous translator motion of the gantry as images are acquired, thus shortening the acquisition time. This results in significantly higher absorbed x-ray doses for the patient. A typical CBCT examination would expose the patient to only about 20 to 500 μSv in a single study, whereas a typical medical examination of the head would approach 2100 μSv because the image data are gathered one section at a time. Therefore, soft-tissue imaging is better with MDCT because the signal intensity is higher. However, this is not a requirement for dental diagnostic tasks because hard tissue visualization is more important. Consequently, CBCT data have a much higher resolution than MDCT data for hard tissue visualization because of the smaller voxel sizes that medical-grade scanners are incapable of achieving at a significantly lower dose. Increased noise is observed as a result of volumetric acquisition, but the SNR is maintained at a desirable level that facilitates adequate diagnosis based on hard tissue signals.

Field of view

The FOV (Figs. 2.9 and 2.10) ranges from as small as a portion of a dental arch to an area as large as the entire head. The selection of the FOV depends on several factors. Among the most important are the following:

- Diagnostic task
- Type of patient
- Spatial resolution requirements

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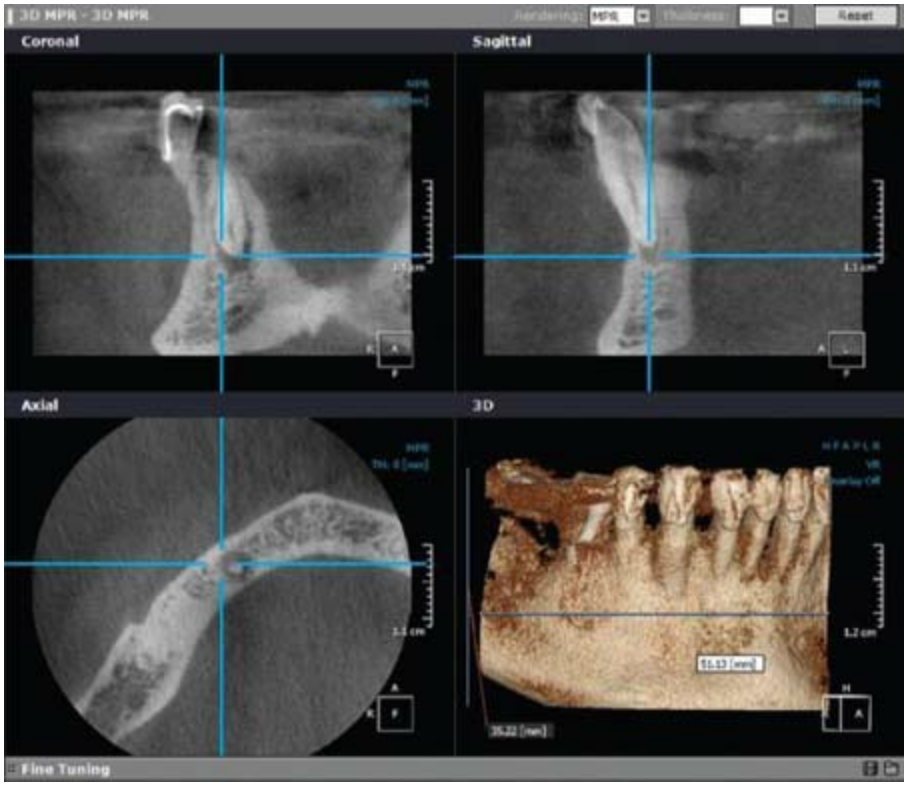

FIG. 2.9 Multiplanar and 3D color reconstructed views of the mandibular quadrant taken on a CBCT machine with a volume size of 37 × 50 mm.

Three radiographs show coronal, sagittal, and axial views of a lower quadrant of the teeth. At the bottom right corner, 3D image of the quadrant is shown.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

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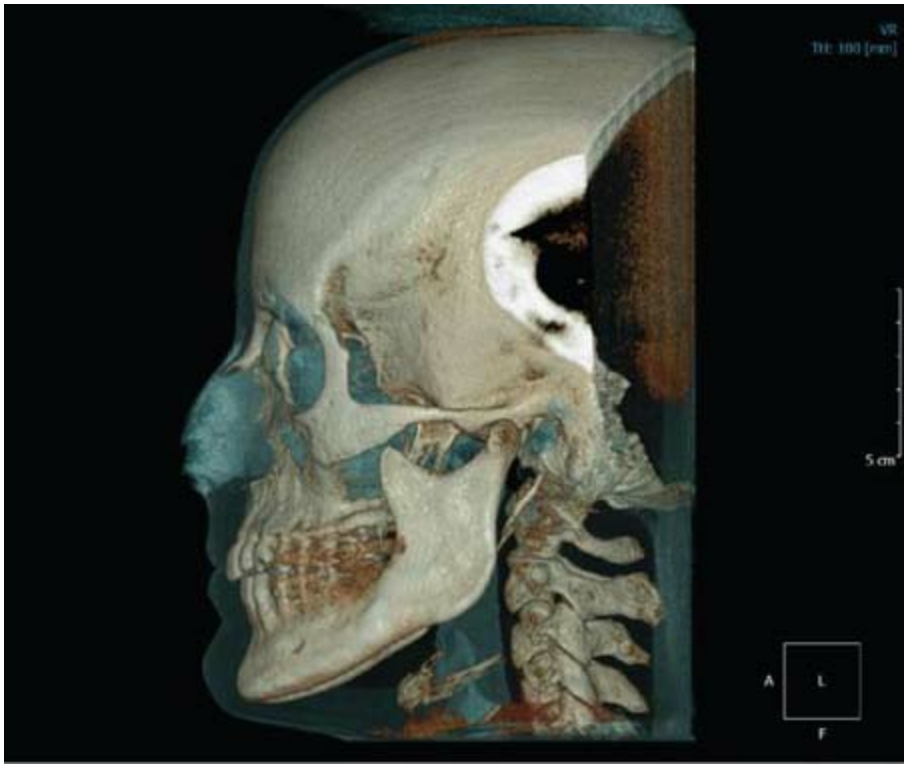

FIG. 2.10 Image of entire head (17 × 23 cm) from a large FOV unit.

Lateral view of human skull.

Source: (Image acquired with iCAT unit [Imaging Sciences International, Hatfield, PA].)

Diagnostic task

The diagnostic task is the single most important determinant of the FOV in any imaging study. Based on the outcome of the clinical assessment, history, and evaluation of previous and other available imaging studies, a segment of the jaw or a larger area may need to be imaged using an appropriate FOV. If systemic conditions or generalized disorders are suspected, a larger FOV is sometimes required. For most endodontic purposes, a limited FOV can be used if no signs or symptoms of systemic conditions are reported or noted. Under no circumstances should a screening study be done using a large FOV in the absence of signs and symptoms justifying the procedure. Several multifunctional cone beam units are available that allow the clinician to acquire several image types. Image quality has a direct impact on the diagnostic outcome; therefore, the choice of a FOV should be made carefully.

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Fig. 2.11 illustrates the advantages of using multiple image types for an endodontic case.

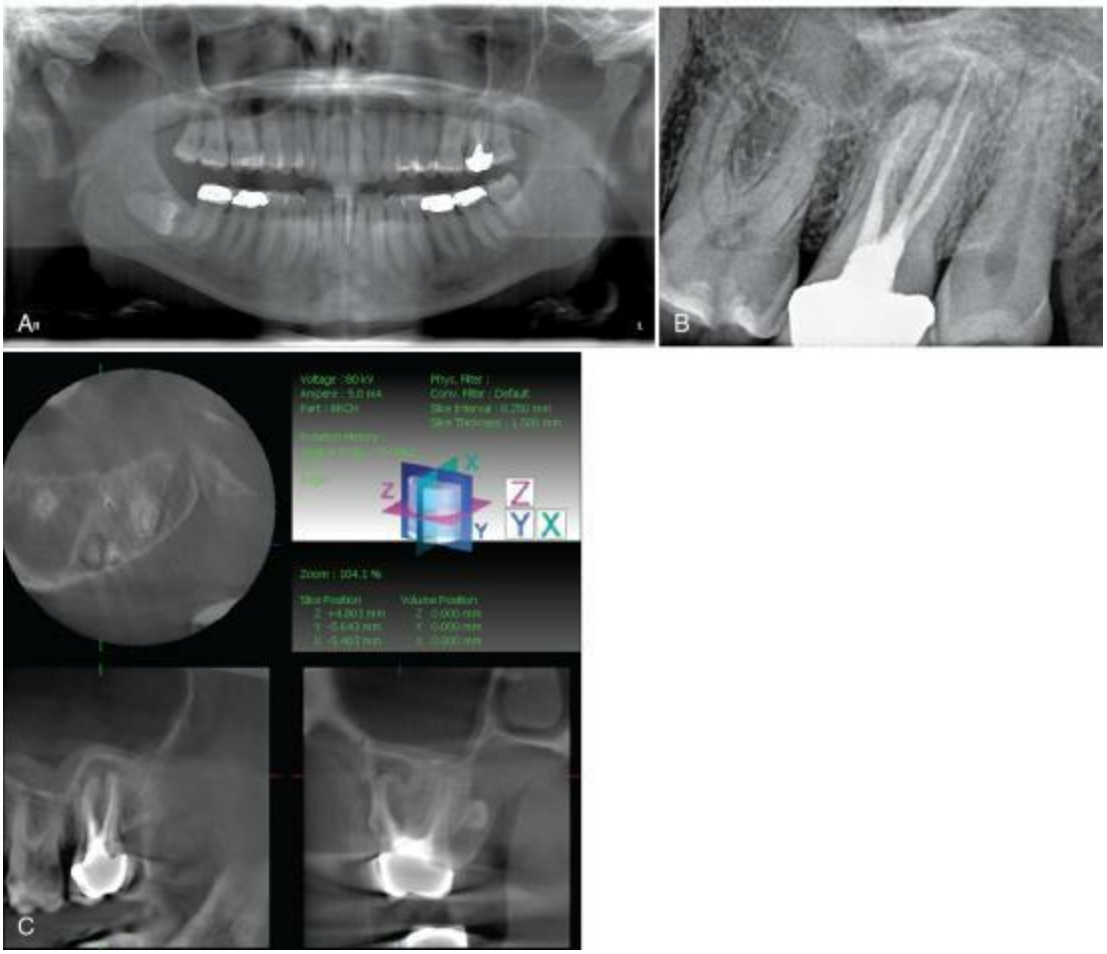

FIG. 2.11 **A,** Panoramic 2D image exposed with the JMorita Veraviewepocs 3D for evaluation of the mandibular left central incisor. Other radiographic findings also were revealed, including the horizontal bony impaction of the mandibular right third molar and a possible lesion of endodontic origin associated with the endodontically treated maxillary left second molar. Data available from the scan allowed a 3D reconstruction to be made for areas of concern. **B,** Periapical radiograph of the maxillary left second molar on the same patient revealed a periapical area of low attenuation in the region of the periapex of the mesiobuccal root. In this case, also, changes could be evaluated in greater detail with CBCT imaging. **C,** CBCT of the maxillary left second molar revealed detailed periapical and periradicular changes in all three orthogonal planes of the section, specifically illustrating the lesion of endodontic origin associated with the mesiobuccal root. Examination of the width of the mesiobuccal root in both the axial and coronal views (buccolingually) showed that the

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mesiobuccal root possibly had two canals and that only a single canal was treated during the initial endodontic therapy.

A) Radiograph shows front view of the open mouth. Crowns of some premolars and molars are extremely dense.

B) CBCT shows a central tooth with radiopaque crown and three root canals.

C) Four panels show three radiographs depicting axial, coronal, and sagittal views of teeth, whereas the fourth panel shows a multitude of planes with X, Y, and Z planes. The first radiograph less dense tissue around the right tooth. The second radiograph shows two teeth. One tooth on the right has two root canals. The central part of crown is less dense. The third radiograph shows close-up of tooth with two root canals.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Additional benefits of CBCT imaging software include allowing the clinician to format the volume to generate an image that looks like a panoramic radiograph. Conventional panoramic machines, although not commonly used by endodontists, use the focal trough, or zone of sharpness, to position patients so as to minimize distortion along multiple axes. All inherent problems associated with panoramic imaging, including distortion, magnification, blurring, ghost shadows, and other artifacts, can be expected on the resulting image if patient positioning is not accurate. With CBCT, such artifacts are not generated, resulting in a distortion-free panoramic reconstruction (Fig. 2.12). However, it must be noted that CBCTs should not be generated in patients requiring a panoramic radiograph alone because of dose concerns.

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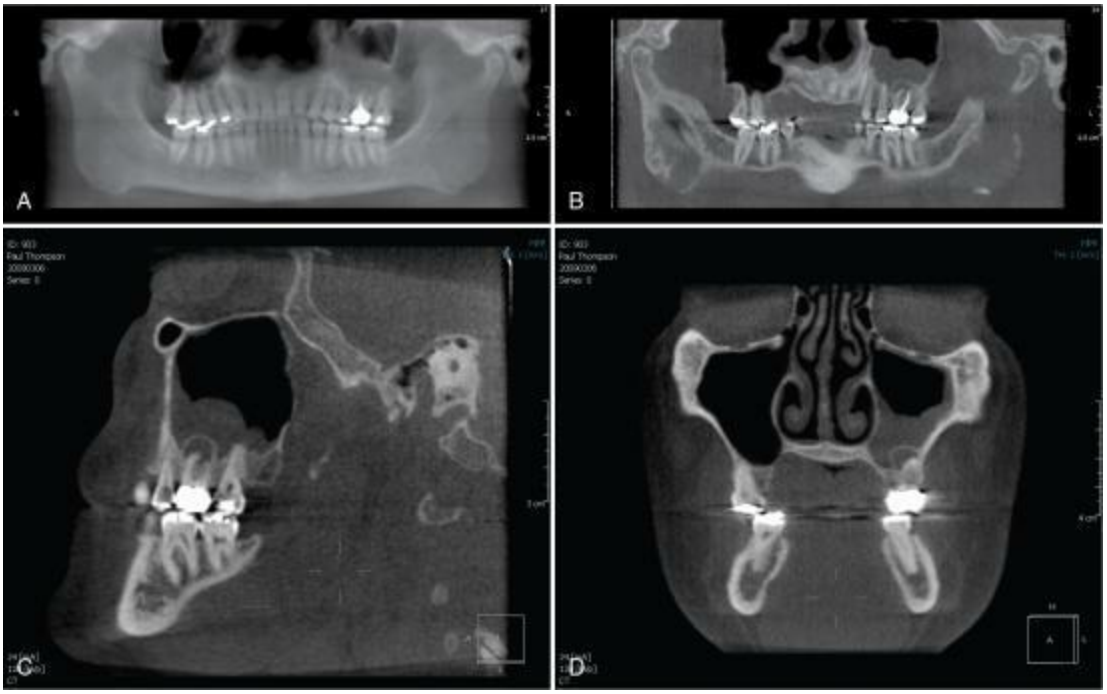

FIG. 2.12 **A,** This reconstructed panoramic image from CBCT data approximates the view one would see with a conventional panoramic radiograph. It is somewhat difficult to see the lesion on the maxillary left first molar. **B,** This thin-slice pseudopanoramic image (0.01 mm) shows the lesion without most of the anatomic superimposition. These slices of the maxillary left first molar show the lesion in sagittal **(C)** and coronal **(D)** views, confirming the features seen in the pseudopanoramic view.

A) Radiograph shows front view of teeth.

B) Front view of teeth with porous bone at the center and dark patches at the top.

C) Lateral view of the jaw shows a large dark cavity above the teeth and inflamed tissue around the lower teeth.

D) Panoramic view shows two teeth with large holes above them.

Newer hybrid units, such as the CS 9300 3D Extraoral Imaging System (Carestream Dental, Atlanta, GA), have a wide range of FOV choices for a variety of diagnostic tasks, in addition to a conventional panoramic imaging option (Fig. 2.13). The CS 9000 unit offers the lowest voxel size of 76 microns, whereas the CS 9300 can resolve down to 90 microns, with a range extending to 500 microns for larger FOV studies. Likewise, the Morita 3D Accuitomo 80 (J. Morita USA, Irvine, CA) generates isotropic voxels of 80

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microns. Although not necessary for use in every case, this technology, when appropriate, improves visualization and ultimately leads to better care in select situations. A record of exposure and doses must be maintained for each patient.

FIG. 2.13 CS 9300 3D Extra-Oral Imaging System.

A 3D dental scanner with multimodality imaging system has a platform installed to rest face on it while the patient stands.

Source: (Courtesy Carestream Dental LLC, Atlanta, GA.)

Type of patient

Patient size and thus the amount of regional anatomy captured in the study also help determine the FOV. The smallest possible FOV must be chosen for the task at hand. Just because a clinician owns a cone beam machine does *not* mean that every patient should be exposed to a cone beam study.[140] If previous studies are available, they need to be evaluated first in a recall patient. Use of imaging in children must be minimized. Cone beam units with smaller FOVs can somewhat limit the radiation dose to critical organs and tissues of the head and neck in these cases.

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Several CBCT units allow the use of a wheelchair to position special needs patients or a regular chair for patients to sit down during image acquisition. The potential to reduce motion artifacts may be an advantage (Figs. 2.14 and 2.15).

FIG. 2.14 Veraviewepocs 3D R100.

A 3D-dental imaging scanner, Veraviewepocs 3D R100 has a panoramic scout.

Source: (Courtesy JMorita USA, Irvine, CA.)

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FIG. 2.15 CS 8100-3D.

A woman sits under CS 8100-3D in a wheel chair for dental examination.

Source: (Courtesy Carestream Dental LLC, Atlanta, GA.)

Spatial resolution requirements

All endodontic imaging procedures require high spatial resolution. Assessment of canal structure, canal length, and lesions of endodontic origin (LEOs)[189] showing apical change, in addition to an understanding of possible retreatment cases, are important tasks requiring minute detail. If CBCT is used, the data acquisition should be performed at the smallest voxel size: the smaller the voxel size, the higher the spatial resolution. Many of the larger stand-alone cone beam machines, such as the i-CAT (Imaging Sciences International, Irvine, CA), default to a 0.4 mm voxel size. This voxel size is inadequate for high spatial detail. However, these units often have a voxel size selection option that allows smaller voxel sizes to be used during the image acquisition. The absolute maximum voxel size for endodontic imaging should be 0.2 mm.[44] Units typically use voxel sizes of 0.075 to 0.16 mm for their native image capture (Fig. 2.16).

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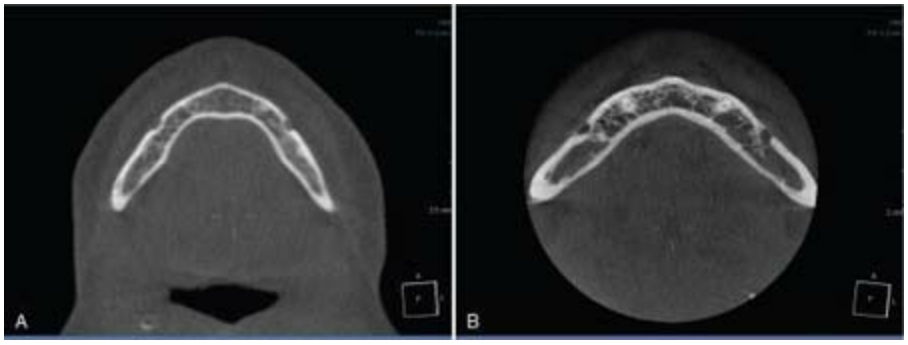

FIG. 2.16 **A,** Axial slice from data obtained with a 0.4 mm voxel size. **B,** Compare the trabecular pattern and outline of the mental foramina to the same location in an axial slice from data obtained with a 0.16 mm voxel size.

A) Radiograph shows inverted U-shaped dental arch with hazy tissue inside.

B) Radiograph shows slightly wide teeth arch with less-density and clear tissues inside.

Imaging tasks improved or simplified by cone beam volumetric computed tomography

The Executive Opinion of the American Academy of Oral and Maxillofacial Radiology and, later, the position paper on the use of CBCT in endodontics jointly developed by the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR), list indications for potential use in selected cases, including evaluation of the anatomy and complex morphology, differential diagnosis of complex pathoses with certain qualifiers, intraoperative or postoperative assessment of endodontic treatment, dentoalveolar trauma, resorption, presurgical case planning, outcomes assessment, and dental implant case planning.[1] Use of CBCT must be determined on a case-by-case basis only. These indications do not in any way mandate the use of CBCT for every case that falls into one of the preceding categories. For endodontic treatment and assessments, there are at least five primary imaging tasks in which CBCT scans have a distinct advantage over traditional 2D radiographs. These tasks include the evaluation of the following factors:

1. Differential diagnosis

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    a. Lesions of endodontic origin
    b. Lesions of nonendodontic origin
2. Evaluation of anatomy and complex morphology
    a. Dental anomalies
    b. Root canal system morphology
3. Diagnosis of endodontic treatment failures
4. Intraoperative or postoperative assessment of endodontic treatment complications
    a. Vertical root fractures
    b. Overextended root canal obturation material
    c. Separated endodontic instruments
    d. Calcified canals
    e. Perforations
5. Presurgical treatment planning
6. Dentoalveolar trauma
7. Internal and external root resorption
8. Endodontic treatment outcomes assessment
9. Dental implant case planning
10. 3D guided endodontics

Differential diagnosis

Lesions of endodontic origin

Clinical endodontic diagnosis relies on subjective and objective information collected during patient examinations. Diagnosis of the pulpal status of the teeth can sometimes be challenging if adequate radiographic information is unavailable. It is fundamental to understand that LEOs arise secondary to pulpal breakdown products and form adjacent to canal portals of exit.[183,190] These radiolucent lesions, formed as a result of loss of bone mineralization, can and do form three dimensionally anywhere along the root surface anatomy.[184] A 30% to 40% mineral content loss is needed for these lesions to be visualized on conventional radiographs.[137] Furthermore, the thickness of the cortical plate covering the lesion may significantly affect the radiographic appearance of the lesion on a conventional image.[229] DSR has been observed to increase diagnostic capability with observers identifying incipient PA

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lesions in more than 70% of the cases.[137]

Before the advent of CBCT, clinicians were unable to routinely visualize the presence, specific location, and extensiveness of PA bone loss using conventional radiography.[127] This was especially true in areas with superimposition of anatomic structures. Visual obstruction from anatomic features, such as buccal bone and the malar process over the apices of maxillary roots, simply "disappears" when the examiner can scroll through the slices of the bone from facial to palatal in 0.1 mm sections while also changing axial orientations. CBCT showed significantly higher rates of detection of PA lesions in maxillary molars and premolars compared to PA radiography.[129] CBCT also showed a significantly higher diagnostic accuracy to detect apical periodontitis (AP) when compared to PA radiographs using human histopathology as the gold standard.[108]

In a comparative investigation of the use of CBCT and PA radiography in the evaluation of the periodontal ligament (PDL), Pope et al.[176] showed that necrotic teeth examined with CBCT had widened PDLs but 60% of vital teeth showed a PA widening of 0.5 mm or more. They called for further investigation to determine whether health and disease can be appropriately judged using CBCT in epidemiologic investigations. See Chapter 3 for more information.

Lesions of nonendodontic origin

Differential diagnosis of PA pathology is crucial to endodontic treatment planning. Substantial evidence in the literature points to a significant chance that lesions of the tooth-supporting structures are nonendodontic in origin, such as PA cemento-osseous dysplasia; central giant cell granulomas; lateral periodontal cyst, simple bone cysts; odontogenic cysts, tumors, or malignancies; and neuropathic pain (Figs. 2.17 and 2.18).[39,68,100,168,179,180]

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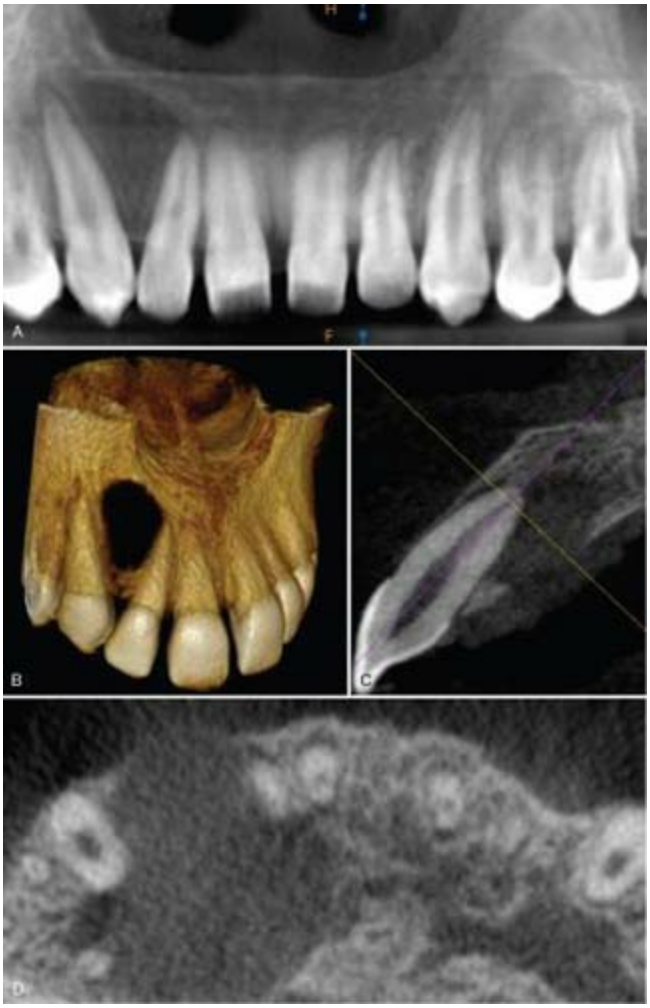

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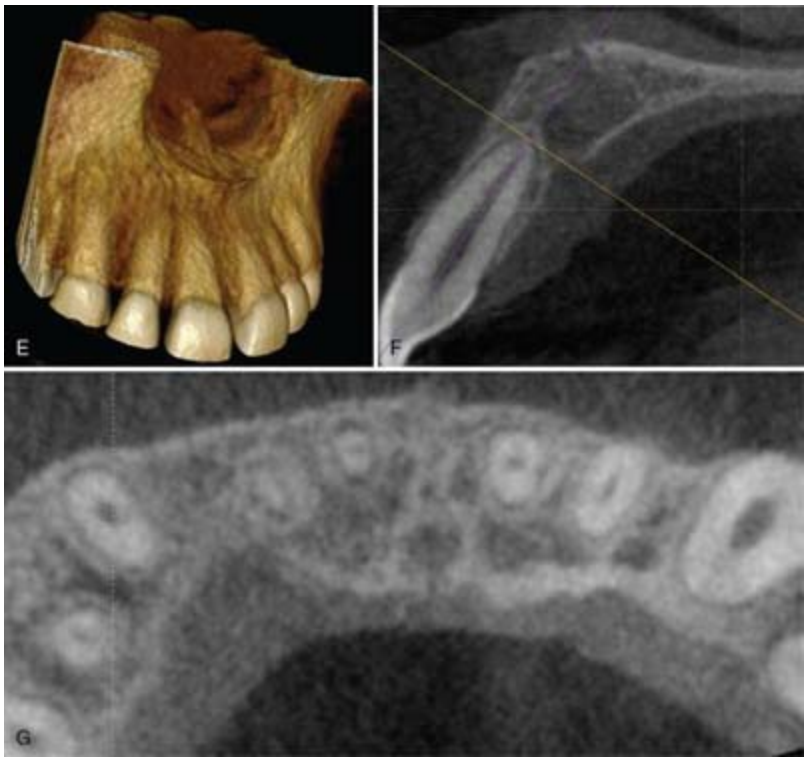

FIG 2.17 Lateral periodontal cyst. A 12-year-old male presented for orthodontic consultation with an elevated lesion in the maxillary right anterior region that was fluctuant under pressure; the patient was referred for endodontic evaluation and radiographic assessment. The pulp vitality was normal for the maxillary anterior dentition. A preoperative CBCT of the anterior maxilla was exposed (**A,** pseudopanoramic reformation; **B,** 3D surface reformation; **C,** cross-sectional reformation of the maxillary right lateral incisor) showing a unilocular/ovoid area of uniform low density that measured approximately 18.0 mm × 15.0 mm. **D,** Axial reformation showing a partially well-defined, noncorticated border mesial to the canine and a poorly defined border between the distal of the right lateral and central incisor. Surrounding tissues showed locally destroyed buccal and palatal cortical plates of the right anterior side of the maxilla with 6 mm of expansion of the buccal and palatal plates in the area between the canine and lateral incisor. Roots of the canine and lateral incisor are displaced mesially and distally, respectively. There was a 3 to 5 mm thickening of the sinus membrane of the overlying floor of the maxillary sinus. Seven months after surgical intervention, the patient was asymptomatic and a follow-up CBCT study was acquired, showing normal healing. **E,** 3D surface. **F,** Cross-sectional. **G,** Axial reformation.

Set of four radiographs marked A through D depict results of a lateral periodontal cyst in the maxillary right anterior region.

E) 3D image shows the front view of teeth with a hollow chamber above it.

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F) Radiograph of the hollow chamber below the root canal.

G) Horizontal view of teeth shows protruding tissue at the center.

Source: (Courtesy Dr. Rina Gonzalez-Albazzaz and Dr. Barry Pass.)

Neuropathic orofacial pain or atypical odontalgia (AO), also known as *chronic continuous dentoalveolar pain* (CCDAP)[153] and *persistent dentoalveolar pain* (PDAP),[98] is related to a tooth, teeth, or pain at an extraction site where no clinical or radiographic pathosis is evident. Two systematic reviews of AO showed the incidence of persistent pain of more than 6 months' duration after nonsurgical and surgical endodontic treatment, excluding local inflammatory causes, was 3.4%.[151] The pathophysiology of this pain is uncertain, but it is hypothesized to involve deafferentation of peripheral sensory neurons in predisposed patients. The diagnosis of AO is challenging and depends on the patient history and clinical examination findings in addition to the absence of radiographic findings. In some cases, the symptoms from AP and AO are closely related. Pigg et al.[173] conducted a study of 20 patients with AO. All the patients had at least one tooth in the region of discomfort that had undergone invasive treatment; 21 of 30 teeth had undergone endodontic treatment. These researchers found that 60% had no PA lesions, and among those who did, CBCT showed 17% more PA lesions than conventional radiography. This study demonstrated that CBCT may be a useful supplement to 2D radiography (see Fig. 2.18).

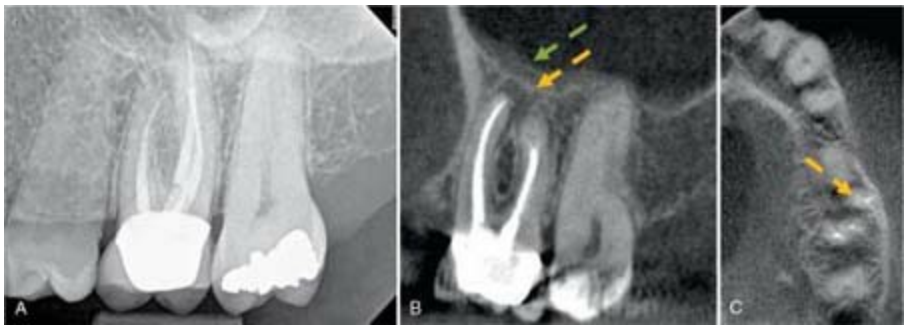

FIG. 2.18 A 62-year-old female was referred for evaluation and possible treatment for persistent dentoalveolar pain (PDAP) in the maxillary left region. The patient could alleviate this condition by placing a moist cotton roll in the adjacent vestibule to prevent the buccal mucosa from contacting the alveolus supporting the maxillary teeth in this area. This condition began after a history of local

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anesthetic administration, persisted for 1 year, and led to three successive new crowns and endodontic treatment on the “offending tooth,” in an effort to remedy the condition without improvement. The response to endodontic tests, TMJ, and myofascial evaluations were normal. Application of topical xylocaine resulted in cessation of pain for 15 minutes. **A,** A PA radiograph showed a root-treated, maxillary left first molar with no apparent radiographic lesion. **B,** A limited FOV CBCT of the maxillary left posterior was acquired. The corrected sagittal view showed an approximately 4 mm, well-defined, ovoid, mildly corticated area of low attenuation (radiolucent) centered over the apex of the mesiobuccal root and extending to the junction of the middle and apical third of the mesiobuccal root *(yellow arrow)*. There was mild mucositis *(green arrow)*. **C,** There was a previously untreated mesioaccessory canal *(yellow arrow)* and mild mucositis. A diagnosis of neuropathic pain and a chronic apical periodontitis was made. Daily application of topical ketamine, gabapentin, and clonidine was prescribed. Endodontic revision of the maxillary left first molar was performed 3 months after the patient was stabilized with the topical medications.

Set of three radiographs marked A through C depict results of evaluation of a 62-year-old female with a complaint of persistent dentoalveolar pain (PDAP) in the maxillary left region.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D [Carestream Dental, LLC. Atlanta, GA].)

The 3D radiographic appearance of a PA lesion provides additional information about the lesion’s relationship to the tooth and other anatomic structures (e.g., the vascular bundle) and about the aggressiveness of the lesion. This information, along with pulp sensitivity testing, is useful for adequate treatment planning and management of these conditions (see also Chapter 3).

Evaluation of anatomy and complex morphology

The precise location and visualization of dental anomalies, root morphology, and canal anatomy are vastly improved with CBCT data. Root curvature, additional roots, and anomalies within the canals themselves (e.g., obstructions, narrowing, bifurcation) are made more apparent when all three anatomic planes of section are available for review, especially with the capability of narrowing the slice thickness to as little as 0.075 mm. Visual obstruction from anatomic features such as buccal bone and the malar process

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over the apices of maxillary roots simply will “disappear” when you can scroll through the slices of the bone from facial to palatal in 0.076 mm sections while also changing axial orientations (Fig. 2.19).

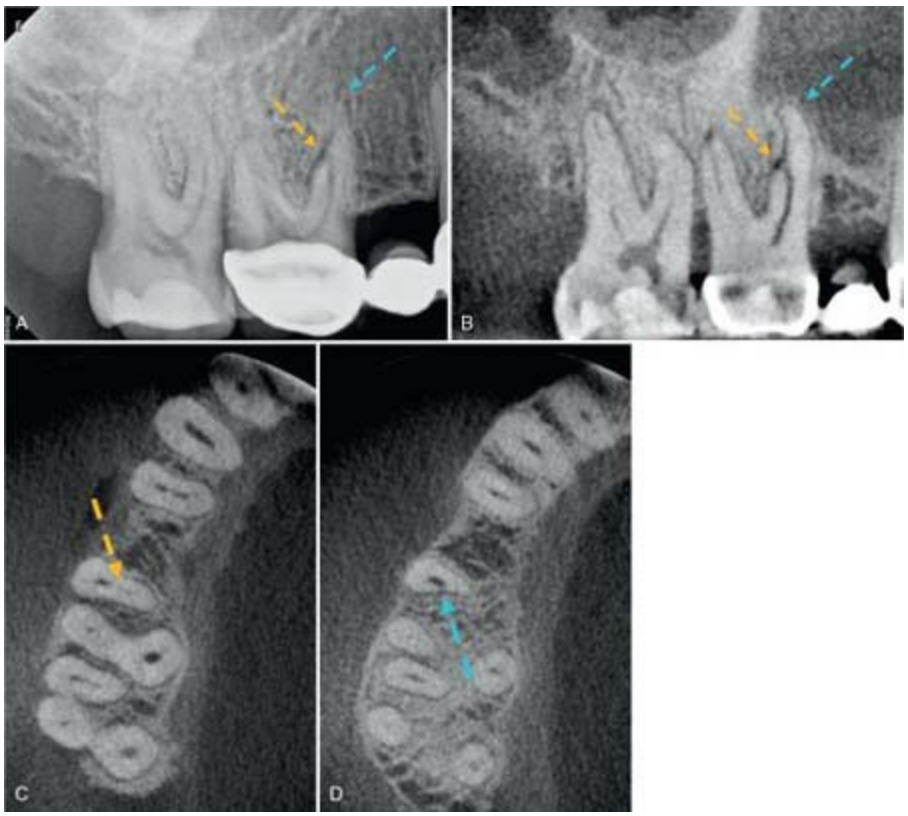

FIG. 2.19 This patient was referred for evaluation and possible treatment after emergency pulp extirpation performed by others. **A,** This PA radiograph shows that the location of the physiologic terminus *(yellow arrow)* and radiographic apex *(blue arrow)* did not appear to coincide within the normal range. **B,** Contemporaneous CBCT sagittal reformation shows the anomalous location of the physiologic terminus *(yellow arrow)* and radiographic apex *(blue arrow)*. **C,** Presence of a mesioaccessory canal with isthmus *(blue arrow)* and **(D)** an oval-shaped canal *(yellow arrow)* is apparent.

Four radiographs are marked A through D.

A) The central tooth is surrounded by a small lesion at the tip of root.

B) Close-up view shows two small holes at the tip of root and canal terminus.

C) Teeth arranged vertically shows mesioaccessory canal.

D) Close-up view of tooth shows mesioaccessory canal and expanded tissue.

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Source: (Courtesy Dr. Anastasia Mischenko, Chevy Chase, MD. Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Dental anomalies

The use of CBCT technology has been reported in the diagnosis and treatment planning of various dental anomalies (e.g., dens invaginatus) that often have complex morphologic presentations.[152] The prevalence of dens invaginatus was as high as 6.8% in the adolescent Swedish population studied.[21] The complex nature of the anomaly presents a diagnostic challenge when conventional radiographs are used.[160] In case reports in which CBCT was used for diagnosis and treatment planning, treatment options included conservative endodontic treatment of the invagination, surgical treatment of the PA pathology, and complete revascularization of the dens after removal of the invagination (Fig. 2.20).[144,222]

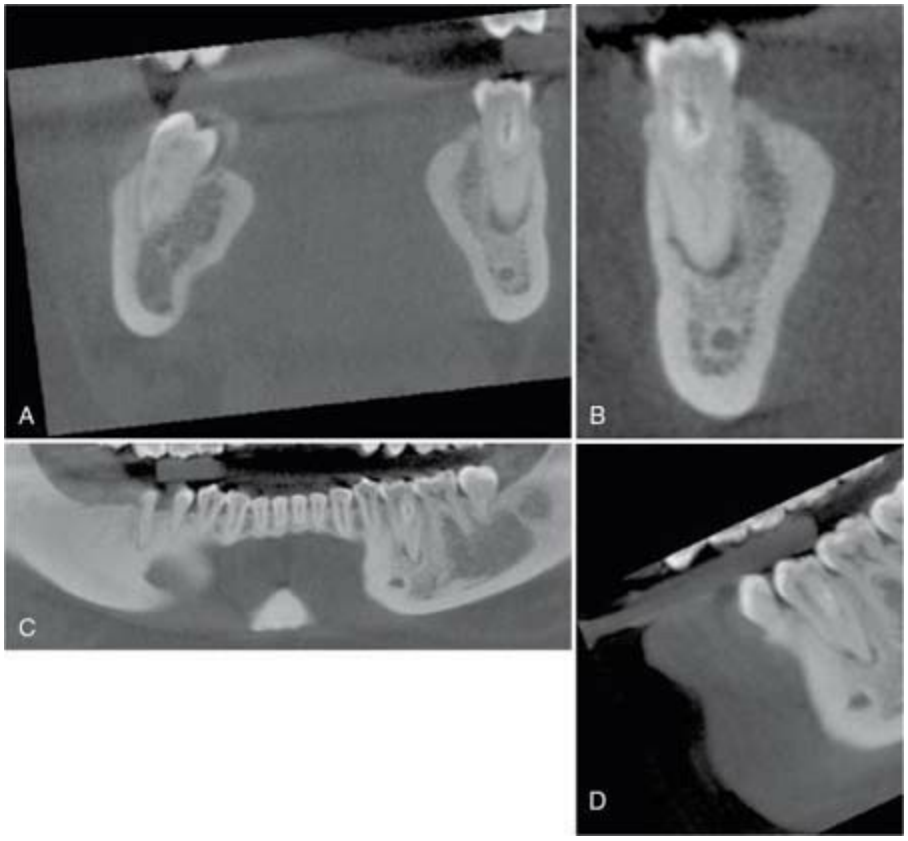

FIG. 2.20 **A,** Dens en dente of the mandibular left second bicuspid;

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coronal slice. **B,** Coronal view. **C,** Panoramic reconstruction from CBCT. **D,** Sagittal view.

Set of four radiographs are marked A through D as follows:

A) Two teeth show the invagination of amelodental structure.

B) Close-up view of tooth with amelodental structure inside.

C) Panoramic view of lower jaw shows an addition triangular structure below the teeth.

D) Sagittal view shows close-up of tooth with amelodental structure at the center.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Root canal system morphology

As the adage goes, nature seldom makes a straight line and never makes two of the same. This statement is dramatically illustrated in the evaluation of root canal system morphology. With ever-present unusual and atypical root shapes and numbers, there is sometimes a need to look further than what a clinician can see or imagine with 2D radiography (Fig. 2.21). Variations in root canal morphology have been studied using various in vitro techniques.[70,174,220,221] The results of these studies point to the fact that there is significant variation in the root canal morphology among various ethnic population groups.[8,90,91,149,224] CBCT has been reported to be comparable to canal staining and clearing techniques for identification of the root canal morphology,[145] and CBCT studies report variation in the root canal morphology among various ethnic groups.[146,204,236,238] Accuracy of CBCT imaging for detection of second mesiobuccal canal in maxillary molars has been shown to be 96%, and the prevalence of midmesial canals in mandibular molars was found to be 16.4%.[203]

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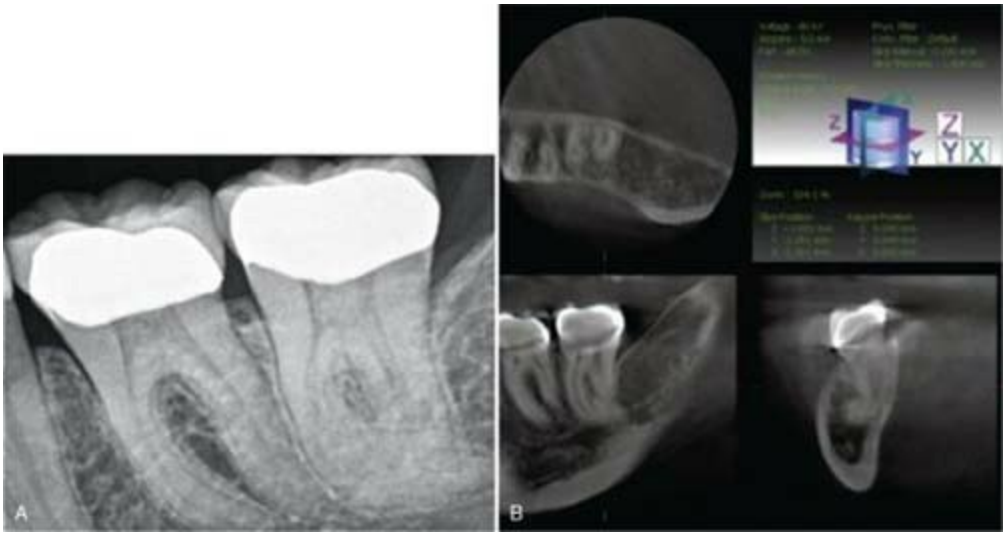

FIG. 2.21 **A,** Mandibular left second molar referred for endodontic evaluation and possible treatment. This 2D radiograph reveals significant pulp stones and canal calcification developing not only in the coronal aspect of the root canal system, but also extending down the visible distal canal. The apical third of the canal system appears unusual and dilacerated. Cone-beam computed tomography (CBCT) would be beneficial in visualizing the root canal anatomy, to create the ideal endodontic access. **B,** Single slice of the CBCT image for the same tooth. Information about the direction of the root canal anatomy is provided in all three planes of the section: axial, coronal, and sagittal. Interestingly, the axial slice shows that the mesial lingual root actually traverses buccally as it approaches its terminal extent. This is valuable information for the clinician before the entire root canal system is cleaned and shaped; it may also establish a higher degree of treatment predictability.

Set of two 2D radiographs marked A and B depict endodontic evaluation and treatment of mandibular left second molar.

Diagnosis of endodontic treatment failures

The failure of previous endodontic therapy can be attributed to various factors, such as procedural errors, missed canals, or persistent PA pathosis. Knowledge of the cause of failure is pertinent to the treatment of these cases because it allows the cause to be adequately rectified. With the advent of CBCT, in select cases of retreatment in which the cause of failure is otherwise undetectable, adequate information may be collected to apply to the treatment plan. Use of CBCT in these situations will aid in treatment

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planning and improve outcomes (Fig. 2.22).[109] A study by Rodriguez et al. showed that the endodontic retreatment strategies were altered in 49.8% of cases after CBCT images were included for diagnosis by general dentists and endodontists.[181]

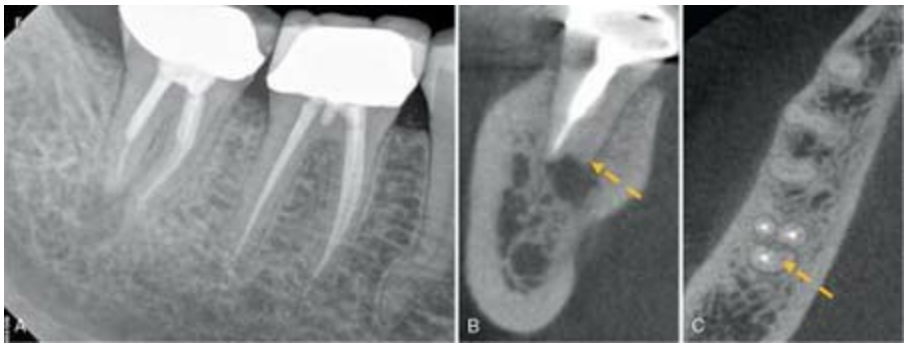

FIG. 2.22 This 38-year-old female patient presented for evaluation and treatment of a symptomatic mandibular right second molar, which had been endodontically treated more than 10 years previously. This tooth was sensitive to percussion and biting forces; periodontal findings were normal. Microscopic examination of the exposed dentin was negative for a vertical fracture. **A,** The PA radiograph showed the previous endodontic treatment; a post present in the distal canal; and an approximately 5 mm diameter, unilocular, well-defined area of uniform low density centered at the periapex of the distal root—features consistent with an apical periodontitis. Contemporaneous **(B)** CBCT sagittal and **(C)** axial reformations revealed a previously untreated distobuccal canal *(yellow arrow)*.

Three radiographs are marked A through C as follows:

A) Two teeth with radiopaque crown and two root canals. The tissue at the tip is less dense.

B) A dashed arrow pointing at a tooth depicting inflammation at the tip of root.

C) Axial view shows a dashed arrow pointing at the three round structures, each with a denser tiny dot at the center in the distobuccal canal.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Use of CBCT in evaluation of previously endodontically treated teeth aids in visualizing missed canals, canal deviations, resorption, procedural errors

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such as perforation, and other pathology.

The technology is most useful in detecting uninstrumented and unfilled canals, extension of the root canal filling, and the presence and extent of periradicular bone loss. The incidence of missed canals in failed endodontic treatments were found to be 23.04% with the highest incidence in upper molars. These teeth had 4.38 more likelihood of having a PA lesion.[109] The sensitivity of CBCT and PA radiographs for diagnosing strip perforations in root-filled teeth has been shown to be low, although CBCT showed a significantly higher sensitivity when compared to PA radiographs.[194] Radiopaque filling materials in the root canals of endodontically treated teeth can produce streak artifacts, which can mimic fracture lines or perforations.[194,237] Use of lower exposure parameters and nonmetallic fiber posts can significantly reduce these artifacts, allowing for an improved diagnosis.[59]

Intraoperative or postoperative assessment of endodontic treatment complications

Vertical root fractures

Vertical root fractures (VRFs) that run along the long axis of a tooth are often difficult to diagnose clinically. The prevalence of VRF in endodontically treated teeth has been reported to range from 8.8% to 13.4%.[77,207,233] These fractures typically run in the buccolingual direction and are confined to the roots, making it difficult to visualize the fracture. Visualizing the fracture on a conventional radiograph is possible when the x-ray beam is parallel to the plane of the fracture.[182] Challenges in diagnosis with regard to the extent and exact location of the fracture often lead to unwarranted extraction of teeth. Since the introduction of CBCT to dentistry, various reports of the application of the technology to detect vertical root fractures have been published. The reported sensitivity for detection of VRFs has ranged from 18.8% to 100%[136]; by comparison, conventional radiographs have a reported sensitivity of approximately 37% (Fig. 2.23).[62,95] CBCT has been used to visualize VRFs in controlled clinical studies in which clinical diagnosis was difficult.[62] VRFs were successfully detected at a spatial resolution ranging

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from 76 to 140 microns. However, only a limited number of units provide such high resolution. A comparison of various CBCT units for the detection of VRFs demonstrated that the units with flat panel detectors (FPDs) were superior to the image intensifier tube (IIT)/CCD–based detectors; the smaller FOV and the ability to view axial slices also improved detection of VRFs.[96] Continued improvement of sensor technology, including the use of FPDs, has resulted in enhanced resolution. Voxel dimensions are smaller in these units. Detection of vertical root fractures with thickness ranging from 0.2 to 0.4 mm was found to be more accurate with CBCT than with digital radiography.[155,161] The presence of root canal filling in the teeth lowers the specificity of CBCT in detecting vertical root fractures[95,96,111]; this has been attributed to the radiopaque material causing streak artifacts that mimic fracture lines.[237]

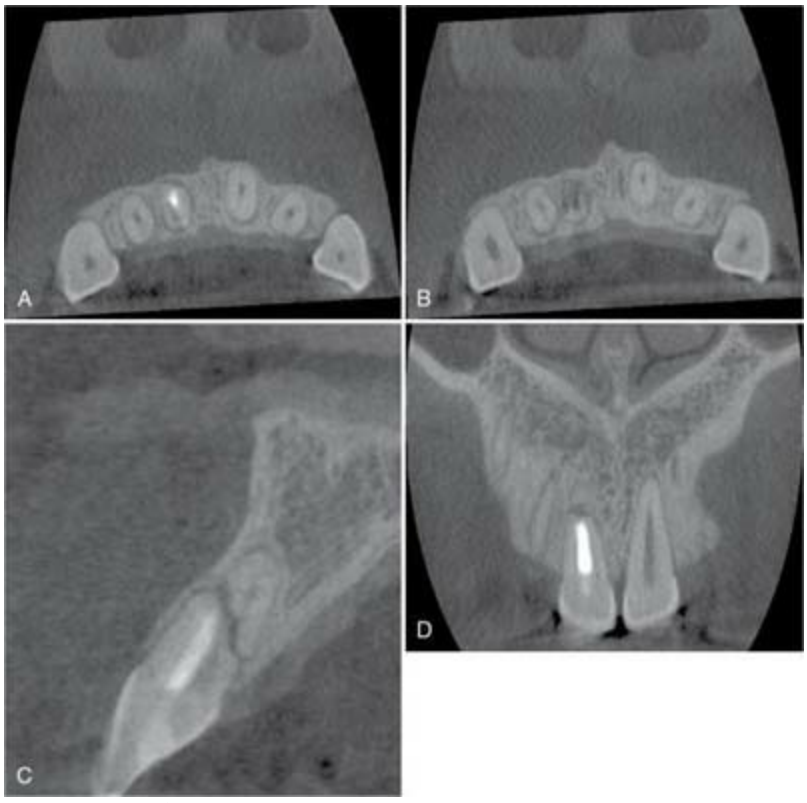

FIG. 2.23 Root fracture in an endodontically obturated maxillary right central incisor. **A,** Axial view with artifacts from a highly attenuating (opaque) obturant. **B,** View without artifacts from obturant. **C,** Oblique parasagittal view. **D,** Paracoronal view.

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Four radiographs are marked A through D as follows:

A) Panoramic view of upper jaw shows a tooth with artifacts that appears hazy.

B) Upper jaw shows tooth at the center with fractured root.

C) Tooth shows the diagonal fracture in-between root.

D) Two teeth are shown. One tooth on the left has radiopaque tissue in root, whereas the tooth on the right has radiolucent tissue.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Zhang et al. reported a low sensitivity of 33.3% for detection of subtle vertical root fractures in a clinical study. However, the study showed the presence of vertical bone loss in a high number of the cases.[235] Bone loss pattern around teeth with vertical root fractures viewed on CBCT images may assist clinicians in diagnosis of VRF.[211]

Overextended root canal obturation material

CBCT scans provide the opportunity to map endodontic treatment complications through the examination of 3D representations of the teeth and supporting structures in different planes. Few high-level studies related to the effects of endodontic treatment complications have been published in the endodontic literature.[206] However, it is generally recognized that the overfilling of the root canal, causing damage to vital structures such as the inferior alveolar neurovascular (IAN) bundle (Fig. 2.24) or the maxillary sinus, can cause significant morbidity.[34,35,76,80]

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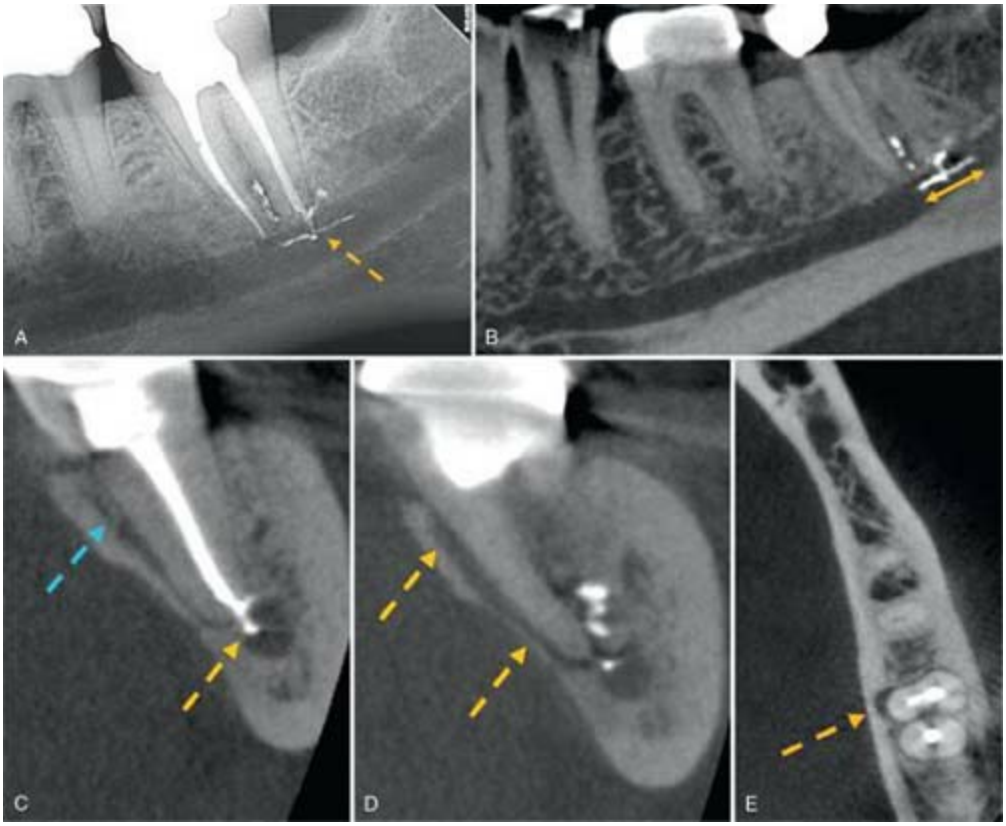

FIG. 2.24 This 64-year-old male patient presented with sensitivity when biting on the mandibular left second molar. The history included endodontic revision more than 6 months earlier and subsequent transient paresthesia and dysesthesia along the distribution of the inferior alveolar nerve, which persisted for 1 week after the retreatment. Epicritic and protopathic sensibility was normal. The results of periodontal probing, staining and microscopic examination of the exposed root surface at the sulcus were normal. **A,** Initial PA radiograph showed the approximate location of excess radiopaque material, a feature consistent with extruded root canal sealer *(yellow arrow)*. **B,** Contemporaneous CBCT corrected sagittal reformation shows approximate length of extruded material, measuring 3.4 mm *(yellow arrow)*. **C and D,** Corrected cross-sectional reformation shows the foreign material interior to the inferior alveolar canal *(yellow arrow)*, an area of low attenuation extending from the apex superiorly to an area near the alveolar crest *(blue arrow)*, and erosion of the lingual endosseous surface. **E,** The same feature was shown in the corrected axial view *(yellow arrow)*, bisected by a dark line extending from the obturant to the surface of the root—features suggestive of a vertical fracture. Examination of the extracted tooth revealed a vertical fracture at the lingual aspect of the distal root.

Five radiographs marked A through E depict results of periodontal probing to treat sensitivity in mandibular left second molar.

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Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Endodontic therapy undertaken in close proximity to the IAN bundle should receive special attention because direct trauma, mechanical compression, chemical neurotoxicity, and an increase in temperature greater than 10°C may cause irreversible damage.[65,85,88,214] Scolozzi et al. reported that sensory disturbances can include pain, anesthesia, paresthesia, hypoesthesia, and dysesthesia.[191] The IAN bundle is located in the cribriform bone-lined mandibular canal and courses obliquely through the ramus of the mandible and horizontally through the mandible body to the mental foramen and the incisive foramen.[11] There are many anatomic variations of the IAN bundle, including the anterior loop and bifid mandibular canals.[45] Kovisto et al.[116] used CBCT measurements from 139 patients to show that the apices of the mandibular second molar were closest to the IAN bundle. In females, the mesial root of the second molar was closer than in males, and the distances in all roots measured increased with the age of the patient. There was a high correlation between the measurements from left to right side in the same patient, and an average distance of 1.51 to 3.43 mm in adults.[116] Procedures involving the mandibular second molar were most likely to cause nerve damage.[126] Further research is required to clarify the risks and benefits of CBCT when endodontic treatment is contemplated on teeth with a proximal relationship between the IAN and root apices. Porgrel treated 61 patients with involvement of the IAN bundle after root canal therapy during a 7-year period. Eight patients were asymptomatic; 42 patients were seen for mild symptoms or were examined more than 3 months postoperatively, with only 10% experiencing improvement. Five patients underwent surgical treatment before 48 hours elapsed and recovered completely. Six patients underwent surgery between 10 days and 3 months, with four experiencing partial recovery; the remaining two had no improvement.[175]

New imaging technologies, such as high-resolution magnetic resonance imaging (MRI-HR) and magnetic resonance neurography (MRN), promise to improve isolation of the IAN from the neighboring artery and vein contained within the inferior alveolar bony canal. MRN studies have documented the ability to demonstrate nerve continuity and localize extraneural nerve

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compression before surgical nerve exploration. Postoperative PA radiographs should be exposed on the day of endodontic treatment completion or a suspected iatrogenic event, and any suspected compromise of the IAN bundle or other vital structures should be evaluated immediately. In all cases in which trauma to the IAN bundle is suspected from PA or panoramic radiography or by the report of symptoms consistent with nerve injury, exposure of a CBCT image volume should be considered. It is generally accepted that immediate surgical debridement should be attempted to maximize recovery.[65,177] With the introduction of MRI algorithms for dental diagnostic purposes, it is expected that this imaging modality will be increasingly used in diagnostic and treatment planning. MRI has the capability to demonstrate vascularity to the tooth of interest, in addition to the presence of inflammatory exudates in the apical regions, without exposing the patient to ionizing radiation. Receiver coils are being developed to enhance the image quality of maxillofacial and dental magnetic resonance studies.

The accidental introduction of root canal instruments, irrigating solutions, obturation material, and root tips into the maxillary sinus has been reported. Serious consequences associated with the intrusion of foreign bodies into the maxillary sinus include pain, paresthesia, and aspergillosis, a rare but well-documented complication of endodontic treatment.[24] Guivarc'h et al. reported that the overextension of heavy metal–containing root canal sealers, such as zinc oxide eugenol cement, may promote fungal infection in immunocompromised patients, leading to bone destruction and damage to adjacent structures. This case report described the use of CT to assess the patient before surgery and at 6 months.[89] The use of CBCT as an aid in the localization and retrieval of an extreme overextension of thermoplasticized injectable gutta-percha into the sinus and contiguous soft tissues has been described by Brooks and Kleinman.[37]

Separated endodontic instruments

Instrument fracture can occur at any stage of endodontic treatment, and in any canal location. The incidence of this complication, reported in clinical studies on a per canal or per tooth basis, ranges from 0.39% to 5.0%.[56,157] Molars are predominantly affected by instrument fracture, with the highest

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incidence found in the apical third of mandibular molars.[7,52,123,147]

A systematic review and meta-analysis showed that when endodontic treatment was performed at a high technical standard, instrument fracture did not significantly reduce the prognosis. More specifically, when no initial radiographic PA lesion was present, 92.4% of cases remained healthy; when a PA lesion was present initially, 80.7% of PA lesions showed radiographic healing. However, the presence or absence of periradicular lesions on preoperative and postoperative examinations was based on planar radiographic assessments, which calls these findings into question.[156] Other studies showed that the chances of endodontic failure increased if the canal system could not be thoroughly disinfected, if a periradicular periodontitis was present, or if technical standards were compromised.[51,110,198,200] Use of CBCT to triangulate the retained instrument and assess the canal shape, especially in cases in which the operating microscope does not allow direct visualization, can be helpful in formulating a removal strategy. If the fractured instrument is lodged in the lingual aspect of a ribbon-shaped canal, for example, an instrument may be inserted toward the buccal to bypass and remove the imbedded instrument without forcing the fragment further apically. Without the use of CBCT, intracanal instruments can be reliably removed or bypassed in 85.3% of cases if straight-line access is possible; however, reliable removal or bypass is possible in only 47.7% of cases if the instrument is not visible (Fig. 2.25).[147] When a separated instrument is lodged in the apical third of a root canal, the chances of retrieval are the lowest, but the apical terminus may be adequately sealed by treatment of an anastomosing canal, if present.[79] The possibility of instrument removal based on CBCT triangulation has not been published to date.

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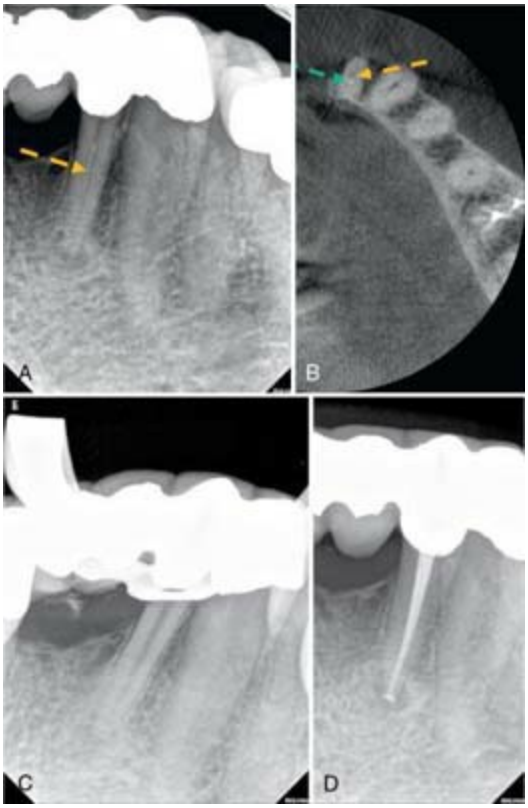

FIG. 2.25 Unexpected torsional and flexural failure of endodontic instruments can occur during instrumentation. **A,** PA radiograph shows a separated file *(yellow arrow)* located at the midroot of the mandibular left lateral incisor in a patient referred for revision treatment. **B,** To aid development of a retreatment strategy, CBCT was used to localize the instrument *(yellow arrow)* in the lingual canal, with identification of the buccal canal *(green arrow)* to facilitate bypassing the instrument and **(C),** subsequent removal. **D,** Obturation was completed without complication.

Four radiographs are marked A through D as follows:

A) Incisor shows a vertical fracture in the root.

B) Axial view shows fracture in the lingual canal of tooth.

C) Incisor shows wedge-shaped radiolucency in the root.

D) Incisor shows wedge-shaped radiopaque region in the root.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Calcified canals

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According to the Pew Research Center, 10,000 US individuals will reach the age of 65 every day until 2030, and the nation's 65-year-old and over cohort will grow to 81 million in 2050, up from 37 million in 2005.[171] This aging population will present increasing challenges for dental clinicians because calcification of the root canal system increases as part of the natural aging process,[86] possibly leading to more untreated canals that may serve as a niche for microorganisms.[28,104] Pulp chambers in the crown of the tooth decrease in size, forming more rapidly on the roof and floor of posterior teeth.[219] Typically, root canals calcify at the coronal aspect first, with decreasing calcification as the canal travels apically. Magnification and illumination are essential tools for the identification and treatment of calcified canals, but CBCT can assist in the perioperative treatment of such conditions.[23] Preoperative assessment of calcified teeth using CBCT can suggest the best tactic for locating calcified canals in the chamber floor and roots using software-based measurement tools. The insertion of radiopaque markers, such as instruments or obturation material, can facilitate reliable canal localization using available multiplanar reformations. The increased sensitivity and specificity provided by CBCT can also assist in the determination of the PA status of calcified root canals that may not require measures that can lead to procedural errors, such as off-course access, instrument fracture, or root perforation.[118] The difficulty in locating calcified canals can be further compounded by morphologic anomalies associated with gender and ethnic origin.[192] CBCT can be an important adjunct to magnification and illumination in these cases.

Perforations

A *perforation* is defined as a "mechanical or pathologic communication between the root canal system and external tooth surface"[9]; it is usually associated with an iatrogenic event, accounting for about 10% of all nonhealed cases.[102] Root perforations can be caused by a post preparation, the search for a calcified canal, a strip perforation, or an attempt to retrieve a fractured instrument. They are often difficult to localize with conventional imaging because no information about the buccolingual dimension can be obtained.[232] Shemesh et al.[194] compared the sensitivity and specificity of

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CBCT scans with two-angulation PA imaging using phosphor plates to assess the likelihood of detecting root or strip perforations after root canal treatment with laterally compacted gutta-percha and sealer. They found that the two methods showed similar specificity, but the CBCT image volumes showed higher sensitivity. Single-angulated PA radiographs showed 40% of the perforations, and two-angled PAs showed 63%, suggesting that if PA radiographs alone are used, two-angled images were superior. There was no significant difference in the detection of root perforations between PA and CBCT radiography. The researchers noted that the results may have been affected by the small size of the perforations and that the method of obturation did not favor extravasation of obturation material.[194] CBCT images suffer from beam hardening artifact resulting from root canal obturation and restorative materials (e.g., gutta-percha, posts, and perforation repair materials), which creates challenges to the interpretation of root integrity. An approach advocated by Bueno et al.[40] suggested that a map-reading strategy of viewing sequential axial slices reduces the beam hardening effect. Improved CBCT software algorithms are expected to reduce artifact formation in the future.

Presurgical treatment planning

Surgical endodontic treatment is often performed in cases of endodontic nonhealing when nonsurgical retreatment is not possible. In the past, conventional and digital 2D PA radiographs were the only means of assessing the apical region. Unfortunately, the information available from these images may not adequately prepare the clinician to treat the pathosis surgically. For example, the clinician may be unable to observe whether the lesion has perforated the buccal or palatal cortical plates, as in the example that follows, or even observe which root or roots are involved. Presurgical confusion is resolved with cone beam imaging. Multiplanar views allow the clinician to see the defect and suspected causes from the axial, sagittal, and coronal aspects; 3D grayscale or color imaging helps the clinician visualize the entire defect before the incision is made. This is an immense improvement over conventional imaging and helps with better assessment of the prognosis prior to the surgical treatment (Fig. 2.26).

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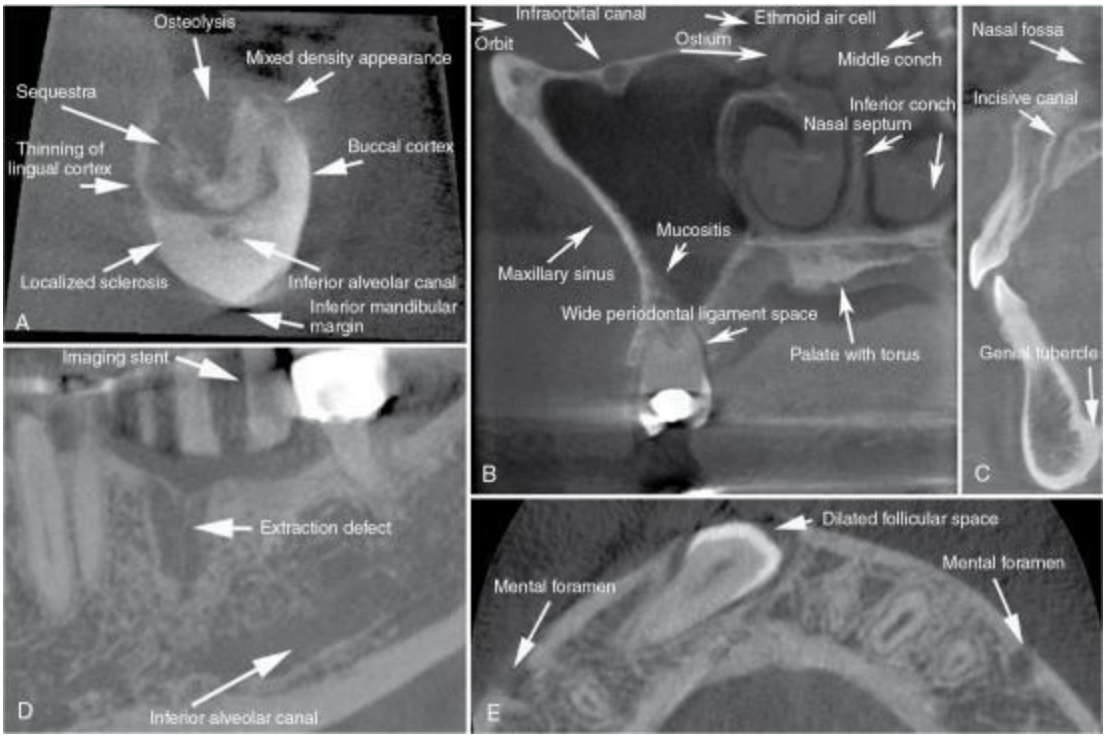

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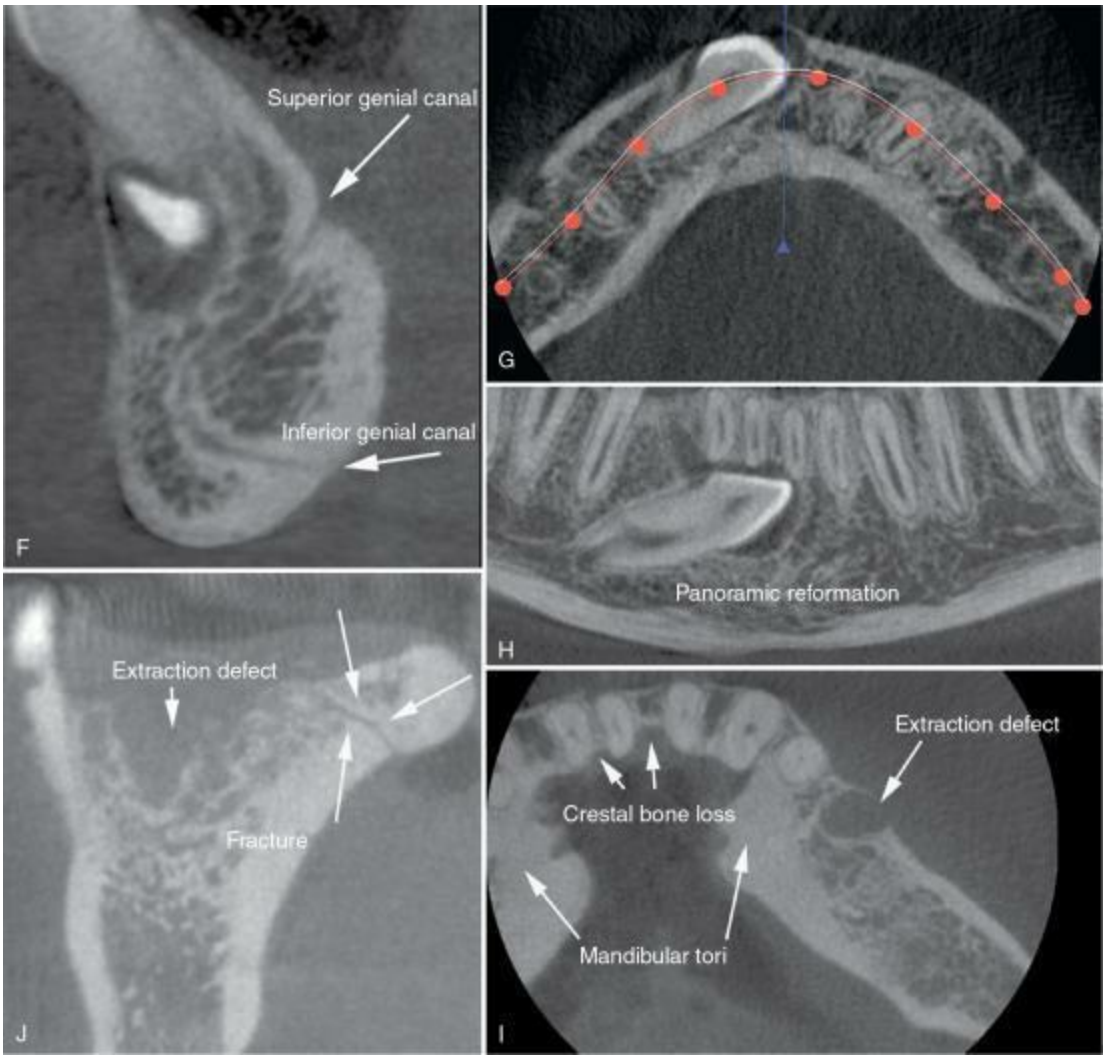

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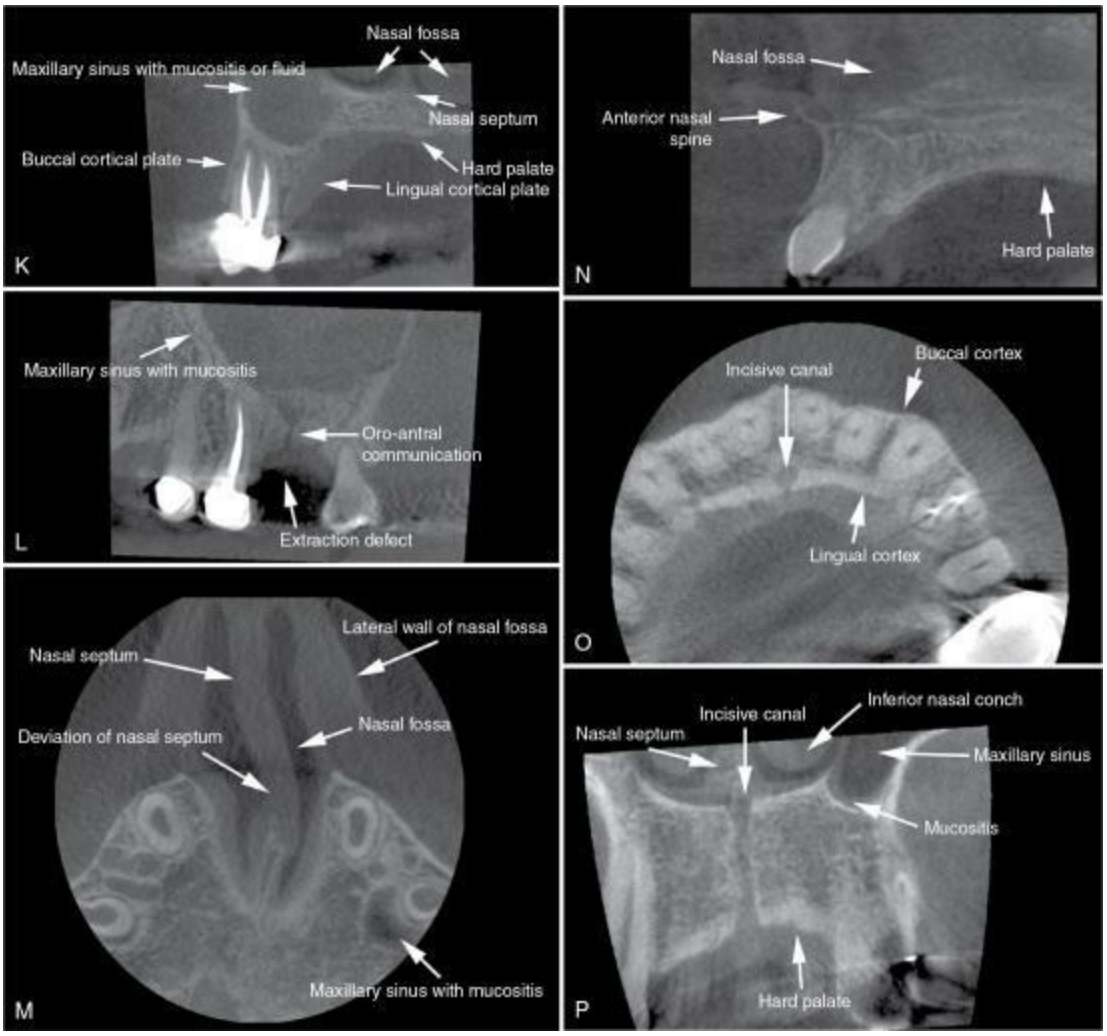

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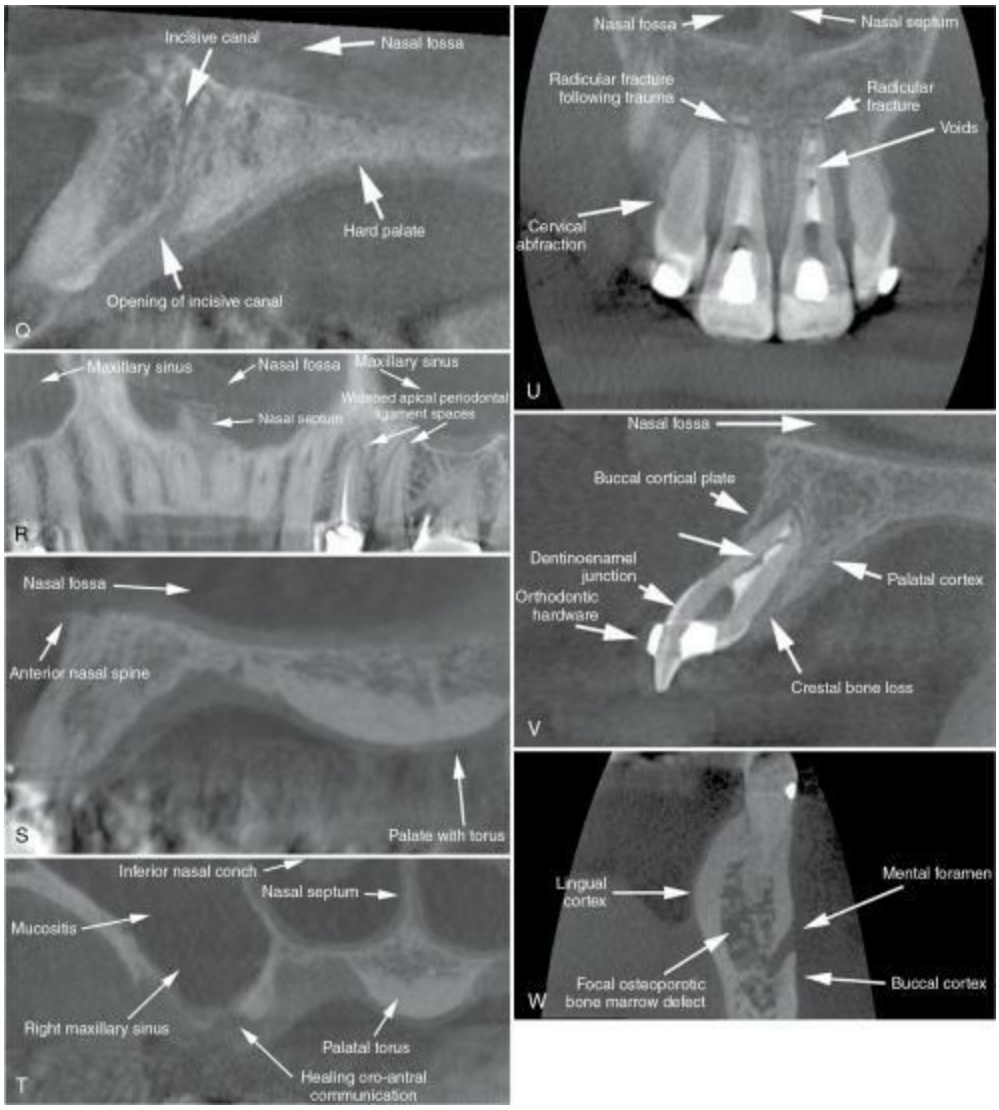

FIG. 2.26 **A** to **W,** Anatomic landmarks on CBCT images acquired using CS 9000, CS 9300, and iCAT units.

Five radiographs, A through E, show labels as follows:

A) Osteolysis, mixed density appearance, buccal cortex, inferior alveolar canal, inferior mandibular margin, localized sclerosis, thinning of lingual cortex, and sequestra.

B) Infraorbital canal, ostium, ethmoid air cell, middle conch, inferior conch, nasal septum, palate with torus, mucositis, wide periodontal ligament space, and maxillary sinus.

C) Nasal fossa, incisive canal, genial tubercle.

D) Imaging stent, extraction defect, inferior alveolar canal.

E) Mental foramen, dilated follicular space, mental foramen.

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Five radiographs, F through I, show labels as follows:

F) Superior genial canal, inferior genial canal.

G) Axial view of mandible.

H) Panoramic reformation.

I) Crestal bone loss, mandibular tori, extraction defect.

J) Extraction defect, fracture.

Six radiographs, K through P, show labels as follows:

K) Nasal fossa, nasal septum, hard palate, lingual cortical plate, buccal cortical plate, maxillary sinus with mucositis or fluid.

L) Maxillary sinus with mucositis, oro-antral communication, extraction defect.

M) Lateral wall of nasal fossa, nasal fossa, maxillary sinus with mucositis, deviation of nasal septum, nasal septum.

N) Nasal fossa, anterior nasal spine, hard palate.

O) Incisive canal, buccal cortex, lingual cortex.

P) Nasal septum, incisive canal, inferior nasal conch, maxillary sinus, mucositis, hard palate.

Seven radiographs, Q through W, show labels as follows:

Q) Incisive canal, nasal fossa, hard palate, opening of incisive canal.

R) Maxillary sinus, nasal fossa, maxillary sinus, nasal septum, widened apical periodontal ligament spaces.

S) Nasal fossa, anterior nasal spine, palate with torus.

T) Inferior nasal conch, nasal septum, mucositis, right maxillary sinus, palatal torus, healing oro-antral communication.

U) Nasal fossa, nasal septum, radicular fracture following trauma, cervical abfraction, voids.

V) Nasal fossa, buccal cortical plate, dentinoenamel junction, orthodontic hardware, palatal cortex, crestal bone loss.

W) Lingual cortex, focal osteoporotic bone marrow defect, mental foramen, buccal cortex.

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Source: (Carestream Dental, Atlanta, GA [CS 9000, CS 9300]; and Imaging Sciences International, Hatfield, PA [iCAT].)

The relationship of the teeth and the associated pathology to important anatomic landmarks must be taken into consideration in the treatment planning for endodontic surgical procedures. These anatomic landmarks include, but are not limited to, the maxillary sinus, the mandibular canal, the mental foramen, the incisive canal, and the buccal and lingual/palatine cortical plate. The close proximity of the maxillary posterior teeth to the sinus has been linked to maxillary sinusitis of odontogenic origin; changes in the maxillary sinus have ranged from thickening of the Schneiderian membrane to actual accumulation of fluid in the sinuses.[130,131,134] The relationship of the roots of the posterior teeth to the sinus during presurgical treatment planning and the changes within the sinus can be best appreciated with the use of CBCT images.[32,131,193]

The relationship of the roots of the mandibular posterior teeth and associated PA pathoses to the mandibular canal, the presence of an anterior loop, and the distance of the mandibular canal from the buccal and lingual cortical plates are pertinent pieces of information when surgical procedures in mandibular posterior teeth are planned.[31] The 3D nature of this relationship can be best assessed using CBCT.[113] A potential difference in the location of the mandibular canal with respect to age and gender has been reported.[116,197]

The determination of the thickness of the buccal cortical plates and anatomy of the root apex prior to endodontic surgical procedures enables the clinician to preplan the osteotomy and the root end resection. The thickest buccal cortical plate was determined to be in the second molar area as per a recent study.[234]

Dentoalveolar trauma

Dental trauma is common during the lifespan, with prevalence in the primary dentition of approximately 30% and in the permanent dentition of approximately 20%.[12] Systematic epidemiologic data at a large medical center suggest a prevalence of 48.2% for dental injuries in all facial trauma, resulting in injuries to the dentition in 57.8% of household and play accidents, 50.1% of sports accidents, 38.6% of work-related accidents, 35.8%

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of acts of violence, and 34.2% of traffic accidents, with 31% unspecified.[78] The prevalence of traumatic dental injuries varies according to the population studied, but these injuries occur most commonly in children 7 to 10 years of age (also see Chapter 21).[18] Dental traumatic injuries affect one fourth of all schoolchildren, with most injuries occurring before the age of 19.[81] Maxillary central incisors sustain approximately 80% of all dental traumatic injuries, followed by maxillary lateral incisors and mandibular incisors.[16] The most common type of traumatic dental injuries in the primary dentition are luxation injuries, whereas crown fractures are the predominant dental injury to the permanent dentition.[117] Determination of the extent of injury to the dentinopulpal complex requires a methodical approach that evaluates the teeth, periodontium, and associated structures (Fig. 2.27) and may result in significant long-term complications.[55]

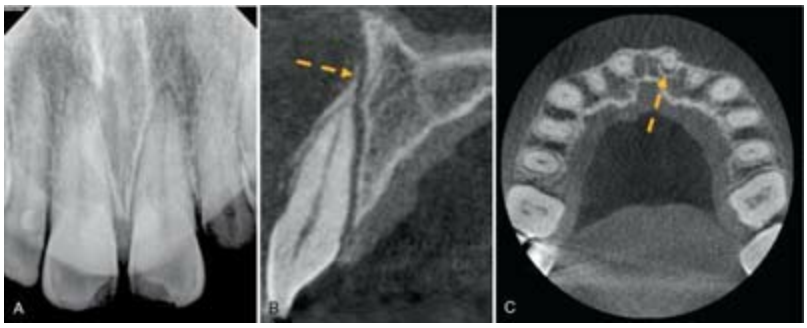

FIG. 2.27 After a traumatic injury to the maxillary right and left central incisors, crown fractures were noted. The PA radiograph showed an extrusive luxation injury in the maxillary left central incisor **(A).** The alveolar fracture (**B,** *yellow arrow*) and true extent of the displacement (**C,** *yellow arrow*) became evident with CBCT.

Three radiographs are marked A through C as follows:

A) Two teeth show part of the crown being broken.

B) Sagittal view of the fractured tooth shows less-density tissue around the periphery of root followed by porous tissue.

C) Panoramic view of teeth shows the fractured tooth at the center.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

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Trauma to the orofacial complex can result in the following injuries to the permanent dentition, based on the International Association of Dental Traumatology (IADT) guidelines: (1) infraction; (2) enamel fracture; (3) enamel-dentin fracture; (4) enamel-dentin-pulp fracture; (5) crown-root fracture without pulp exposure, (6) crown-root fracture with pulp exposure; (7) root fracture; (8) alveolar fracture; (9) concussion; (10) subluxation; (11) extrusive luxation; (12) lateral luxation; (13) intrusive luxation; and (14) avulsion.[13,58] (These guidelines can be reviewed at www.dentaltraumaguide.org.) The IADT guidelines for assessment of permanent teeth at initial visit recommend exposing several projections and angulations, with additional radiographs indicated when foreign objects become embedded in soft tissue. However, the recommendations suggest that CBCT may be beneficial when used to assess patients after dental traumatic injuries, especially in cases of lateral luxation, root fracture, complications, and monitor healing. The heterogeneity of dental traumatic injuries and difficulty in assessing affected teeth using intraoral radiography, especially when in the plane of PA radiographs, is evident from the guideline recommendations.[58]

Intra-alveolar root fractures generally affect the permanent dentition of males and are relatively uncommon, accounting for 0.5% to 7% of dental impact injuries.[19,54,84,154] Root-fractured teeth present diagnostic challenges, and the limitations of planar radiography have been well documented in the dental literature.[50,58,115,159] A systematic retrospective study showed that maxillary central (68%) and lateral (27%) incisors were primarily affected, with only limited occurrence in mandibular incisors (5%). This retrospective study concluded that CBCT allowed for improved treatment planning and showed the true nature of the injury compared with PA imaging alone.[228] At least seven systematic laboratory studies and one systematic in vivo animal study reported significantly improved accuracy for the detection of root fractures when CBCT was compared with PA radiography.[95,96,101,135,155,225] In a systematic clinical study, Bornstein et al.[33] examined 38 patients with 44 permanent teeth that sustained intra-alveolar fractures. In the study sample reported, 68.2% of teeth had oblique fractures that extended to the cervical third of the root, contradicting the findings of previous studies conducted with PA imaging alone. CBCT imaging offered improved visualization of the

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location and angulation of root fractures compared to PA and occlusal intraoral radiographs.[33]

Injuries that include fractures and comminution injures to the supporting structures or are difficult to visualize with intraoral imaging because of compression of overlying tissues, may be evident on CBCT scans. Several of these studies suggest that lower resolution scans using voxel sizes in excess of 0.3 mm may not improve radiographic assessments.[96,225] A study by Wang et al.[223] showed that the sensitivity and specificity of PA radiography for root fractures were 26.3% and 100%, respectively; the findings for CBCT were 89.5% and 97.5%, respectively. CBCT images of root-filled teeth with fractures showed lower sensitivity and unchanged specificity, whereas 2D images showed the same sensitivity and specificity.[223] CBCT allows for the management of traumatic injuries in which a root fracture or alveolar fracture is suspected by providing undistorted multiplanar views of the dentition and supporting bone without the superimposition of anatomic structures.[47,127,187] CBCT image volumes provide superior sensitivity in detecting intra-alveolar root fractures compared with multiple PA radiographs; this allows for the detection of dental and alveolar displacements, including damage to other perioral structures, such as the maxillary sinus and nasal floor.[105] The presence of root canal fillings and posts affected the specificity of the findings as a result of artifact generation.[96,135] Outcome measurements of a region of interest can be compared over time with greater geometric accuracy using CBCT.[94]

Injury to the soft tissues, such as the lips, cheek and tongue, by embedded debris and tooth fragments, usually require additional radiographic images when relying on intraoral radiography. CBCT scans will aid in wound management by locating the precise size and shape of the foreign body, possibly reducing the scope of surgical interventions.[195]

The healing of root fractures is influenced by many factors, most prominently the stage of root development, with immature roots showing better healing than mature roots.[73] Other factors that influence healing are the extent of dislocation and repositioning, the type of splinting, the use of antibiotics, and the location of the fracture on the root. The long-term survival of teeth with intra-alveolar root fractures was evaluated in a systematic review by Andreasen et al.[17] This study showed that the type of

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healing (e.g., hard tissue fusion, PDL interposition with and without bone) and the location of the fracture on the root had the most influence on tooth loss. Yearly clinical and intraoral radiographic follow-up examinations are recommended by the IADT for some injuries for up to 5 years posttrauma.[58] Further evidence-based comparisons of outcomes for traumatic dental injuries after treatment interventions were addressed by the IADT with the development of a Core Outcome Set for children and adults.

Limited FOV CBCT should be considered when placement of individual PA radiographs will adversely affect patient management or result in retakes; PA radiographs will produce a higher radiation dose for assessment of the region of interest[105]; intra-alveolar fracture of the root or supporting structures is suspected and sufficient information cannot be obtained with conventional radiography; or foreign bodies are present in the lip, cheek, or tongue.[164] The decision to use CBCT imaging for assessment of traumatic injuries should be based on the diagnostic yield expected and in accordance with the "as low as reasonably achievable" (ALARA) principle. CBCT scan volumes that use the most appropriate detector size and shape, beam projection geometry, and beam collimation should be selected to produce high-resolution images and reduce x-radiation exposure whenever possible.[188] In all cases, it should be recognized that children and young adults are more susceptible to the effects of radiation than adults, and CBCT studies should answer specific clinical questions that cannot be answered by lower dose PA and panoramic imaging techniques.[97] In compliance with the Image Gently Alliance recommendations for pediatric imaging, always select x-rays when essential for diagnosis and treatment; use the fastest image receptor available, use CBCT only when necessary, collimate beam to area of interest, always use thyroid shield, and reduce exposure time and technique factors, such as mA.[227] New technologies that allow for comparison of matched CBCT images in a serial fashion at a reduced dose have been tested. This technology promises a dose reduction of 10 to 40 times by using the initial scan as prior knowledge and adaptive prior image constrained compressed sensing (APICCS) algorithms to greatly reduce the number of projections and x-ray tube current levels required (Fig. 2.28).

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FIG. 2.28 **A,** Horizontal root fractures resulting from trauma were evident in this 22-year-old male patient, who was referred with a contemporaneous film–based PA radiograph **(B)** for evaluation and possible treatment 9 months after trauma to his maxillary lateral and central incisors. Since the trauma, the teeth had been stabilized with a ribbon-type splint on the palatal surface; they were of normal color and responded normally to all sensibility tests. There was slight mobility of the traumatized teeth. **C,** The true nature of the root fractures are shown in the 3D segmented reformation and the corrected cross-sectional views of the **(D)** maxillary right lateral and **(E)** central incisors, the **(F)** maxillary left central and **(G)** lateral incisors, and the 3D surface-rendered reformation **(H).** Temporal reevaluation at 7 years showed minimal changes when compared to the initial presentation *(left to right in each group)*. Task-specific exposure parameters allowed each successive CBCT image volume to be exposed with lower mA, resulting in a 20% radiation dose reduction.

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A) Close-view of mouth shows stained teeth, inflamed lips, and wound on the chin.

B) Radiograph shows injured teeth.

C) 3D image shows stained teeth with totally fractured roots and apex.

D) Initial CBCT shows the side view of one of the upper tooth on the right that shows radiolucent root, followed by re-evaluation of tooth after 7-years.

E) Initial CBCT shows side view of the central incisors, followed by re-evaluation of tooth after 7-years.

F) Initial CBCT shows fractured root, followed by re-evaluation of tooth after 7-years.

G) Initial CBCT shows lateral incisors, followed by re-evaluation of tooth after 7-years.

H) 3D image shows reformed teeth and fractures in root.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

Internal and external root resorption

As described in Chapter 18, root resorption results in the loss of dentin, cementum, or bone by the action of clastic cells.[22] In the primary dentition, root resorption is caused by normal physiologic processes, except when resorption is premature; in the permanent dentition, root resorption is caused by inflammatory processes.[46,66,165] Root resorption can be classified according to location as either internal, appearing on the wall of the root canal, and external, affecting the outer surface of the root. Both internal and external root resorption have subtypes that show specific radiographic characteristics.[208]

The successful management of root resorption in the adult dentition depends on clinical and radiographic examination leading to early detection and accurate diagnosis.[166] Unfortunately, teeth affected by root resorption have a poor prognosis if the causative lesion is not treated.[163]

2D radiographic projections suffer from inherent limitations that mask lesions: superimposition of 3D structures,[27] geometric distortion,[215] and

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misrepresentation.[127] Although parallax intraoral imaging techniques can be helpful in localizing root resorption[15] by observing lesion shift on changing angulation, only CBCT assessments can provide the true size and position of all resorptive defects in the region of interest.[46,163] Intraoral radiography is not reliable when lesions are small or located on the labial or lingual/palatal surfaces.[15] In ex vivo studies, CBCT showed improved accuracy in the detection of simulated internal and external resorptive lesions of all sizes.[107] Intraoral imaging produced false-negative results in 51.9% of cases studied and false-positive results in 15.3%.[143]

The use of CBCT in the evaluation of root resorption eliminates superimposition and compression of 3D features, is accurate in all multiplanar views, and can reliably represent the anatomy of three-dimensional structures. Patel et al.[163] compared the sensitivity and specificity of PA imaging with CBCT scans using the receiver operating characteristic (ROC) analysis, a standard measure of diagnostic performance. PA imaging showed satisfactory accuracy (Az 0.78), whereas CBCT showed perfect results (Az 1.00).[163]

Internal root resorption (IRR) is usually idiopathic and can be subdivided into either internal inflammatory or internal replacement resorption. IRR is a relatively rare occurrence that is usually detected on routine diagnostic radiographs.[122,162] It is characterized by internal structural changes in the tooth that can appear in any location and can exhibit varied features, such as smooth or irregular borders and radiodensity of uniform or mixed attenuation.[99] IRR is usually asymptomatic, associated with pulpal necrosis coronal to the resorptive lesion and vital or partially vital pulps where active.[163] When viewed with intraoral radiographs, IRR can easily be confused with external cervical resorption (ECR) owing to the similarity of the two lesions.[92] Parallax intraoral radiographs may aid identification because the lesion will maintain its position relative to the root canal. However, assessment may be affected by radiographic superimposition and by teeth with complex anatomy. CBCT is helpful for diagnosing the location and exact size of IRR. In a study by Estrela et al.,[66] 48 PA radiographs and CBCT scans were exposed on 40 individuals.[66] IRR was detected in 68.8% of PA radiographs, whereas CBCT scans showed 100% of the lesions.

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Conventional radiographs were able to detect only lesions between 1 and 4 mm in 52.1% of the images, whereas CBCT was able to show 95.8% of the lesions. This finding agreed with other studies that demonstrated the value of tomographic analysis.[46,125] In a study by Kim et al.,[112] the extent and location of the IRR was accurately reproduced with the fabrication of a rapid prototyping tooth model. Although relatively few systematic studies on artificially induced IRR have been reported because of the difficulty in creating such defects, Kamburoglu and Kursun[106] concluded that high-resolution CBCT imaging performed better than low-resolution CBCT imaging in detecting simulated small internal resorptive lesions.

External root resorption (ERR) can be divided into four subtypes: external surface resorption, external inflammatory resorption, external replacement resorption, and external cervical resorption (ECR). ERR may progress rapidly, and early treatment is recommended.[61]

The recommended timing of radiographic examinations using CBCT has not been established with a strong evidence grade. Many ERR cases involve young patients in whom the radiation dose is a critical factor and multiple scans would be difficult to justify. To minimize radiation dose to children the following guidelines have been suggested: Select radiographs based on a patient's individual needs, not as a routine; use the fastest image receptor possible; collimate the x-ray beam to expose only the area of interest; use thyroid collars; reduce the exposure as appropriate for the size of the child; use only when necessary, never as a screening tool.[227]

External surface resorption depends on the nature of the stimulus and can vary from small, concave lesions to extensive destruction of the root.[14] Commonly caused by orthodontic treatment, proximity to other teeth, or cysts and tumors, radiographic imaging with CBCT allows for accurate multiplanar mapping of the lesion. These excavations normally show small irregularities on the root surface with intact PDL and lamina dura. The appearance of the ERR may help indicate the nature of the lesion, where benign lesions tend to displace teeth and where more aggressive lesions, such as malignant tumors, tend to cause extensive root resorption, showing either blunting of the root apex or spiked/knife-edge destruction (Fig. 2.29).[165]

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FIG. 2.29 **A,** External cervical resorption was evaluated in this patient after PA radiographic image, and **(B)** visual examination showed pathognomonic signs of this lesion *(yellow arrow)*. A CBCT image was exposed to determine the true extent of resorption and restorability. **C,** The lesion showed a perforative defect at the CEJ on the facial *(yellow arrow)* and **(D)** palatal aspects *(yellow arrow)*. **D,** The presence of an intact layer of mineralized dentin, the "pericanalar resorption-resistant sheet" *(blue arrow)*, is a hallmark of this condition.

Set of four radiographs are marked A through D as follows:

A) Radiograph shows dark patch at the root of tooth on the right and radiolucent roots on the left.

B) Close-up view shows receding gum line below tooth.

C) Tooth shows fracture at one side of the tooth on the left between crown and root.

D) Tooth shows less-density pulp along with a fracture on the right.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA].)

External inflammatory resorption resulting from pulpal necrosis or traumatic injuries is radiographically characterized by concave and occasionally patchy excavations of low density that affect the root surface

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and the supporting bone. Lesion borders are irregular, show loss of lamina dura, and are often associated with the apical region due to the proximity of the apical terminus.

External replacement resorption is caused by damage to the periodontium, which may result from dental trauma or orthodontic treatment.[74] The teeth are immobile, have a distinctive tone on percussion, and show disappearance of the normal periodontal membrane—features consistent with a direct union with alveolar bone.

External cervical resorption is a result of damage to the cementum layer of the root by clastic activity. Further destruction of cementum and then dentin[93] is dependent on stimulating factors that include microorganisms from the periodontium.[82] ECR is a complex and aggressive process and usually begins at the cervical region and may result in a pink discoloration of the overlying enamel. Patients affected by ECR are usually asymptomatic because the pericanal resorption-resistant/retarding sheet protects the pulp until the later stages.[209] Orthodontic treatment, trauma, parafunctional habits, and malocclusion account for most of these lesions, with a combination of factors resulting in increased occurance.[167] Intraoral radiography shows significant variability, with areas of low density affecting the dentin extending coronally and/or apically with sometimes greater destruction distant from the origin on the buccal or lingual/palatal aspect of the root, confounding 2D radiographic assessment. The radiographic appearance of these lesions is often mixed owing to the level of fibro-osseous and granulomatous tissue present. External cervical resorption is located on the external surface of the root, showing an intact root canal wall that can be traced through the lesion, whereas IRR is continuous with the canal wall.

External cervical resorption is always associated with bony resorption, making comparability of laboratory studies problematic because the lesions lack the changes in the periodontal membrane and associated bony changes that would improve visualization. The early stages of ERR are difficult to view with conventional radiography, and lesions less than 0.6 mm in diameter and 0.3 mm in depth could not be detected. Medium-sized lesions were visible in 6 of 13 cases, with improved visualization for proximal lesions without regard to the root third being examined.[15] Patel et al. showed that the accuracy of parallax and single parallel PA radiographs were similar,

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and that more teeth were considered unrestorable when assessed with CBCT (78.7%) than with PA imaging (49.3%).[158] This study further highlights the benefits of using CBCT to assess ECR.[158] Vaz de Souza et al. showed that CBCT was significantly better than PA radiography at diagnosing ECR according to the Heithersay classification.[216]

ERR is difficult to detect if the lesion is confined to the buccal, palatal, or lingual surfaces of the root.[30,83] Liedke et al.[125] conducted systematic diagnostic performance tests and showed similar sensitivity and specificity among the different voxel sizes studied (0.4, 0.3, and 0.2 mm); however, the likelihood ratio showed better probability of correct identification of ERR with either 0.3 or 0.2 mm scans. These researchers suggested the use of a 0.3 mm voxel size protocol rather than a 0.2 mm one, to reduce scanning time and the resulting dose.[125] Although voxel size is an important consideration, the SNR of different detectors, the radiation dose, viewing conditions, and the processing algorithms also affect detection probability.

Even though many in vitro studies have been performed on the ability of CBCT to detect root resorption, additional evaluations that use in vivo methodology will add to the cumulative knowledge.

Endodontic treatment outcomes assessment

The reduction of inflammation and healing through regeneration or repair are the ideal outcomes for endodontic treatment.[148] However, endodontic treatment outcome predictors using PA radiographs and CBCT imaging have been shown to vary and are influenced by factors such as patient inclusion and exclusion criteria.[124] Historically, PA radiographs and physical examinations were used to determine the success of endodontic treatment, and the absence of posttreatment periradicular radiolucencies and symptoms was considered by many to be the criterion for success. However, these planar imaging-based studies have resulted in an overestimation of successful outcomes, compared to CBCT assessments,[230] because AP confined within the cancellous bone or lesions covered by a thick cortex may be undetectable with conventional radiographic assessments.[26] Additional discrepancies between PA radiography and CBCT have resulted from geometric distortion, limiting comparisons of temporal evaluations, even with careful attention to

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paralleling technique factors.[133]

A clinical study comparing the sensitivity, specificity, predictive values, and accuracy of PA and panoramic radiography and CBCT imaging in 888 consecutive patients showed that the prevalence of AP in root-treated teeth was 17.6%, 35.3%, and 63.3%, respectively. Conventional radiography showed increased accuracy when the larger lesions were assessed.[67]

Using histologic examination as the gold standard, a systematic review and meta-analysis of the literature by Dutra et al.[121] compared different radiographic modalities in the binary assessment of AP versus no lesion in humans—a very important measure in outcome studies. Significantly, only nine studies were ultimately selected for qualitative synthesis and only six for meta-analysis. All included studies were limited by using only artificially induced apical lesions, possible bias, variation in bone lesion sizes, poor or no inter-and intraobserver agreement, and except for one study, did not evaluate the maxilla, where more tissue compression occurred. This study showed the accuracy values of 0.72 for digital PA radiographs, 0.73 for film-based PA radiographs, and 0.96 for CBCT.[121]

Outcome predictors identified with PA radiographs and CBCT scans may be different depending on the research performed. Liang et al.[124] retrospectively evaluated 115 endodontically treated teeth with vital pulps 2 years after treatment. The authors noted that the recall rate of 36% was comparable to that in other studies, but the result may have been affected because patients with symptomatic or already extracted teeth may not have responded. This lost-to-follow-up cohort significantly reduced the level of evidence.[75] PA radiography identified PA lesions in only 12.6% of teeth compared to CBCT images, which identified 25.9% of teeth with PA lesions. In multivariate logistic regression analysis, the extent of root fillings and density were outcome predictors when using PA imaging, whereas density of root fillings and quality of the coronal restoration were outcome predictors when using CBCT scans.

The predictive value and diagnostic accuracy of radiologic assessments are critical to the practice of dentistry, and the diagnostic value of radiographs depends on the radiograph's ability to show the histology of AP. De Paula-Silva et al.[53] evaluated the periapex of 83 root-treated and untreated dogs' teeth using PA radiography, CBCT scans, and histopathologic analysis. PA

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radiography detected AP in 71% of roots; CBCT scans detected AP in 84%; and histologic analysis detected AP in 93%. These findings, corroborated by other studies,[38,87,178] emphasized the low negative predictive value (NPV) of PA radiography at 0.25, showing that when the PA tissues had a normal appearance, 75% actually had AP. CBCT scans resulted in an NPV almost two times higher than that for PA radiography; however, CBCT scans were not able to detect some AP that was confined to the apical foramen or had little volumetric bone loss. The positive predictive value was the same for PA radiography and CBCT scans compared with histologic examination, but true positive and true negative diagnosis of AP using CBCT scans occurred in 92% of cases.

Continuing development of sophisticated postprocessing software now allows for modeling of AP and anatomic structures in 3D. Using either manual or semiautomatic segmentation, the resulting virtual models allow for alignment and analysis of temporal changes, with some limitations. Bony changes can be depicted in quantitative and qualitative datasets, showing bone density changes such as repair or osteolysis. One of these programs, ITK-SNAP, an open-source software originally developed for MRI assessments, allows for construction of virtual 3D models from DICOM datasets, showing volumetric measurements in voxels and cubic millimeters. Additional tools allow for calculation of the mean intensity of the PA rarifying lesion(s) of interest. The resulting scan data can then be exported in STerioLithography format (STL) and shown in 3DMeshMetric software (Figs. 2.30 and 2.31).

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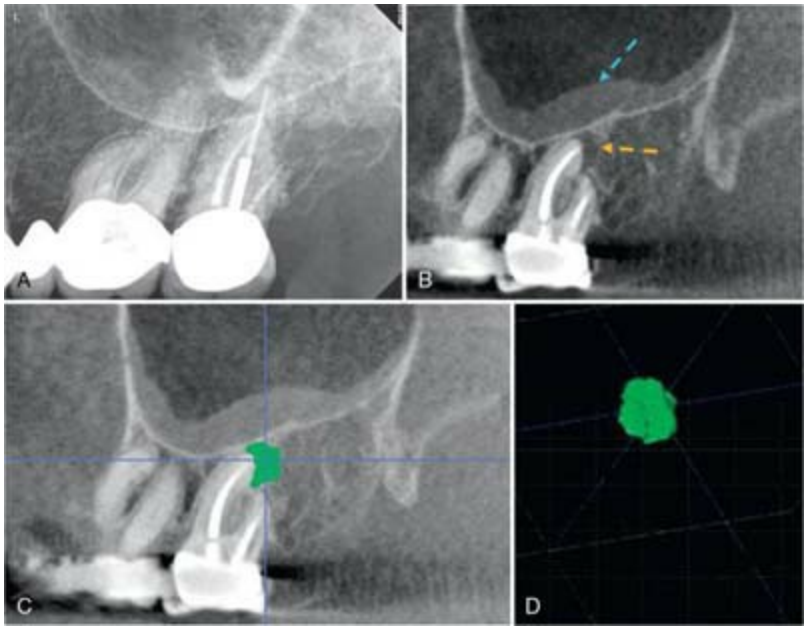

FIG. 2.30 The problem of superimposition of unrelated structures onto the features of interest is reduced when tomographic slices are used instead of images in which an entire volume of data is compressed into a planar image. **A,** This PA radiographic image of the maxillary left second molar shows no radiographic indicators of pathosis. A contemporaneously exposed CBCT image (**B,** corrected sagittal view) shows a 4.3 × 1.9 mm, well-defined unilocular, noncorticated area of low attenuation centered over the apex of the mesiobuccal root, consistent with a periradicular periodontitis *(yellow arrow).* There is a moderate mucositis in the region of the maxillary sinus adjacent to this tooth *(blue arrow).* Experimental semiautomated segmentation of this image using active contour methods (ITK-SNAP) allowed for measurement of the true volume of the lesion **(C)** and for future temporal comparisons based on volumetric measurements **(D).** This lesion measured 85,112 voxels and 38.1044 cubic mm.

A) CBCT image shows upper teeth. One tooth shows three root canals.

B) Tooth with two root canals and radiolucent region in-between. A lesion lies at the tip of long root canal.

C) A green-colored structure is shown in place of the lesion.

D) 3D image of lesion is depicted as a green dot on a black background.

Source: (Data acquired and reformatted at 0.076 mm voxel size using a CS 9000 3D unit [Carestream Dental, Atlanta, GA]; volumetric segmentation and measurement using ITK-SNAP [Radiology Department, School of Medicine, University of Pennsylvania].)

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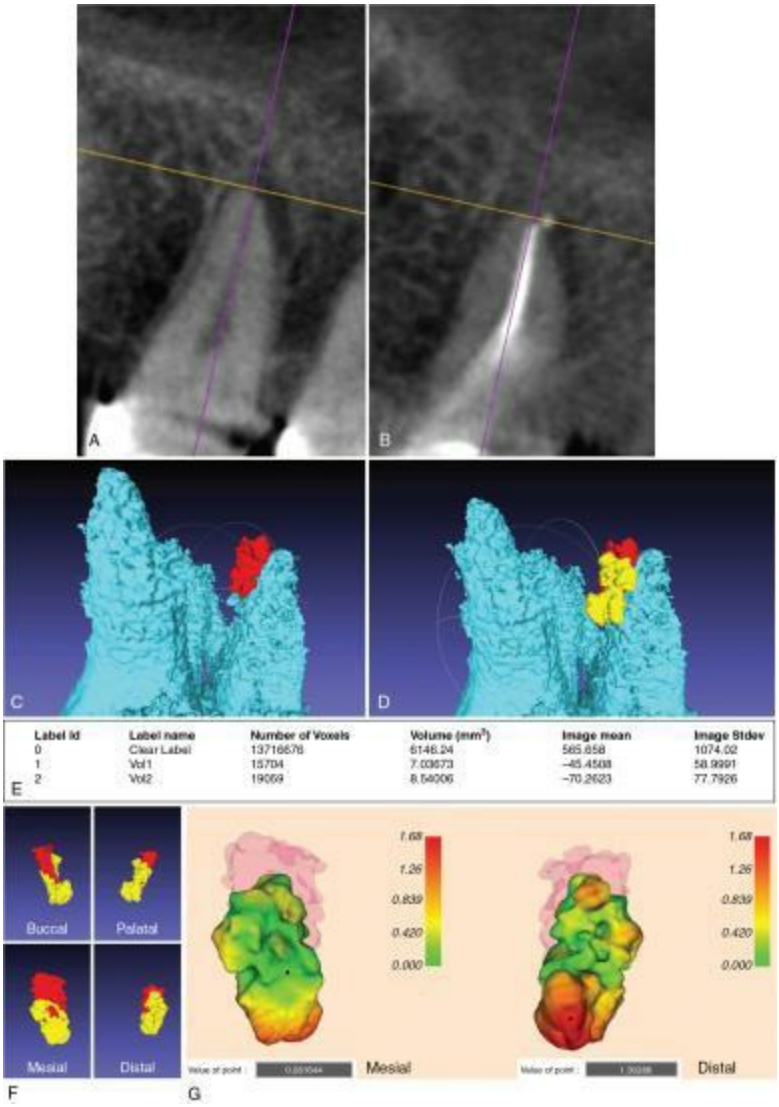

FIG. 2.31 Radiographic interval change is shown by comparing two volumetric datasets exposed at different times. **A,** Initial CBCT corrected sagittal reformation of maxillary right second molar, distobuccal root and **(B)** corrected sagittal reformation of same root at 13-month checkup. **C** and **D,** ITK-SNAP segmented sagittal reformation of same root. Alignment and overlap of the 3D models: tooth-blue, initial lesion is red, lesion at 13-month check-up is yellow, respectively. **E,** Voxel counting, volumetric measurement (mm) and mean intensity of the periapical lesion. **F,** Models were exported to the.stl format and then viewed with MeshLab software. **G,** 3D Mesh Metric Qualitative (color-coded map) and quantitative (point value) assessments of the lesions before (translucent), and after (colored). The color map shows changes with green color in the mesial region (0.28 mm) and changes with dark red color in the distal region (1.30 mm).

A) Radiograph shows tooth with less density under root and radiolucent tissue in between.

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B) Image of the same tooth is shown after 13-month checkup. The radiolucent region appears radiopaque.

C) 3D model shows tooth model with a lesion toward the top right. The size of lesion is big.

D) 3D model shows tooth after 13-month checkup. The size of lesion is reduced at a greater extent.

E) Four lesions after 13-month checkup are labeled buccal, palatal, mesial, and distal.

F) Two color-code map represent mesial and distal views of tooth.

Source: (Courtesy Dr. Ane Poly.)

Risks and benefits for patients who are medically complex and subject to possible increased morbidity from AP, such as patients with an altered immune system (i.e., chemotherapy or anti-HIV protocols or prosthetic joints and/or infective endocarditis), should be considered in the decision about whether to expose a CBCT scan. The American Academy of Periodontology has published a position paper stating that periodontal disease may contribute to adverse systemic health conditions.[186] The scientific basis for the relationship of AP and adverse systemic health conditions has not been established[43]; however, new associations between AP and systemic health should be predicated on research that uses CBCT in the detection of endodontic disease.[49]

The determination of successful outcomes is nuanced and complex. New AAE-sponsored initiatives to study outcomes is underway and will ultimately result in an agreed standardized set of criteria, called a “core outcomes set,” facilitating data synthesis and possibly resulting in more transparent and complete reporting.

Dental implant case planning

Successful endosseous implant site assessment requires the development of a prosthetically driven approach,[2] with special emphasis on the evaluation of bone volume, the osseous topography, and the location of anatomic structures in relation to the positioning of the implant. The AAOMR has published advisory recommendations suggesting all radiographic studies should

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interface with the dental and medical histories, clinical examinations, and treatment planning. Panoramic radiography, which may be supplemented by PA radiography, should be used for initial imaging assessments. Cross-sectional imaging, including CBCT, should not be used as an initial imaging examination. The AAOMR affirms the need for cross-sectional imaging in the preoperative diagnostic phase, recommending CBCT because it provides the highest diagnostic yield at an acceptable radiation dose risk. CBCT should be used at the smallest FOV necessary, with optimized technique factors, to minimize radiation dose in accordance with the ALARA principle.[213]

CBCT allows for precise planning and delivery of implants that can reproduce the anatomy with submillimetric accuracy, leading to improved outcomes.[201] The use of CBCT to assess linear measurements, proximity to vital anatomic structures, mapping of the alveolar ridge topography, and fabrication of surgical guides is supported by the dental literature. The use of CBCT to gauge bone density, provide intraoperative surgical navigation, and assess implant integration is generally considered an area that requires further research.[25]

Virtual implant planning using CBCT data allows clinicians to visualize the result before the commencement of treatment, facilitating the virtual investigation of multiple treatment scenarios until the best plan is attained. The evaluation of bone dimensions, bone quality, the long axis of the alveolar bone, internal anatomy, and jaw boundaries; the detection of pathologic features; and the transfer of radiographic information are the main imaging goals. Use of static, CT-generated guide stents and dynamic navigation systems provide improved accuracy when compared with freehand methods.[29]

Pathoses of the jaws, such as retained root tips, inflammatory lesions, cysts, and tumors, in addition to extraoral structures, such as the sinuses and temporomandibular joints (TMJs), must also be assessed.[97] CBCT imaging should be considered to evaluate implant sites in the region of teeth with a high likelihood of periradicular pathosis.[239]

3D-guided endodontics

3D-guided surgical and nonsurgical endodontic treatment is an emerging

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application using guides manufactured with the aid of CBCT images and intraoral scanning of the area of interest. Although studies with high level of evidence is lacking in this area, several in vitro studies and case reports have been published.[5,48,118,132]

The technology is currently being utilized in endodontic treatment of teeth with calcified canals and teeth that have anatomical variations of the root canal system. A template is created by superimposition of CBCT images on intraoral scanned images with sleeves placed in the images for creating a 3D printed guide with a directed sleeve. Most often implant treatment planning software is used for the creation of the guide (Fig. 2.32).[48,118,132] Recent study evaluating access on calcified teeth showed a higher detection of canal (91.7% vs. 41.7%) using guided approach. There was significantly lower amount of tooth substance loss using the guided access (49.9 mm vs. 9.8 mm).[48]

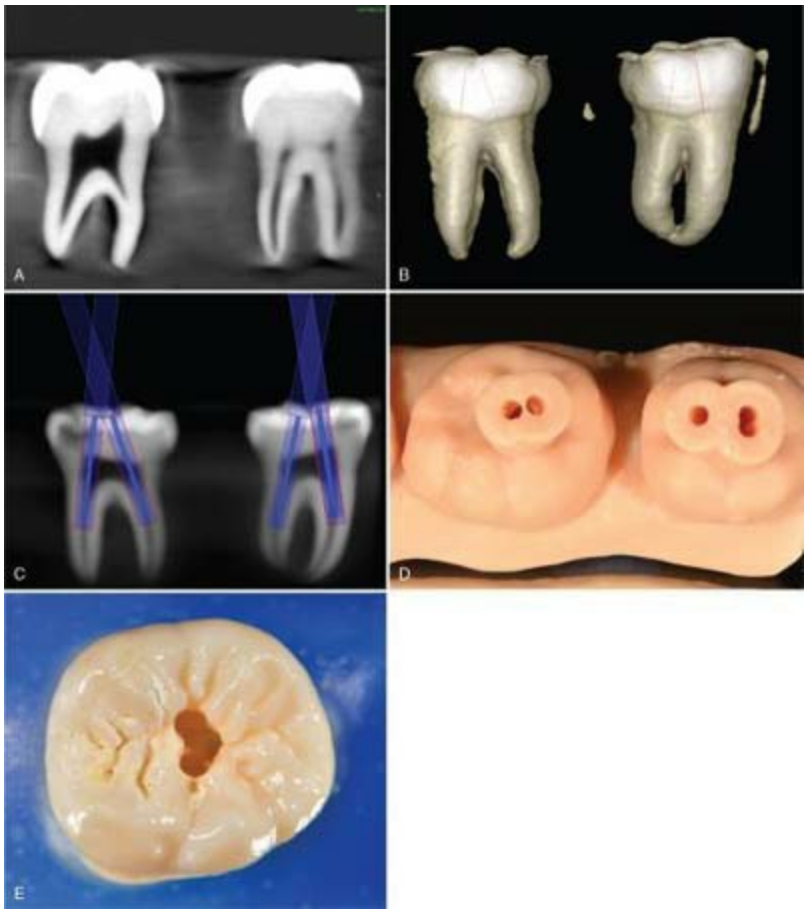

FIG. 2.32 Guided access flow chart. **A,** 2D rendering from the CBCT

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of a block of 2 molars. **B,** Volumetric rendering of the teeth. **C,** Lateral view of the teeth with virtual files in position. **D,** Printed guide. **E,** Representative occlusal view of an accessed tooth.

Set of three radiographs A to C and two photographs D and R are given as follows:

A) The first molar shows radiolucent pulp. The second molar shows comparatively denser pulp.

B) Two 3D image show two molars. The second molar shows fused root canals at the bottom.

C) Radiograph shows X rays on two molars.

D) A model shows two molars each with two holes.

E) Tooth with a cavity at the center.

Source: (Courtesy Dr. Zachary Evans and Dr. Bryce Szczepanik, MUSC, Charleston, SC.)

Endodontic microsurgery aims to create conservative osteotomy utilizing magnification and microsurgical instruments. Various studies have shown improved prognosis with the microsurgical procedures when compared to traditional endodontic surgery.[210] Surgical treatment planning with CBCT images have aided clinicians in identifying anatomical structures in the surgical area and increasing precision of the procedure.[119] Surgical endodontics with 3D-guided templates has further helped in making microsurgical procedures more precise.[3,5,71] A recent study comparing "freehand" CBCT-aided surgery to surgery using template guides showed a deviation of 1.743 mm when using guides from the target location identified on preoperative CBCT. Freehand surgery showed a deviation of 2.638 mm.[3] Use of prefabricated grids placed while acquiring CBCT images were used as guides for creating osteotomy during endodontic surgery in a recent study. Deviation from the target point was significantly lower when compared to nonguided drilling (Fig. 2.33).[71]

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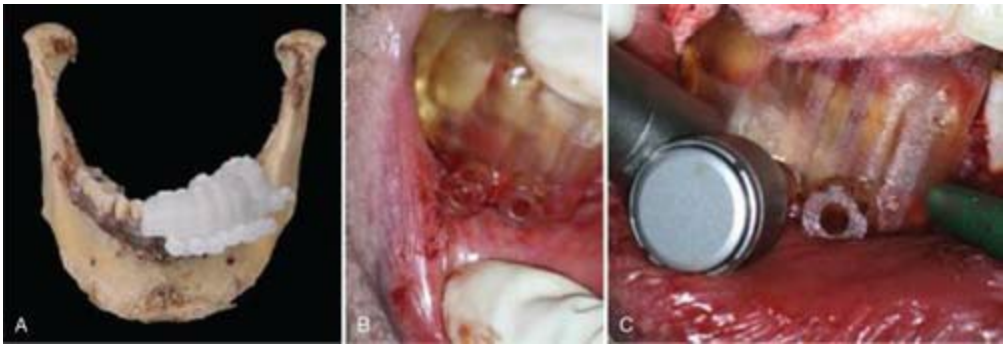

FIG. 2.33 Guided endodontic surgery. **A,** 3D printed guide. **B,** Guide placed in position prior to endodontic surgery. **C,** Surgical access with printed guide in position.

A) Lower jaw shows mandible quadrant with white deposition.

B) Close-up view shows a guide at the infected site before the surgery.

C) Close-up view shows mandible quadrant undergoing surgery.

Source: (Courtesy Dr. Ackerman and Dr. Jalali, Texas A&M College of Dentistry, Dallas, TX.)

Image perception and viewing environment

Medical image perception is an important area of knowledge, and ongoing research relies on an understanding of perceptual issues, such as psychological factors, dwell time, visual search physiology, search tactics, appreciation for the reading environment, and fatigue factors, to increase search satisfaction. Understanding these issues may improve the ability to interpret and report on dental radiographic findings.[185]

The increasing use of digital radiography in the dental environment has led to a sea change in workflow and the necessity for new ways to view and document radiographic images. Simple to accomplish, but important to improve, are the viewing conditions for softcopy interpretation, including moderately reduced ambient lighting, ranging from 25 to 40 lux.[36]

The future of cone-beam computed tomography

The first decade of the 21st century saw the development of a wide range of

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CBCT applications, especially in dentistry. Lower radiation dose, higher spatial resolution, smaller FOV, and relatively lower cost may contribute to CBCT becoming the standard of care in 3D dentomaxillofacial imaging in selected cases. CBCT systems are increasingly being used in medical applications, such as operating rooms, emergency departments, intensive care units, and private otolaryngology offices. Operating room–based C-arm systems have been in use for a number of years, with applications in interventional angiography, cancer surgery, vascular surgery, orthopedic surgery, neurosurgery, and radiotherapy.[69] Applications in otolaryngology and mammography are common, and imaging of extremities in weight-bearing scenarios is under development. Many of these applications rely on systems that use task-specific protocols that benefit from CBCT's 2D FPDs, which allow for a single rotation of the source to generate a study of the region of interest, as opposed to complex MDCTs that use redundant imaging via multiple slice acquisitions to generate a 3D volume.[196]

The introduction of new, high-performance, flat-panel detectors and software algorithms centering on improving the noise-power spectrum and noise-equivalent quanta will continue to increase the utility of CBCT systems in the future. Areas of research include (1) image perception and image quality assessment, to better understand how physicians and dentists analyze radiographic images and thereby improve diagnostic decision making[202]; (2) iterative reconstruction that uses sophisticated algorithms to reduce artifacts; (3) known-component reconstructions that use a model-based 3D image reconstruction and iterative software to reduce image artifacts in the presence of metallic devices such as screws and implants; (4) image registration to align tissues for image-guided surgery and outcomes assessment;[141] (5) image-guided procedures that provide instantaneous surgical navigation; (6) segmentation to allow discrimination between normal and diseased tissues and permit volumetric measurements (see Figs. 2.30 and 2.31); and (7) use of metallic artifact reduction algorithms that help partly remove distracting signals and artifacts from a CBCT study. Note, however, that there is potential for loss of diagnostic information as well, especially in situations where high spatial resolution is required, such as detection of vertical root fractures in roots with posts.

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Magnetic resonance imaging

MRI has been explored as a potentially useful imaging modality in dentistry, and in particular endodontics. Advantages include nonexposure to ionizing radiation; capability to acquire images in any desired plane without having to reformat data; capability to image edema, effusion, and inflammation in the adjacent bone; spread of infection; better soft-tissue delineation; and assessment of root structure and variations in canal morphology and/or number. Disadvantages include high cost of a scanner, longer acquisition times, interpretation challenges due to differences in appearance of hard and soft tissue as compared to CBCT or conventional radiographs, potential for motion artifacts, limited resolution, lack of easy access, choice of appropriate protocol based on scanner parameters, and generation of artifacts unlike those seen on x-ray–based images. All of these challenges need to be addressed in order for the imaging modality to be widely accepted and used in endodontics. Various pulse sequences have been developed for optimizing image quality for endodontic diagnostic purposes, but a magnetic field strength that is sufficiently high to improve resolution and further fine-tuning of task-specific regional receiver coils are required.[20,57]

Conclusions

Digital radiography has several advantages and has become an indispensable diagnostic tool for many dentists in daily practice. Once the digital image appears on the monitor, the dental x-ray software allows image enhancement, which should be used with caution and be based on the diagnostic task. Inappropriate use of enhancement has been shown to adversely affect diagnosis.[142] If digital radiographs are exported using various software packages created for graphic design and image manipulation, digital information can be altered, added, or removed. The DICOM standard has been accepted as the universal standard for image transmission and archiving, so that each image can be transmitted and stored without the use of proprietary software that would seriously limit its distribution. DICOM ensures that all images are readable in any viewing software without loss of fidelity or diagnostic information. Image enhancement features of digital radiography allow mishandling of images, leading to potential abuse.

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Published studies illustrate the potential for fraudulent use of digital radiography.[42]

There is a dearth of studies related to the diagnostic performance of the different sensor types currently available on the market. Slight to moderate differences in spatial resolution capabilities exist. With rapid advancement in sensor technology and frequent software upgrades, selection of one system over another for a specific diagnostic task may appear challenging. A review of the most commonly used solid-state sensors notes that most systems perform comparably with intraoral film and also allow for postprocessing of images, which is not possible with film-based images. Other factors that assume significance in this context are the availability of technical and customer support, frequency of both hardware and software upgrades, dimensions of the sensor and its active area, number of sensors needed in a practice (and thus cost issues), the detector quantum efficiency (DQE), and conformance to the DICOM standard for seamless integration with other systems. CCD/CMOS sensors appear to offer the best contrast and spatial resolution, in addition to facilitating instantaneous image capture, and therefore are recommended for endodontic applications. Careful and appropriate image processing further helps tease out the signal of interest. In an enterprise-wide setting or in larger private practices that have multiple specialty areas, PSP-based sensors may be more cost effective for large volume imaging (e.g., full mouth series of radiographs). However, at least one or two CCD/CMOS-based systems should be available for faster image acquisition, such as for endodontic purposes and intraoperative procedures.

It is recommended that the literature be constantly reviewed for updates on digital radiography and advanced imaging modalities for specific endodontic applications because hardware and software upgrades continue to make rapid progress. Previous studies have shown that most digital images performed comparably to conventional intraoral film for a variety of diagnostic tasks. Most of these studies were done with earlier generation sensors. The advances in sensor technology have resulted in greatly enhanced image quality, and this trend is expected to continue. Also of interest in the future will be the use of task-based, appropriate image processing parameters that result in a reduced radiation dose and significant enhancement of the diagnostic information. Automation of this process will result in faster and

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more consistent image processing based on the diagnostic task. Such procedures are routinely carried out in medical radiology.

Three-dimensional imaging will continue to be used extensively as sensor characteristics improve and more user-friendly software is introduced. As bit-depth and spatial resolution of images increase, CBCT will continue to be explored for more applications in endodontics. Image interpretation also is important. Occult pathology and incidental findings in adjacent regions can be easily missed or go unrecognized by those who have not received specific training in the interpretation of regional anatomy. Image processing can greatly alter signal characteristics, thus rendering the task fairly challenging. Besides, if other pathoses are discovered, additional imaging may be necessary, including MRI, nuclear medicine studies, or even MDCT, for the evaluation of soft tissues, with and without the use of contrast.

The advent of 3D imaging has provided the endodontist with tools that were not available until now, facilitating interactive image manipulation and enhancement and thus significantly increasing the amount of information that can be gleaned from a volume. Lack of distortion, magnification, artifacts associated with conventional radiography, and the relative low radiation dose compared to medical-grade CT will result in more clinicians adopting such technology to enable accurate diagnoses and treatment planning, in addition to long-term follow-up and the evaluation of healing. Judicious use of CBCT and all other imaging modalities using ionizing radiation is advocated. The AAE/AAOMR position paper provides recommendations for the use of CBCT in endodontics[1] and is presented at the end of this chapter.

When the clinician works with different vendor products, it is important to have a quality assurance program in place. This is not being done currently. Additionally, accreditation of stand-alone imaging laboratories is now a requirement for reimbursement of medical and dental diagnostic procedures from government agencies and some major third-party insurance providers. Several states are considering enforcing this requirement to prevent abuse of CBCT.

Likewise, definitive referral criteria are lacking. Indications, contraindications, and choice of alternate imaging modalities need to be considered before CBCT is used. There is a learning curve to this technology, and appropriate positioning, choice of exposure parameters (and thus the

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effective dose), reconstruction schemes, choice of postprocessing algorithms based on diagnostic task, voxel sizes, and cost must be considered. The literature contains few studies to help us formulate definitive guidelines for the use of CBCT in dentistry.

It is equally important to record the doses associated with each study. Accreditation criteria have been developed by the Intersocietal Accreditation Commission for CT in dentistry that are useful for ensuring the safe use of these units. The lowest possible dose must be imparted to the patient as part of a radiologic examination to minimize stochastic effects that have no known threshold for expression. No dose can be considered a “safe dose.” The benefits of any radiographic study must outweigh the risks. All studies must be interpreted fully because signals from adjacent areas may appear in the volume of interest, including small FOV studies. Retakes can be avoided by adhering to protocol selection based on the task at hand. The ALARA principle must be followed, regardless of the dose values reported by the vendor, to optimize the dose for the specific examination. Use of thyroid collars and lead aprons is recommended in the NCRP guidelines, as long as they do not interfere with image acquisition.

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Joint position statement of the American Association of endodontists and the American Academy of Oral and Maxillofacial Radiology on the use of cone beam computed tomography in endodontics: 2015/2016 update

This statement was prepared by the Special Committee to Revise the Joint AAE/AAOMR Position Statement on Use of CBCT in Endodontics and approved by the AAE Board of Directors and AAOMR Executive Council in 2016.

Introduction

This updated joint position statement of the American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR) is intended to provide scientifically based guidance to clinicians regarding the use of cone-beam computed tomography (CBCT) in endodontic treatment and reflects new developments since the 2010 statement.[1] The guidance in this statement is not intended to substitute for a clinician's independent judgment in light of the conditions and needs of a specific patient.

Endodontic disease adversely affects quality of life and can produce significant morbidity in afflicted patients. Radiography is essential for the successful diagnosis of odontogenic and nonodontogenic pathoses, treatment of the root canal systems of a compromised tooth, biomechanical instrumentation, evaluation of final canal obturation, and assessment of

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healing.

Until recently, radiographic assessments in endodontic treatment were limited to intraoral and panoramic radiography. These radiographic technologies provide two-dimensional representations of three-dimensional anatomic structures. If any element of the geometric configuration is compromised, the image may demonstrate errors.[2] In more complex cases, radiographic projections with different beam angulations can allow parallax localization. However, complex anatomy and surrounding structures can render interpretation of planar images difficult.

The advent of CBCT has made it possible to visualize the dentition, the maxillofacial skeleton, and the relationship of anatomic structures in three dimensions.[3] CBCT, as with any technology, has known limitations, including a possible higher radiation dose to the patient. Other limitations include the potential for artifact generation, high levels of scatter and noise, and variations in dose distribution within a volume of interest.[4]

CBCT should be used only when the patient's history and a clinical examination demonstrate that the benefits to the patient outweigh the potential risks. CBCT should not be used routinely for endodontic diagnosis nor for screening purposes in the absence of clinical signs and symptoms. Clinicians should use CBCT only when the need for imaging cannot be met by lower dose two-dimensional (2D) radiography.

Volume size(s)/field of view

There are numerous CBCT equipment manufacturers, and several models are available. In general, CBCT is categorized into large, medium, and limited-volume units based on the size of their "field of view" (FOV). The size of the FOV describes the scan volume of CBCT machines. That volume determines the extent of anatomy included. It is dependent on the detector size and shape, beam projection geometry, and the ability to collimate the beam. To the extent practical, FOV should only slightly exceed the dimensions of the anatomy of interest.

Generally, the smaller the FOV, the lower the dose associated with the study. Beam collimation limits the radiation exposure to the region of interest and helps ensure that an optimal FOV can be selected based on disease

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presentation. Smaller scan volumes generally produce higher resolution images. Because endodontics relies on detecting small alterations such as disruptions in the periodontal ligament space, optimal resolution should be sought.[5]

The principal limitations of large FOV CBCT imaging are the size of the field irradiated and the reduced resolution compared to intraoral radiographs and limited volume CBCT units with inherent small voxel sizes.[4] The smaller the voxel size, the higher is the spatial resolution. Moreover, the overall scatter generated is reduced due to the limited size of the FOV. Optimization of the exposure protocols keeps doses to a minimum without compromising image quality. If a low-dose protocol can be used for a diagnostic task that requires lower resolution, it should be employed, so long as strong indications to the contrary are absent.

In endodontics, the area of interest is limited and determined prior to imaging. For most endodontic applications, limited FOV CBCT is preferred to medium or large FOV CBCT because there is less radiation dose to the patient, higher spatial resolution, and shorter volumes to be interpreted.

Dose considerations

Selection of the most appropriate imaging protocol for the diagnostic task must be consistent with the “as low as reasonably achievable” (ALARA) principles that every effort should be made to reduce the effective radiation dose to the patient “as low as reasonably achievable.” Because radiation dose for a CBCT study is higher than that for an intraoral radiograph, clinicians must consider overall radiation dose over time. For example, will acquiring a CBCT study now eliminate the need for additional imaging procedures in the future? It is recommended to use the smallest possible FOV, the smallest voxel size, the lowest mA setting (depending on the patient’s size), and the shortest exposure time in conjunction with a pulsed exposure-mode of acquisition.

If extension of pathoses beyond the area surrounding the tooth apices or a multifocal lesion with possible systemic etiology is suspected, and/or a nonendodontic cause for devitalization of the tooth is established clinically, appropriate larger field of view protocols may be employed on a case-by-case

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basis.

There is a special concern with overexposure of children (up to and including 18-year-olds) to radiation, especially with the increased use of computed tomography scans in medicine. The AAE and the AAOMR support the Image Gently Campaign led by the Alliance for Radiation Safety in Pediatric Imaging. The goal of the campaign is "to change practice; to raise awareness of the opportunities to lower radiation dose in the imaging of children." Information on use of CT is available at http://www.imagegently.org/Procedures/ComputedTomography.aspx.

Interpretation

If a clinician has a question regarding image interpretation, it should be referred to an oral and maxillofacial radiologist.[6]

Recommendations

The following recommendations are for limited FOV CBCT scans.

Diagnosis

Endodontic diagnosis is dependent upon thorough evaluation of the patient's chief complaint, history, and clinical and radiographic examination. Preoperative radiographs are an essential part of the diagnostic phase of endodontic therapy. Accurate diagnostic imaging supports the clinical diagnosis.

Recommendation 1: Intraoral radiographs should be considered the imaging modality of choice in the evaluation of the endodontic patient.

Recommendation 2: Limited FOV CBCT should be considered the imaging modality of choice for diagnosis in patients who present with contradictory or nonspecific clinical signs and symptoms associated with untreated or previously endodontically treated teeth.

Rationale

- In some cases, the clinical and planar radiographic examinations are inconclusive. Inability to confidently determine the etiology of

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endodontic pathosis may be attributed to limitations in both clinical vitality testing and intraoral radiographs to detect odontogenic pathoses. CBCT imaging has the ability to detect periapical pathosis before it is apparent on 2D radiographs.[7]

- Preoperative factors such as the presence and true size of a periapical lesion play an important role in endodontic treatment outcome. Success, when measured by radiographic criteria, is higher when teeth are endodontically treated before radiographic signs of periapical disease are detected.[8]
- Previous findings have been validated in clinical studies in which primary endodontic disease detected with intraoral radiographs and CBCT was 20% and 48%, respectively. Several clinical studies had similar findings, although with slightly different percentages.[9,10] Ex vivo experiments in which simulated periapical lesions were created yielded similar results.[11,12] Results of in vivo animal studies, using histologic assessments as the gold standard, also showed similar results as those observed in human clinical and ex vivo studies.[13]
- Persistent intraoral pain following root canal therapy often presents a diagnostic challenge. An example is persistent dentoalveolar pain also known as atypical odontalgia.[14] The diagnostic yield of conventional intraoral radiographs and CBCT scans was evaluated in the differentiation between patients presenting with suspected atypical odontalgia versus symptomatic apical periodontitis, without radiographic evidence of periapical bone destruction.[15] CBCT imaging detected 17% more teeth with periapical bone loss than conventional radiography.

Initial treatment

Preoperative

Recommendation 3: Limited FOV CBCT should be considered the imaging modality of choice for the initial treatment of teeth with the potential for extra canals and suspected complex morphology, such as mandibular anterior teeth, and maxillary and mandibular premolars and molars, and dental anomalies.

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Intraoperative

Recommendation 4: If a preoperative CBCT has not been taken, limited FOV CBCT should be considered as the imaging modality of choice for intra-appointment identification and localization of calcified canals.

Postoperative

Recommendation 5: Intraoral radiographs should be considered the imaging modality of choice for immediate postoperative imaging.

Rationale

- Anatomical variations exist among different types of teeth. The success of nonsurgical root canal therapy depends on the identification of canals, the cleaning, shaping and obturation of root canal systems as well as the quality of the final restoration.
- 2D imaging does not consistently reveal the actual number of roots and canals. In studies, data acquired by CBCT showed a very strong correlation between sectioning and histologic examination.[16,17]
- In a 2013 study, CBCT showed higher mean values of specificity and sensitivity when compared to intraoral radiographic assessments in the detection of the MB2 canal.[18]

Nonsurgical retreatment

Recommendation 6: Limited FOV CBCT should be considered the imaging modality of choice if clinical examination and 2D intraoral radiography are inconclusive in the detection of vertical root fracture (VRF).

Rationale

- In nonsurgical retreatment, the presence of a vertical root fracture significantly decreases prognosis. In the majority of cases, the indication of a vertical root fracture is more often due to the specific pattern of bone loss and periodontal ligament space enlargement than to direct visualization of the fracture. CBCT may be

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recommended for the diagnosis of vertical root fracture in unrestored teeth when clinical signs and symptoms exist.

- Higher sensitivity and specificity were observed in a clinical study where the definitive diagnosis of vertical root fracture was confirmed at the time of surgery to validate CBCT findings, with sensitivity being 88% and specificity 75%.[19] Several case series studies have concluded that CBCT is a useful tool for the diagnosis of vertical root fractures. In vivo and laboratory studies evaluating CBCT in the detection of vertical root fractures agreed that the sensitivity, specificity, and accuracy of CBCT were generally higher and reproducible.[20,21] The detection of fractures was significantly higher for all CBCT systems when compared to intraoral radiographs. However, these results should be interpreted with caution because detection of vertical root fracture is dependent on the size of the fracture, presence of artifacts caused by obturation materials and posts, and the spatial resolution of the CBCT.

Recommendation 7: Limited FOV CBCT should be the imaging modality of choice when evaluating the nonhealing of previous endodontic treatment to help determine the need for further treatment, such as nonsurgical, surgical or extraction.

Recommendation 8: Limited FOV CBCT should be the imaging modality of choice for nonsurgical re-treatment to assess endodontic treatment complications, such as overextended root canal obturation material, separated endodontic instruments, and localization of perforations.

Rationale

- It is important to evaluate the factors that impact the outcome of root canal treatment. The outcome predictors identified with periapical radiographs and CBCT were evaluated by Liang et al.[22] The results showed that periapical radiographs detected periapical lesions in 18 roots (12%) as compared to 37 on CBCT scans (25%); 80% of apparently short root fillings based on intraoral radiographs images appeared flush on CBCT. Treatment outcome, length and density of

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root fillings, and outcome predictors determined by CBCT showed different values when compared with intraoral radiographs.

- Accurate treatment planning is an essential part of endodontic retreatment. Incorrect, delayed, or inadequate endodontic diagnosis and treatment planning places the patient at risk and may result in unnecessary treatment. Treatment planning decisions using CBCT versus intraoral radiographs were compared to the gold standard diagnosis.[23] An accurate diagnosis was reached in 36% to 40% of the cases with intraoral radiographs compared to 76% to 83% with CBCT. A high level of misdiagnosis was noted in invasive cervical resorption and vertical root fracture. In this study, the examiners altered their treatment plan after reviewing the CBCT in 56% to 62.2% of the cases, thus indicating the significant influence of CBCT.

Surgical retreatment

Recommendation 9: Limited FOV CBCT should be considered as the imaging modality of choice for presurgical treatment planning to localize root apex/apices and to evaluate the proximity to adjacent anatomical structures.

Rationale

The use of CBCT has been recommended for treatment planning of endodontic surgery.[24,25] CBCT visualization of the true extent of periapical lesions and their proximity to important vital structures and anatomical landmarks is superior to that of periapical radiographs.

Special conditions

a. Implant placement

Recommendation 10: Limited FOV CBCT should be considered as the imaging modality of choice for surgical placement of implants.[26]

b. Traumatic injuries

Recommendation 11: Limited FOV CBCT should be considered the

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imaging modality of choice for diagnosis and management of limited dento-alveolar trauma, root fractures, luxation, and /or displacement of teeth and localized alveolar fractures, in the absence of other maxillofacial or soft-tissue injury that may require other advanced imaging modalities.[27]

c. Resorptive defects

Recommendation 12: Limited FOV CBCT is the imaging modality of choice in the localization and differentiation of external and internal resorptive defects and the determination of appropriate treatment and prognosis.[28,29]

Outcome assessment

Recommendation 13: In the absence of clinical signs or symptoms, intraoral radiographs should be considered the imaging modality of choice for the evaluation of healing following nonsurgical and surgical endodontic treatment.

Recommendation 14: In the absence of signs and symptoms, if limited FOV CBCT was the imaging modality of choice at the time of evaluation and treatment, it may be the modality of choice for follow-up evaluation. In the presence of signs and symptoms, refer to Recommendation #7.

Rationale

- Use of limited FOV CBCT for evaluation of healing following nonsurgical and surgical treatment must be considered on a case-by-case basis, with due consideration given the risks and benefits of exposing the patient to ionizing radiation, the patient's history, clinical findings, mode of endodontic intervention, preexisting radiographic appearance, temporal changes, and any other systemic or extraneous factors that could confound the healing process.
- Accurate diagnosis of complete and uneventful healing as determined radiographically, with unequivocal clinical correlation supporting the radiographic diagnosis, is challenging.[30–33] The healing process is dependent on various factors, both extraneous and

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host related. In the future, once the parameters associated with healing are fully confirmed by evidence-based studies, the use of limited FOV CBCT for outcome assessment can be better interpreted.

Special Committee to Revise the Joint AAE/AAOMR Position Statement on Use of Limited FOV CBCT in Endodontics

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Joint Position Paper References

1. American Association of Endodontists, American Academy of Oral and Maxillofacial Radiology. *Use of cone-beam computed tomography in endodontics Joint Position Statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2, 2011;111: 234-237.
2. Grondahl HG, Huumonen S. *Radiographic manifestations of periapical inflammatory lesions*, Endod Topics 2004;8: 55-67.
3. Patel S, Durack C, Abella F, et al: *Cone beam computed tomography in Endodontics—a review*, Int Endod J 2015;48: 3-15.
4. Suomalainen A, Pakbaznejad Esmaeili E, Robinson S. *Dentomaxillofacial imaging with panoramic views and cone beam CT*, Insights imaging 2015;6: 1-16.
5. Venskutonis T, Plotino G, Juodzbalys G, et al: *The importance of cone-beam computed tomography in the management of endodontic problems: a review of the literature*, J Endod 12, 2014;40: 1895-1901.
6. Carter L, Farman AG, Geist J, et al: *American Academy of Oral and Maxillofacial Radiology executive opinion statement on performing and interpreting diagnostic cone beam computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 4, 2008;106: 561-562.
7. De Paula-Silva FW, Wu MK, Leonardo MR, et al: *Accuracy of periapical radiography and cone-beam computed tomography scans in diagnosing apical periodontitis using histopathological findings as a gold*

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www.konkur.in

standard, J Endod 7, 2009;35: 1009-1012.

8. Friedman S. *Prognosis of initial endodontic therapy*, Endod Topics 2002;2: 59-98.
9. Patel S, Wilson R, Dawood A, et al: *The detection of periapical pathosis using periapical radiography and cone beam computed tomography—part 1: preoperative status*, Int Endod J 2012;8: 702-710.
10. Abella F, Patel S, Duran-Sindreu F, et al: *Evaluating the periapical status of teeth with irreversible pulpitis by using cone-beam computed tomography scanning and periapical radiographs*, J Endod 12, 2012;38: 1588-1591.
11. Cheung G, Wei L, McGrath C. *Agreement between periapical radiographs and cone-beam computed tomography for assessment of periapical status of root filled molar teeth*, Int Endod J 10, 2013;46: 889-895.
12. Sogur E, Grondahl H, Bakst G, et al: *Does a combination of two radiographs increase accuracy in detecting acid-induced periapical lesions and does it approach the accuracy of cone-beam computed tomography scanning?*, J Endod 2, 2012;38: 131-136.
13. Patel S, Dawood A, Mannocci F, et al: *Detection of periapical bone defects in human jaws using cone beam computed tomography and intraoral radiography*, Int Endod J 6, 2009;42: 507-515.
14. Nixdorf D, Moana-Filho E. *Persistent dento-alveolar pain disorder (PDAP): Working towards a better understanding*, Rev Pain 4, 2011;5: 18-27.
15. Pigg M, List T, Petersson K, et al: *Diagnostic yield of conventional radiographic and cone-beam computed tomographic images in patients with atypical odontalgia*, Int Endod J 12, 2011;44: 1365-2591.
16. Blattner TC, Goerge N, Lee CC, et al: *Efficacy of CBCT*

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as a modality to accurately identify the presence of second mesiobuccal canals in maxillary first and second molars: a pilot study, J Endod 5, 2010;36: 867-870.

17. Michetti J, Maret D, Mallet JP, et al: *Validation of cone beam computed tomography as a tool to explore root canal anatomy*, J Endod 7, 2010;36: 1187-1190.
18. Vizzotto MB, Silveira PF, Arús NA, et al: *CBCT for the assessment of second mesiobuccal (MB2) canals in maxillary molar teeth: effect of voxel size and presence of root filling*, Int Endod J 9, 2013;46: 870-876.
19. Edlund M, Nair MK, Nair UP. *Detection of vertical root fractures by using cone-beam computed tomography: a clinical study*, J Endod 6, 2011;37: 768-772.
20. Metska ME, Aartman IH, Wesselink PR, et al: *Detection of vertical root fracture in vivo in endodontically treated teeth by cone-beam computed tomography scans*, J Endod 10, 2012;38: 1344-1347.
21. Brady E, Mannocci F, Wilson R, et al: *A comparison of CBCT and periapical radiography for the detection of vertical root fractures in non-endodontically treated teeth*, Int Endod J 8, 2014;47: 735-746.
22. Liang H, Li Gang, Wesselink P, et al: *Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans*, J Endod 3, 2011;37: 326-331.
23. Ee J, Fayad IM, Johnson B. *Comparison of endodontic diagnosis and treatment planning decisions using cone-beam volumetric tomography versus periapical radiography*, J Endod 7, 2014;40: 910-916.
24. Venskutonis T, Plotino G, Tocci L, et al: *Periapical and endodontic status scale based on periapical bone lesions and endodontic treatment quality evaluation using cone-*

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www.konkur.in

beam computed tomography, J Endod 2, 2015;41: 190-196.

25. Low KM, Dula K, Bürgin W, et al: *Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery*, J Endod 5, 2008;34: 557-562.
26. Tyndall D, Price J, Tetradis S, et al: *Position statement of the American Academy of Oral and Maxillofacial Radiology on selection criteria for the use of radiology in dental implantology with emphasis on cone beam computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol 6, 2012;113: 817-826.
27. May JJ, Cohenca N, Peters OA. *Contemporary management of horizontal root fractures to the permanent dentition: diagnosis, radiologic assessment to include cone-beam computed tomography*, Pediatr Dent 2013;35: 120-124.
28. Estrela C, Bueno MR, De Alencar AH, et al: *Method to evaluate Inflammatory Root Resorption by using Cone Beam computed tomography*, J Endod 11, 2009;35: 1491-1497.
29. Durack C, Patel S, Davies J, et al: *Diagnostic accuracy of small volume cone beam computed tomography and intraoral periapical radiography for the detection of simulated external inflammatory root resorption*, Int Endod J 2, 2011;44: 136-147.
30. Cheung G, Wei W, McGrath C. *Agreement between periapical radiographs and cone-beam computed tomography for assessment of periapical status of root filled molar teeth*, Int Endod J 10, 2013;46: 889-895.
31. Estrela C, Bueno MR, Azevedo BC, et al: *A new periapical index based on cone beam computed*

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www.konkur.in

tomography, J Endod 11, 2008;34: 1325-1331.

32. Tanomaru-Filho M, Jorge EG, Guerreiro-Tanomaru JM, et al: *Two- and tridimensional analysis of periapical repair after endodontic surgery*, Clin Oral Investig 1, 2015;19: 17-25.
33. von Arx T, Janner SF, Hanni S, et al: *Agreement between 2D and 3D radiographic outcome assessment one year after periapical surgery*, Int Endod J 1, 2015;49: 915-925.

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www.konkur.in

References

1. AAE and AAOMR joint position statement. *use of cone beam computed tomography in endodontics 2015 update*, J Endod 9, 2015;41: 1393-1396.
2. Academy of Osseointegration. *2010 guidelines of the academy of osseointegration for the provision of dental implants and associated patient care*, Int J Oral Maxillofac Implants 3, 2010;25: 620-627.
3. Ackerman S, Aguilera FC, Buie JM, et al: *Accuracy of 3-dimensional-printed endodontic surgical guide: a human cadaver study*, J Endod 5, 2019;45: 615-618.
4. ADA Council on Scientific Affairs. *An update on radiographic practices: information and recommendations*, J Am Dent Assoc 2, 2001;132: 234-238.
5. Ahn SY, Kim NH, Kim S, et al: *computer-aided design/computer-aided manufacturing-guided endodontic surgery: guided osteotomy and apex localization in a mandibular molar with a thick buccal bone plate*, J Endod 4, 2018;44: 665-670.
6. Akdeniz BG, Sogur E. *An ex vivo comparison of conventional and digital radiography for perceived image quality of root fillings*, Int Endod J 6, 2005;38: 397-401.
7. Al-Fouzan KS. *Incidence of rotary ProFile instrument fracture and the potential for bypassing in vivo*, Int Endod J 12, 2003;36: 864-867.
8. Alavi AM, Opasanon A, Ng YL, et al: *Root and canal morphology of Thai maxillary molars*, Int Endod J 5, 2002;35: 478-485.
9. American Association of Endodontists: *Glossary of*

Telegram: @dental_k

www.konkur.in

endodontic terms. https://www.aae.org/specialty/clinical-resources/glossary-endodontic-terms/. Chicago, 2019, update.

10. American Dental Association moves forward on electronic standards. ADA News 15, 1999;30: A.
11. Anderson LC, Kosinski TF, Mentag PJ. *A review of the intraosseous course of the nerves of the mandible*, J Oral Implantol 4, 1991;17: 394-403.
12. Andersson L. *Epidemiology of traumatic dental injuries*, J Endod 3 Suppl, 2013;39: S2- S5.
13. Andersson L, Andreasen JO, Day P, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth*, Dent Traumatol 2, 2012;28: 88-96.
14. Andreasen FM, Pedersen BV. *Prognosis of luxated permanent teeth—the development of pulp necrosis*, Endod Dent Traumatol 6, 1985;1: 207-220.
15. Andreasen FM, Sewerin I, Mandel U, et al: *Radiographic assessment of simulated root resorption cavities*, Endod Dent Traumatol 1, 1987;3: 21-27.
16. Andreasen FM, Andreasen JO. Crown fractures. *Textbook and color atlas of traumatic injuries to the teeth* ed 3 1994; Munksgaard Copenhagen.
17. Andreasen JO, Ahrensburg SS, Tsilingaridis G. *Root fractures: the influence of type of healing and location of fracture on tooth survival rates - an analysis of 492 cases*, Dent Traumatol 5, 2012;28: 404-409.
18. Andreasen JO, Ravn JJ. *Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample*, Int J Oral Surg 5, 1972;1: 235-239.
19. Andreasen JO, Andreasen FM, Andersson L. Textbook and color atlas of traumatic injuries to the teeth 2007; John

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www.konkur.in

Wiley & Sons Copenhagen, Munksgaard.
20. Ariji Y, Ariji E, Nakashima M, et al: *Magnetic resonance imaging in endodontics: a literature review*, Oral Radiol 1, 2018;34: 10-16.
21. Backman B, Wahlin YB. *Variations in number and morphology of permanent teeth in 7-year-old Swedish children*, Int J Paediatr Dent 1, 2001;11: 11-17.
22. Bakland LK. *Root resorption*, Dent Clin North Am 2, 1992;36: 491-507.
23. Ball RL, Barbizam JV, Cohenca N. *Intraoperative endodontic applications of cone-beam computed tomography*, J Endod 4, 2013;39: 548-557.
24. Beck-Mannagetta J, Necek D, Grasserbauer M. *Solitary aspergillosis of maxillary sinus, a complication of dental treatment*, Lancet 8361, 1983;2: 1260.
25. Benavides E, Rios HF, Ganz SD, et al: *Use of cone beam computed tomography in implant dentistry: the international congress of oral implantologists consensus report*, Implant Dent 2, 2012;21: 78-86.
26. Bender IB. *Factors influencing the radiographic appearance of bony lesions*, J Endod 4, 1982;8: 161-170.
27. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone: I*, J Am Dent Assoc 1961;62: 152-160.
28. Bergenholtz G. *Micro-organisms from necrotic pulp of traumatized teeth*, Odontol Revy 4, 1974;25: 347-358.
29. Block MS, Emery RW. *Static or dynamic navigation for implant placement-choosing the method of guidance*, J Oral Maxillofac Surg 2, 2016;74: 269-277.
30. Borg E, Kallqvist A, Grondahl K, et al: *Film and digital radiography for detection of simulated root resorption cavities*, Oral Surg Oral Med Oral Pathol Oral Radiol

Telegram: @dental_k

www.konkur.in

Endod 1, 1998;86: 110-114.
31. Bornstein MM, Lauber R, Sendi P, et al: *Comparison of periapical radiography and limited cone-beam computed tomography in mandibular molars for analysis of anatomical landmarks before apical surgery*, J Endod 2, 2011;37: 151-157.
32. Bornstein MM, Wasmer J, Sendi P, et al: *Characteristics and dimensions of the Schneiderian membrane and apical bone in maxillary molars referred for apical surgery: a comparative radiographic analysis using limited cone beam computed tomography*, J Endod 1, 2012;38: 51-57.
33. Bornstein MM, Wolner-Hanssen AB, Sendi P, et al: *Comparison of intraoral radiography and limited cone beam computed tomography for the assessment of root-fractured permanent teeth*, Dent Traumatol 6, 2009;25: 571-577.
34. Bouillaguet S, Wataha JC, Tay FR, et al: *Initial in vitro biological response to contemporary endodontic sealers*, J Endod 10, 2006;32: 989-992.
35. Bratel J, Jontell M, Dahlgren U, et al: *Effects of root canal sealers on immunocompetent cells in vitro and in vivo*, Int Endod J 3, 1998;31: 178-188.
36. Brennan PC, McEntee M, Evanoff M, et al: *Ambient lighting: effect of illumination on soft-copy viewing of radiographs of the wrist*, AJR Am J Roentgenol 2, 2007;188: W177- W180.
37. Brooks JK, Kleinman JW. *Retrieval of extensive gutta-percha extruded into the maxillary sinus: use of 3-dimensional cone-beam computed tomography*, J Endod 9, 2013;39: 1189-1193.
38. Brynolf I. *A histological and roentgenological study of periapical region of human upper incisors*, Odontol Revy

Telegram: @dental_k

www.konkur.in

11, 1967;18: 1.
39. Bueno MR, De Carvalhosa AA, De Souza Castro PH, et al: *Mesenchymal chondrosarcoma mimicking apical periodontitis*, J Endod 11, 2008;34: 1415-1419.
40. Bueno MR, Estrela C, De Figueiredo JA, et al: *Map-reading strategy to diagnose root perforations near metallic intracanal posts by using cone beam computed tomography*, J Endod 1, 2011;37: 85-90.
41. Burger CL, Mork TO, Hutter JW, et al: *Direct digital radiography versus conventional radiography for estimation of canal length in curved canals*, J Endod 4, 1999;25: 260-263.
42. Calberson FL, Hommez GM, De Moor RJ. *Fraudulent use of digital radiography: methods to detect and protect digital radiographs*, J Endod 5, 2008;34: 530-536.
43. Caplan D. *Epidemiologic issues in studies of association between apical periodontitis and systemic health*, Endod Topics 15, 2008;8:.
44. Carter L, Farman AG, Geist J, et al: *American academy of oral and maxillofacial radiology executive opinion statement on performing and interpreting diagnostic cone beam computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 4, 2008;106: 561-562.
45. Claeys V, Wackens G. *Bifid mandibular canal: literature review and case report*, Dentomaxillofac Radiol 1, 2005;34: 55-58.
46. Cohenca N, Simon JH, Mathur A, et al: *Clinical indications for digital imaging in dento-alveolar trauma. Part 2: Root resorption*, Dent Traumatol 2, 2007;23: 105-113.
47. Cohenca N, Simon JH, Roges R, et al: *Clinical indications for digital imaging in dento-alveolar trauma.*

Telegram: @dental_k

www.konkur.in

Part 1: Traumatic injuries, Dent Traumatol 2, 2007;23: 95-104.

48. Connert T, Krug R, Eggmann F, et al: *Guided endodontics versus conventional access cavity preparation: a comparative study on substance loss using 3-dimensional-printed teeth*, J Endod 3, 2019;45: 327-331.
49. Cotti E, Dessi C, Piras A, et al: *Can a chronic dental infection be considered a cause of cardiovascular disease?, A review of the literature*, Int J Cardiol 1, 2011;148: 4-10.
50. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J Endod 9, 2007;33: 1121-1132.
51. Crump MC, Natkin E. *Relationship of broken root canal instruments to endodontic case prognosis: a clinical investigation*, J Am Dent Assoc 6, 1970;80: 1341-1347.
52. Cuje J, Bargholz C, Hulsmann M. *The outcome of retained instrument removal in a specialist practice*, Int Endod J 7, 2010;43: 545-554.
53. de Paula-Silva FW, Wu MK, Leonardo MR, et al: *Accuracy of periapical radiography and cone-beam computed tomography scans in diagnosing apical periodontitis using histopathological findings as a gold standard*, J Endod 7, 2009;35: 1009-1012.
54. Davidovich E, Heling I, Fuks AB. *The fate of a mid-root fracture: a case report*, Dent Traumatol 3, 2005;21: 170-173.
55. Day PF, Duggal MS. *A multicentre investigation into the role of structured histories for patients with tooth avulsion at their initial visit to a dental hospital*, Dent Traumatol 5, 2003;19: 243-347.
56. Di Fiore PM, Genov KA, Komaroff E, et al: *Nickel-*

Telegram: @dental_k

www.konkur.in

titanium rotary instrument fracture: a clinical practice assessment, Int Endod J 9, 2006;39: 700-708.

57. Di Nardo D, Gambarini G, Capuani S, et al: *Nuclear magnetic resonance imaging in endodontics: a review*, J Endod 4, 2018;44: 536-542.
58. Diangelis AJ, Andreasen JO, Ebeleseder KA, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth*, Dent Traumatol 1, 2012;28: 2-12.
59. Diniz de Lima E, Lira de Farias Freitas AP, Mariz Suassuna FC, et al: *Assessment of cone-beam computed tomographic artifacts from different intracanal materials on birooted teeth*, J Endod 2, 2019;45: 209-213.
60. DICOM: *Digital imaging and communications in medicine (DICOM) Part 1: Introduction and overview*. ftp://medical.nema.org/medical/dicom/2008/08_01pu.pdf. Accessed July 26, 2009.
61. Durack C, Patel S, Davies J, et al: *Diagnostic accuracy of small volume cone beam computed tomography and intraoral periapical radiography for the detection of simulated external inflammatory root resorption*, Int Endod J 2, 2011;44: 136-147.
62. Edlund M, Nair MK, Nair UP. *Detection of vertical root fractures by using cone-beam computed tomography: a clinical study*, J Endod 6, 2011;37: 768-772.
63. Eikenberg S, Vandre R. *Comparison of digital dental X-ray systems with self-developing film and manual processing for endodontic file length determination*, J Endod 2, 2000;26: 65-67.
64. Eraso FE, Analoui M, Watson AB, et al: *Impact of lossy compression on diagnostic accuracy of radiographs for*

Telegram: @dental_k

www.konkur.in

periapical lesions, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 5, 2002;93: 621-625.

65. Escoda-Francoli J, Canalda-Sahli C, Soler A, et al: *Inferior alveolar nerve damage because of overextended endodontic material: a problem of sealer cement biocompatibility?*, J Endod 12, 2007;33: 1484-1489.
66. Estrela C, Bueno MR, De Alencar AH, et al: *Method to evaluate inflammatory root resorption by using cone beam computed tomography*, J Endod 11, 2009;35: 1491-1497.
67. Estrela C, Bueno MR, Leles CR, et al: *Accuracy of cone beam computed tomography and panoramic and periapical radiography for detection of apical periodontitis*, J Endod 3, 2008;34: 273-279.
68. Eversole LR, Leider AS, Hansen LS. *Ameloblastomas with pronounced desmoplasia*, J Oral Maxillofac Surg 11, 1984;42: 735-740.
69. Fahrig R, Fox AJ, Lownie S, et al: *Use of a C-arm system to generate true three-dimensional computed rotational angiograms: preliminary in vitro and in vivo results*, AJNR Am J Neuroradiol 8, 1997;18: 1507-1514.
70. Fan W, Fan B, Gutmann JL, et al: *Identification of a C-shaped canal system in mandibular second molars. Part III. Anatomic features revealed by digital subtraction radiography*, J Endod 10, 2008;34: 1187-1190.
71. Fan Y, Glickman GN, Umorin M, et al: *A novel prefabricated grid for guided endodontic microsurgery*, J Endod 5, 2019;45: 606-610.
72. Farman AG, Avant SL, Scarfe WC, et al: *In vivo comparison of Visualix-2 and Ektaspeed Plus in the assessment of periradicular lesion dimensions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2, 1998;85: 203-209.

Telegram: @dental_k

www.konkur.in

73. Feely L, Mackie IC, Macfarlane T. *An investigation of root-fractured permanent incisor teeth in children*, Dent Traumatol 1, 2003;19: 52-54.
74. Finucane D, Kinirons MJ. *External inflammatory and replacement resorption of luxated, and avulsed replanted permanent incisors: a review and case presentation*, Dent Traumatol 3, 2003;19: 170-174.
75. Fletcher R, Fletcher S. Clinical epidemiology: the essentials 2005; Lippincott Williams & Wilkins Baltimore.
76. Forman GH, Rood JP. *Successful retrieval of endodontic material from the inferior alveolar nerve*, J Dent 1, 1977;5: 47-50.
77. Fuss Z, Lustig J, Tamse A. *Prevalence of vertical root fractures in extracted endodontically treated teeth*, Int Endod J 4, 1999;32: 283-286.
78. Gassner R, Bosch R, Tuli T, et al: *Prevalence of dental trauma in 6000 patients with facial injuries: implications for prevention*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1, 1999;87: 27-33.
79. Gencoglu N, Helvacioglu D. *Comparison of the different techniques to remove fractured endodontic instruments from root canal systems*, Eur J Dent 2, 2009;3: 90-95.
80. Geurtsen W, Leyhausen G. *Biological aspects of root canal filling materials—histocompatibility,cytotoxicity, and mutagenicity*, Clin Oral Investig 1, 1997;1: 5-11.
81. Glendor U. *Epidemiology of traumatic dental injuries—a 12 year review of the literature*, Dent Traumatol 6, 2008;24: 603-611.
82. Gold SI, Hasselgren G. *Peripheral inflammatory root resorption. A review of the literature with case reports*, J Clin Periodontol 8, 1992;19: 523-534.
83. Goldberg F, De Silvio A, Dreyer C. *Radiographic*

Telegram: @dental_k

www.konkur.in

assessment of simulated external root resorption cavities in maxillary incisors, Endod Dent Traumatol 3, 1998;14: 133-136.

84. Gomes AP, de Araujo EA, Goncalves SE, et al: *Treatment of traumatized permanent incisors with crown and root fractures: a case report*, Dent Traumatol 5, 2001;17: 236-239.
85. Gonzalez-Martin M, Torres-Lagares D, Gutierrez-Perez JL, et al: *Inferior alveolar nerve paresthesia after overfilling of endodontic sealer into the mandibular canal*, J Endod 8, 2010;36: 1419-1421.
86. Goodis HE, Rossall JC, Kahn AJ. *Endodontic status in older U.S. adults. Report of a survey*, J Am Dent Assoc 11, 2001;132: 1525-1530 quiz 95–96.
87. Green TL, Walton RE, Taylor JK, et al: *Radiographic and histologic periapical findings of root canal treated teeth in cadaver*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 6, 1997;83: 707-711.
88. Grotz KA, Al-Nawas B, de Aguiar EG, et al: *Treatment of injuries to the inferior alveolar nerve after endodontic procedures*, Clin Oral Investig 2, 1998;2: 73-76.
89. Guivarc'h M, Ordioni U, Catherine JH, et al: *Implications of endodontic-related sinus aspergillosis in a patient treated by infliximab: a case report*, J Endod 1, 2015;41: 125-129.
90. Gulabivala K, Aung TH, Alavi A, et al: *Root and canal morphology of Burmese mandibular molars*, Int Endod J 5, 2001;34: 359-370.
91. Gulabivala K, Opasanon A, Ng YL, et al: *Root and canal morphology of Thai mandibular molars*, Int Endod J 1, 2002;35: 56-62.
92. Gulabivala K, Searson LJ. *Clinical diagnosis of internal*

Telegram: @dental_k

www.konkur.in

resorption: an exception to the rule, Int Endod J 5, 1995;28: 255-260.

93. Gunst V, Mavridou A, Huybrechts B, et al: *External cervical resorption: an analysis using cone beam and microfocus computed tomography and scanning electron microscopy*, Int Endod J 9, 2013;46: 877-887.
94. Gutteridge DL. *The use of radiographic techniques in the diagnosis and management of periodontal diseases*, Dentomaxillofac Radiol 2, 1995;24: 107-113.
95. Hassan B, Metska ME, Ozok AR, et al: *Detection of vertical root fractures in endodontically treated teeth by a cone beam computed tomography scan*, J Endod 5, 2009;35: 719-722.
96. Hassan B, Metska ME, Ozok AR, et al: *Comparison of five cone beam computed tomography systems for the detection of vertical root fractures*, J Endod 1, 2010;36: 126-129.
97. Hatcher DC. *Operational principles for cone-beam computed tomography*, J Am Dent Assoc Suppl 3, 2010;141: 3S- 6S.
98. Headache Classification Subcommittee, International Headache Society. *The international classification of headache disorders. Second edition.*, Cephalgia 1, 2004;24:.
99. Heithersay GS. *Management of tooth resorption*, Aust Dent J 1 Suppl, 2007;52: S105- S121.
100. Hopp RN, Marchi MT, Kellermann MG, et al: *Lymphoma mimicking a dental periapical lesion*, Leuk Lymphoma 5, 2012;53: 1008-1010.
101. Iikubo M, Kobayashi K, Mishima A, et al: *Accuracy of intraoral radiography, multidetector helical CT, and limited cone-beam CT for the detection of horizontal tooth*

Telegram: @dental_k

www.konkur.in

root fracture, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 5, 2009;108: e70- e74.

102. Ingle JI. *A standardized endodontic technique utilizing newly designed instruments and filling materials*, Oral Surg Oral Med Oral Pathol 1961;14: 83-91.
103. Jung M, Lommel D, Klimek J. *The imaging of root canal obturation using micro-CT*, Int Endod J 9, 2005;38: 617-626.
104. Kakehashi S, Stanley HR, Fitzgerald RJ. *The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral Pathol 1965;20: 340-349.
105. Kamburoglu K, Ilker Cebeci AR, Grondahl HG. *Effectiveness of limited cone-beam computed tomography in the detection of horizontal root fracture*, Dent Traumatol 3, 2009;25: 256-261.
106. Kamburoglu K, Kursun S. *A comparison of the diagnostic accuracy of CBCT images of different voxel resolutions used to detect simulated small internal resorption cavities*, Int Endod J 9, 2010;43: 798-807.
107. Kamburoglu K, Kursun S, Yuksel S, et al: *Observer ability to detect ex vivo simulated internal or external cervical root resorption*, J Endod 2, 2011;37: 168-175.
108. Kanagasingam S, Lim CX, Yong CP, et al: *Diagnostic accuracy of periapical radiography and cone beam computed tomography in detecting apical periodontitis using histopathological findings as a reference standard*, Int Endod J 5, 2017;50: 417-426.
109. Karabucak B, Bunes A, Chehoud C, et al: *Prevalence of apical periodontitis in endodontically treated premolars and molars with untreated canal: a cone-beam computed tomography study*, J Endod 4, 2016;42: 538-541.

Telegram: @dental_k

www.konkur.in

110. Kerekes K, Tronstad L. *Long-term results of endodontic treatment performed with a standardized technique*, J Endod 3, 1979;5: 83-90.
111. Khedmat S, Rouhi N, Drage N, et al: *Evaluation of three imaging techniques for the detection of vertical root fractures in the absence and presence of gutta-percha root fillings*, Int Endod J 11, 2012;45: 1004-1009.
112. Kim E, Kim KD, Roh BD, et al: *Computed tomography as a diagnostic aid for extracanal invasive resorption*, J Endod 7, 2003;29: 463-465.
113. Kim TS, Caruso JM, Christensen H, et al: *A comparison of cone-beam computed tomography and direct measurement in the examination of the mandibular canal and adjacent structures*, J Endod 7, 2010;36: 1191-1194.
114. Kitagawa H, Scheetz JP, Farman AG. *Comparison of complementary metal oxide semiconductor and charge-coupled device intraoral X-ray detectors using subjective image quality*, Dentomaxillofac Radiol 6, 2003;32: 408-411.
115. Kositbowornchai S, Nuansakul R, Sikram S, et al: *Root fracture detection: a comparison of direct digital radiography with conventional radiography*, Dentomaxillofac Radiol 2, 2001;30: 106-109.
116. Kovisto T, Ahmad M, Bowles WR. *Proximity of the mandibular canal to the tooth apex*, J Endod 3, 2011;37: 311-315.
117. Lamus F, Katz JO, Glaros AG. *Evaluation of a digital measurement tool to estimate working length in endodontics*, J Contemp Dent Pract 1, 2001;2: 24-30.
118. Lara-Mendes STO, Barbosa CFM, Machado VC, et al: *A new approach for minimally invasive access to severely calcified anterior teeth using the guided endodontics*

Telegram: @dental_k

www.konkur.in

technique, J Endod 10, 2018;44: 1578-1582.
119. Lavasani SA, Tyler C, Roach SH, et al: *Cone-beam computed tomography: anatomic analysis of maxillary posterior teeth-impact on endodontic microsurgery*, J Endod 6, 2016;42: 890-895.
120. Leddy BJ, Miles DA, Newton CW, et al: *Interpretation of endodontic file lengths using RadioVisiography*, J Endod 11, 1994;20: 542-545.
121. Leonardi Dutra K, Haas L, Porporatti AL, et al: *Diagnostic accuracy of cone-beam computed tomography and conventional radiography on apical periodontitis: a systematic review and meta-analysis*, J Endod 3, 2016;42: 356-364.
122. Levin L, M T. *Root resorption*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Chicago 425-448.
123. Li G, Sanderink GC, Welander U, et al: *Evaluation of endodontic files in digital radiographs before and after employing three image processing algorithms*, Dentomaxillofac Radiol 1, 2004;33: 6-11.
124. Liang YH, Li G, Wesselink PR, et al: *Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans*, J Endod 3, 2011;37: 326-331.
125. Liedke GS, da Silveira HE, da Silveira HL, et al: *Influence of voxel size in the diagnostic ability of cone beam tomography to evaluate simulated external root resorption*, J Endod 2, 2009;35: 233-235.
126. Littner MM, Kaffe I, Tamse A, et al: *Relationship between the apices of the lower molars and mandibular canal—a radiographic study*, Oral Surg Oral Med Oral Pathol 5, 1986;62: 595-602.

Telegram: @dental_k

www.konkur.in

127. Lofthag-Hansen S, Huumonen S, Grondahl K, et al: *Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1, 2007;103: 114-119.
128. Loushine RJ, Weller RN, Kimbrough WF, et al: *Measurement of endodontic file lengths: calibrated versus uncalibrated digital images*, J Endod 12, 2001;27: 779-781.
129. Low KM, Dula K, Burgin W, et al: *Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery*, J Endod 5, 2008;34: 557-562.
130. Lu Y, Liu Z, Zhang L, et al: *Associations between maxillary sinus mucosal thickening and apical periodontitis using cone-beam computed tomography scanning: a retrospective study*, J Endod 8, 2012;38: 1069-1074.
131. Maillet M, Bowles WR, McClanahan SL, et al: *Cone-beam computed tomography evaluation of maxillary sinusitis*, J Endod 6, 2011;37: 753-757.
132. Marchesan MA, Lloyd A, Clement DJ, et al: *Impacts of contracted endodontic cavities on primary root canal curvature parameters in mandibular molars*, J Endod 10, 2018;44: 1558-1562.
133. Marmulla R, Wortche R, Muhling J, et al: *Geometric accuracy of the NewTom 9000 Cone Beam CT*, Dentomaxillofac Radiol 1, 2005;34: 28-31.
134. Mehra P, Murad H. *Maxillary sinus disease of odontogenic origin*, Otolaryngol Clin North Am 2, 2004;37: 347-364.
135. Melo SL, Bortoluzzi EA, Abreu M Jr, et al: *Diagnostic ability of a cone-beam computed tomography scan to*

Telegram: @dental_k

www.konkur.in

assess longitudinal root fractures in prosthetically treated teeth, J Endod 11, 2010;36: 1879-1882.

136. Metska ME, Aartman IH, Wesselink PR, et al: *Detection of vertical root fractures in vivo in endodontically treated teeth by cone-beam computed tomography scans*, J Endod 10, 2012;38: 1344-1347.
137. Miguens SA Jr, Veeck EB, Fontanella VR, et al: *A comparison between panoramic digital and digitized images to detect simulated periapical lesions using radiographic subtraction*, J Endod 12, 2008;34: 1500-1503.
138. Mikrogeorgis G, Lyroudia K, Molyvdas I, et al: *Digital radiograph registration and subtraction: a useful tool for the evaluation of the progress of chronic apical periodontitis*, J Endod 7, 2004;30: 513-517.
139. Miles D. Color atlas of cone beam volumetric imaging for dental applications 2008; Quintessence Publishing Hanover Park, IL.
140. Miles DA, Danfort RA. *A clinician's guide to understanding cone beam volumetric imaging (CBVI)*, Acad Dent Ther Stomatol Special Issue, 2007; 1-13 www.ineedce.com.
141. Mirota DJ, Uneri A, Schafer S, et al: *Evaluation of a system for high-accuracy 3D image-based registration of endoscopic video to C-arm cone-beam CT for image-guided skull base surgery*, IEEE Trans Med Imaging 7, 2013;32: 1215-1226.
142. Nair MK, Nair UP. *Digital and advanced imaging in endodontics: a review*, J Endod 1, 2007;33: 1-6.
143. Nance RS, Tyndall D, Levin LG, et al: *Diagnosis of external root resorption using TACT (tuned-aperture computed tomography)*, Endod Dent Traumatol 1,

Telegram: @dental_k

www.konkur.in

2000;16: 24-28.
144. Narayana P, Hartwell GR, Wallace R, et al: *Endodontic clinical management of a dens invaginatus case by using a unique treatment approach: a case report*, J Endod 8, 2012;38: 1145-1148.
145. Neelakantan P, Subbarao C, Subbarao CV. *Comparative evaluation of modified canal staining and clearing technique, cone-beam computed tomography, peripheral quantitative computed tomography, spiral computed tomography, and plain and contrast medium-enhanced digital radiography in studying root canal morphology*, J Endod 9, 2010;36: 1547-1551.
146. Neelakantan P, Subbarao C, Subbarao CV, et al: *Root and canal morphology of mandibular second molars in an Indian population*, J Endod 8, 2010;36: 1319-1322.
147. Nevares G, Cunha RS, Zuolo ML, et al: *Success rates for removing or bypassing fractured instruments: a prospective clinical study*, J Endod 4, 2012;38: 442-444.
148. Ng Y, Gulabivala K. *Evaluation of outcomes*, Pathways of the pulp ed 11 2016; Elsevier St. Louis, MO 474-531.
149. Ng YL, Aung TH, Alavi A, et al: *Root and canal morphology of Burmese maxillary molars*, Int Endod J 8, 2001;34: 620-630.
150. Nicopoulou-Karayianni K, Bragger U, Patrikiou A, et al: *Image processing for enhanced observer agreement in the evaluation of periapical bone changes*, Int Endod J 7, 2002;35: 615-622.
151. Nixdorf DR, Moana-Filho EJ, Law AS, et al: *Frequency of persistent tooth pain after root canal therapy: a systematic review and meta-analysis*, J Endod 2, 2010;36: 224-230.
152. Oehlers FA. *Dens invaginatus (dilated composite*

Telegram: @dental_k

www.konkur.in

odontome). II. Associated posterior crown forms and pathogenesis, Oral Surg Oral Med Oral Pathol 12, 1957;10: 1302-1316.

153. Ohrbach R, List T, Goulet JP, et al: *Recommendations from the International Consensus Workshop: convergence on an orofacial pain taxonomy*, J Oral Rehabil 10, 2010;37: 807-812.
154. Orhan K, Aksoy U, Kalender A. *Cone-beam computed tomographic evaluation of spontaneously healed root fracture*, J Endod 9, 2010;36: 1584-1587.
155. Ozer SY. *Detection of vertical root fractures of different thicknesses in endodontically enlarged teeth by cone beam computed tomography versus digital radiography*, J Endod 7, 2010;36: 1245-1249.
156. Panitvisai P, Parunnit P, Sathorn C, et al: *Impact of a retained instrument on treatment outcome: a systematic review and meta-analysis*, J Endod 5, 2010;36: 775-780.
157. Parashos P, Gordon I, Messer HH. *Factors influencing defects of rotary nickel-titanium endodontic instruments after clinical use*, J Endod 10, 2004;30: 722-725.
158. Patel K, Mannocci F, Patel S. *The assessment and management of external cervical resorption with periapical radiographs and cone-beam computed tomography: a clinical study*, J Endod 10, 2016;42: 1435-1440.
159. Patel S. *New dimensions in endodontic imaging: Part 2. Cone beam computed tomography*, Int Endod J 6, 2009;42: 463-475.
160. Patel S. *The use of cone beam computed tomography in the conservative management of dens invaginatus: a case report*, Int Endod J 8, 2010;43: 707-713.
161. Patel S, Brady E, Wilson R, et al: *The detection of*

Telegram: @dental_k

www.konkur.in

vertical root fractures in root filled teeth with periapical radiographs and CBCT scans, Int Endod J 12, 2013;46: 1140-1452.

162. Patel S, Dawood A. *The use of cone beam computed tomography in the management of external cervical resorption lesions*, Int Endod J 9, 2007;40: 730-737.
163. Patel S, Dawood A, Wilson R, et al: *The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography - an in vivo investigation*, Int Endod J 9, 2009;42: 831-838.
164. Patel S, Durack C, Abella F, et al: *Cone beam computed tomography in Endodontics - a review*, Int Endod J 1, 2015;48: 3-15.
165. Patel S, Ford TP. *Is the resorption external or internal?*, Dent Update 4, 2007;34: 218-220 222, 224–226, 229.
166. Patel S, Kanagasingam S, Pitt Ford T. *External cervical resorption: a review*, J Endod 5, 2009;35: 616-625.
167. Patel S, Mavridou AM, Lambrechts P, et al: *External cervical resorption-part 1: histopathology, distribution and presentation*, Int Endod J 11, 2018;51: 1205-1223.
168. Peters E, Lau M. *Histopathologic examination to confirm diagnosis of periapical lesions: a review*, J Can Dent Assoc 9, 2003;69: 598-600.
169. Peters OA, Peters CI, Schonenberger K, et al: *ProTaper rotary root canal preparation: effects of canal anatomy on final shape analysed by micro CT*, Int Endod J 2, 2003;36: 86-92.
170. Peters OA, Schonenberger K, Laib A. *Effects of four Ni-Ti preparation techniques on root canal geometry assessed by micro computed tomography*, Int Endod J 3, 2001;34: 221-230.
171. Pew Research Center: *US population projections: 2005-*

Telegram: @dental_k

www.konkur.in

2050. www.pewhispanic.org/2008/02/11/us-population-projections-2005-2050.

172. Piepenbring ME, Potter BJ, Weller RN, et al: *Measurement of endodontic file lengths: a density profile plot analysis*, J Endod 10, 2000;26: 615-618.
173. Pigg M, List T, Petersson K, et al: *Diagnostic yield of conventional radiographic and cone-beam computed tomographic images in patients with atypical odontalgia*, Int Endod J 12, 2011;44: 1092-1101.
174. Pineda F, Kuttler Y. *Mesiodistal and buccolingual roentgenographic investigation of 7,275 root canals*, Oral Surg Oral Med Oral Pathol 1, 1972;33: 101-110.
175. Pogrel MA. *Damage to the inferior alveolar nerve as the result of root canal therapy*, J Am Dent Assoc 1, 2007;138: 65-69.
176. Pope O, Sathorn C, Parashos P. *A comparative investigation of cone-beam computed tomography and periapical radiography in the diagnosis of a healthy periapex*, J Endod 3, 2014;40: 360-365.
177. Renton T. *Prevention of iatrogenic inferior alveolar nerve injuries in relation to dental procedures*, Dent Update 6, 2010;37: 350-352 354–356, 358–360 passim.
178. Ricucci D, Langeland K. *Apical limit of root canal instrumentation and obturation, part 2. A histological study*, Int Endod J 6, 1998;31: 394-409.
179. Rodrigues CD, Estrela C. *Traumatic bone cyst suggestive of large apical periodontitis*, J Endod 4, 2008;34: 484-489.
180. Rodrigues CD, Villar-Neto MJ, Sobral AP, et al: *Lymphangioma mimicking apical periodontitis*, J Endod 1, 2011;37: 91-96.
181. Rodriguez G, Patel S, Duran-Sindreu F, et al: *Influence of cone-beam computed tomography on endodontic*

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retreatment strategies among general dental practitioners and endodontists, J Endod 9, 2017;43: 1433-1437.

182. Rud J, Omnell KA. *Root fractures due to corrosion. Diagnostic aspects*, Scand J Dent Res 5, 1970;78: 397-403.
183. Ruddle C. *Endodontic disinfection-tsunami irrigation*, Endod Prac Feb, 2008; 7-15.
184. Ruddle CJ. *Endodontic diagnosis*, Dent Today 10, 2002;21: 90-92 94, 96–101; quiz 01, 78.
185. Samei E, Krupinski E. Medical imaging: perception and techniques 2010; Cambridge University Press Cambridge, UK.
186. Scannapieco FA. *Position paper of The American Academy of Periodontology: periodontal disease as a potential risk factor for systemic diseases*, J Periodontol 7, 1998;69: 841-850.
187. Scarfe WC. *Imaging of maxillofacial trauma: evolutions and emerging revolutions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2 Suppl, 2005;100: S75- S96.
188. Scarfe WC, Levin MD, Gane D, et al: *Use of cone beam computed tomography in endodontics*, Int J Dent 2009;2009: 634567.
189. Schilder H. *Cleaning and shaping the root canal*, Dent Clin North Am 2, 1974;18: 269-296.
190. Schilder H. *Canal debridement and disinfection*, Cohen S Burns C Pathways of the pulp ed 1 1976; Mosby St. Louis 111-113.
191. Scolozzi P, Lombardi T, Jaques B. *Successful inferior alveolar nerve decompression for dysesthesia following endodontic treatment: report of 4 cases treated by mandibular sagittal osteotomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 5, 2004;97: 625-631.

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192. Sert S, Bayirli GS. *Evaluation of the root canal configurations of the mandibular and maxillary permanent teeth by gender in the Turkish population*, J Endod 6, 2004;30: 391-398.
193. Sharan A, Madjar D. *Correlation between maxillary sinus floor topography and related root position of posterior teeth using panoramic and cross-sectional computed tomography imaging*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 3, 2006;102: 375-381.
194. Shemesh H, Cristescu RC, Wesselink PR, et al: *The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations*, J Endod 4, 2011;37: 513-516.
195. Shishvan HH, Ebrahimnejad H. *A study on the ability of panoramic, CT, cone-beam CT, MRI and ultrasonography in detecting different foreign-bodies in the maxillofacial region (an in-vitro study)*, Electron J Gen Med 16, 2018;15:.
196. Siewerdsen JH, Jaffray DA. *Cone-beam computed tomography with a flat-panel imager: effects of image lag*, Med Phys 12, 1999;26: 2635-2647.
197. Simonton JD, Azevedo B, Schindler WG, et al: *Age- and gender-related differences in the position of the inferior alveolar nerve by using cone beam computed tomography*, J Endod 7, 2009;35: 944-949.
198. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 10, 1990;16: 498-504.
199. Sonoda M, Takano M, Miyahara J, et al: *Computed radiography utilizing scanning laser stimulated luminescence*, Radiology 3, 1983;148: 833-838.
200. Spili P, Parashos P, Messer HH. *The impact of instrument*

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www.konkur.in

fracture on outcome of endodontic treatment, J Endod 12, 2005;31: 845-850.

201. Stratemann SA, Huang JC, Maki K, et al: *Comparison of cone beam computed tomography imaging with physical measures*, Dentomaxillofac Radiol 2, 2008;37: 80-93.
202. Swets J. Signal detection theory and ROC analysis in psychology and diagnostics: collected papers 1996; Lawrence Erlbaum Associates Mahwah, NJ.
203. Tahmasbi M, Jalali P, Nair MK, et al: *Prevalence of middle mesial canals and isthmi in the mesial root of mandibular molars: an in vivo cone-beam computed tomographic study*, J Endod 7, 2017;43: 1080-1083.
204. Tian YY, Guo B, Zhang R, et al: *Root and canal morphology of maxillary first premolars in a Chinese subpopulation evaluated using cone-beam computed tomography*, Int Endod J 11, 2012;45: 996-1003.
205. Torabinejad M. Endodontics: principles and practice 2009; Saunders St Louis.
206. Torabinejad M, Bahjri K. *Essential elements of evidenced-based endodontics: steps involved in conducting clinical research*, J Endod 8, 2005;31: 563-569.
207. Toure B, Faye B, Kane AW, et al: *Analysis of reasons for extraction of endodontically treated teeth: a prospective study*, J Endod 11, 2011;37: 1512-1515.
208. Tronstad L. *Root resorption—etiology, terminology and clinical manifestations*, Endod Dent Traumatol 6, 1988;4: 241-252.
209. Trope M. *Root resorption of dental and traumatic origin: classification based on etiology*, Pract Periodontics Aesthet Dent 4, 1998;10: 515-522.
210. Tsesis I, Rosen E, Schwartz-Arad D, et al: *Retrospective evaluation of surgical endodontic treatment: traditional*

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versus modern technique, J Endod 5, 2006;32: 412-416.
211. Tsesis I, Rosen E, Tamse A, et al: *Diagnosis of vertical root fractures in endodontically treated teeth based on clinical and radiographic indices: a systematic review*, J Endod 9, 2010;36: 1455-1458.
212. Tyndall DA, Ludlow JB, Platin E, et al: *A comparison of Kodak Ektaspeed Plus film and the Siemens Sidexis digital imaging system for caries detection using receiver operating characteristic analysis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1, 1998;85: 113-118.
213. Tyndall DA, Price JB, Tetradis S, et al: *Position statement of the American Academy of Oral and Maxillofacial Radiology on selection criteria for the use of radiology in dental implantology with emphasis on cone beam computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol 6, 2012;113: 817-826.
214. Valmaseda-Castellon E, Berini-Aytes L, Gay-Escoda C. *Inferior alveolar nerve damage after lower third molar surgical extraction: a prospective study of 1117 surgical extractions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 4, 2001;92: 377-383.
215. Vande Voorde HE BA. *Estimating endodontic "working length" with paralleling radiographs*, Oral Surg Oral Med Oral Pathol. 1969;27: 106-110.
216. Vaz de Souza D, Schirru E, Mannocci F, et al: *External cervical resorption: a comparison of the diagnostic efficacy using 2 different cone-beam computed tomographic units and periapical radiographs*, J Endod 1, 2017;43: 121-125.
217. Versteeg CH, Sanderink GC, Lobach SR, et al: *Reduction in size of digital images: does it lead to less detectability or loss of diagnostic information?*, Dentomaxillofac

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www.konkur.in

Radiol 2, 1998;27: 93-96.
218. Versteeg KH, Sanderink GC, van Ginkel FC, et al: *Estimating distances on direct digital images and conventional radiographs*, J Am Dent Assoc 4, 1997;128: 439-443.
219. Vertucci F. *Root canal morphology and its relationship to endodontic procedures*, Endod Topics 3, 2005;10:.
220. Vertucci FJ. *Root canal morphology of mandibular premolars*, J Am Dent Assoc 1, 1978;97: 47-50.
221. Vertucci FJ. *Root canal anatomy of the human permanent teeth*, Oral Surg Oral Med Oral Pathol 5, 1984;58: 589-599.
222. Vier-Pelisser FV, Pelisser A, Recuero LC, et al: *Use of cone beam computed tomography in the diagnosis, planning and follow up of a type III dens invaginatus case*, Int Endod J 2, 2012;45: 198-208.
223. Wang P, Yan XB, Lui DG, et al: *Detection of dental root fractures by using cone-beam computed tomography*, Dentomaxillofac Radiol 5, 2011;40: 290-298.
224. Weng XL, Yu SB, Zhao SL, et al: *Root canal morphology of permanent maxillary teeth in the Han nationality in Chinese Guanzhong area: a new modified root canal staining technique*, J Endod 5, 2009;35: 651-656.
225. Wenzel A, Haiter-Neto F, Frydenberg M, et al: *Variable-resolution cone-beam computerized tomography with enhancement filtration compared with intraoral photostimulable phosphor radiography in detection of transverse root fractures in an in vitro model*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 6, 2009;108: 939-945.
226. Westphalen VP, Gomes de Moraes I, Westphalen FH, et

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www.konkur.in

al: *Conventional and digital radiographic methods in the detection of simulated external root resorptions: a comparative study*, Dentomaxillofac Radiol 4, 2004;33: 233-235.

227. White SC, Scarfe WC, Schulze RK, et al: *The Image Gently in Dentistry campaign: promotion of responsible use of maxillofacial radiology in dentistry for children*, Oral Surg Oral Med Oral Pathol Oral Radiol 3, 2014;118: 257-261.
228. Wolner-Hanssen AB, von Arx T. *Permanent teeth with horizontal root fractures after dental trauma*, A retrospective study. Schweiz Monatsschr Zahnmed 3, 2010;120: 200-212.
229. Wu MK, Dummer PM, Wesselink PR. *Consequences of and strategies to deal with residual post-treatment root canal infection*, Int Endod J 5, 2006;39: 343-356.
230. Wu MK, Shemesh H, Wesselink PR. *Limitations of previously published systematic reviews evaluating the outcome of endodontic treatment*, Int Endod J 8, 2009;42: 656-666.
231. Yoshioka T, Kobayashi C, Suda H, et al: *An observation of the healing process of periapical lesions by digital subtraction radiography*, J Endod 8, 2002;28: 589-591.
232. Young GR. *Contemporary management of lateral root perforation diagnosed with the aid of dental computed tomography*, Aust Endod J 3, 2007;33: 112-118.
233. Zadik Y, Sandler V, Bechor R, et al: *Analysis of factors related to extraction of endodontically treated teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 5, 2008;106: e31- e35.
234. Zahedi S, Mostafavi M, Lotfirikan N. *Anatomic study of mandibular posterior teeth using cone-beam computed*

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tomography for endodontic surgery, J Endod 5, 2018;44: 738-743.

235. Zhang L, Wang T, Cao Y, et al: *In vivo detection of subtle vertical root fracture in endodontically treated teeth by cone-beam computed tomography*, J Endod 2019.
236. Zhang R, Wang H, Tian YY, et al: *Use of cone-beam computed tomography to evaluate root and canal morphology of mandibular molars in Chinese individuals*, Int Endod J 11, 2011;44: 990-999.
237. Zhang Y, Zhang L, Zhu XR, et al: *Reducing metal artifacts in cone-beam CT images by preprocessing projection data*, Int J Radiat Oncol Biol Phys 3, 2007;67: 924-932.
238. Zheng Q, Zhang L, Zhou X, et al: *C-shaped root canal system in mandibular second molars in a Chinese population evaluated by cone-beam computed tomography*, Int Endod J 9, 2011;44: 857-862.
239. Zhou W, Han C, Li D, et al: *Endodontic treatment of teeth induces retrograde peri-implantitis*, Clin Oral Implants Res 12, 2009;20: 1326-1332.

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3: Lesions that mimic endodontic pathosis

David J. Landwehr

CHAPTER OUTLINE

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Importance of differential diagnosis

It has long been recognized that periapical cysts and granulomas make up the majority of radiographic lesions in the tooth-bearing regions of the jaws.[17,28,137,243] Many of these radiolucencies are asymptomatic and discovered on routine radiographs. For clinicians, it is easy to assume that any periapical radiolucency associated with pain is of pulpal or inflammatory origin, especially if the tooth has a significant restorative history. However, numerous disease entities of variable clinical significance can be identified in the periradicular regions, and differentiating the typical endodontic presentation from that of a potentially more life-threatening or insidious etiology is of critical importance to ensure ideal management and outcome (Fig. 3.1).[139,141,187,216,235]

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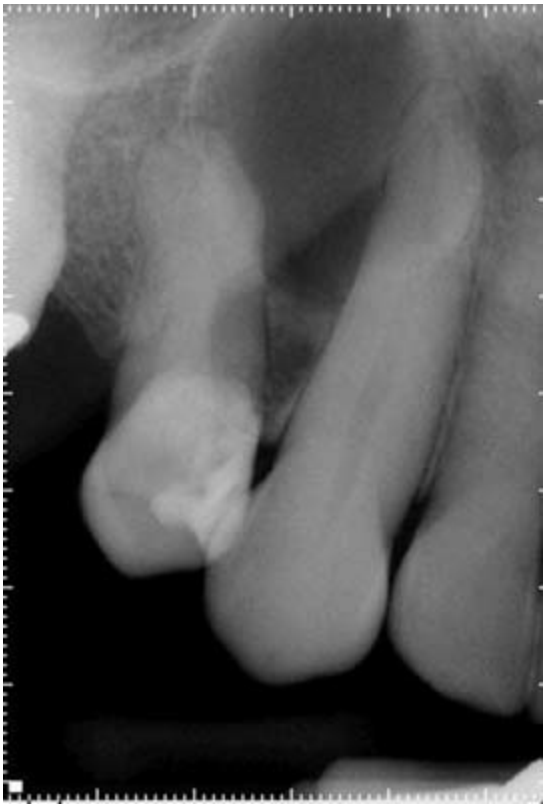

FIG. 3.1 A well-defined radiolucency near the apex of the maxillary first premolar and canine. Although the premolar did not respond to pulp testing, the location of the lesion and clinical presentation were suspicious and a biopsy revealed an odontogenic keratocyst.

Radiograph shows radiolucency between canine and first premolar. Crown of the first premolar is less dense toward the edge and is fused to adjacent canine.

Limits of pulp testing

The use of pulp testing in conjunction with medical history, dental history, clinical examination and radiographic findings is considered the standard routine to differentiate pulpal pathology from that of a noninflammatory or nondental etiology.[73] In spite of this, pulp testing has significant limitations as historical studies suggest there is not a correlation between the histologic status of the pulp and a patient's clinical signs and symptoms.[228,229] However, a more recent investigation reported a correlation between the histologic and clinical diagnosis of irreversible pulpitis in 84.4% of cases and agreement was even higher for normal pulps and reversible pulpitis.[209] It is

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not uncommon to encounter patients with extensive caries throughout the dentition, suggesting significant pulpal inflammation, but no history of pain (Fig. 3.2).

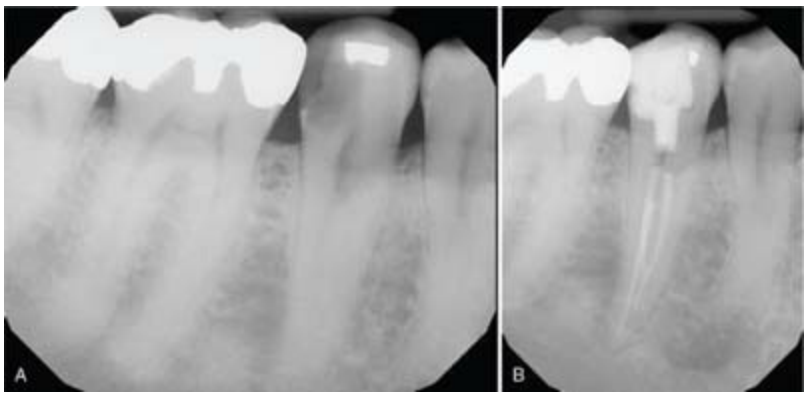

FIG. 3.2 **A,** Deep caries in the mandibular second premolar, but the patient was asymptomatic with no history of pain. **B,** Tooth #29 responded normally to both cold and electric pulp testing, but nonsurgical root canal treatment was completed for restorative reasons.

A) Radiograph of lower jaw shows a small radiopaque rectangular patch with a large radiolucent blotch in the second premolar toward the molar tooth.

B) Radiograph shows second premolar with two filled root canals and a large radiopaque region in the crown.

The accuracy of diagnostic testing is further complicated by a lack of proprioceptive fibers in the dental pulp, which results in the patient's inability to localize the source of pain.[167,228] Thermal and electric pulp testing are actually a measure of the pulp's ability to sense a stimulus rather than a representation of overall health, and these measurements are susceptible to both false-positive and false-negative responses.[5,98,118,119,168,197,264] Given the diagnostic limitations of pulp testing, many clinicians place an undue amount of importance on radiographic findings.

Limits of radiology

According to a 2012 systematic review of cross-sectional studies evaluating the periapical status of over 300,000 teeth, 5% of all teeth had periapical

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radiolucencies.[188] The prevalence of periapical radiolucencies in the population illustrates the critical importance of being able to formulate an accurate differential diagnosis of these radiographic abnormalities. Historically, radiographs could not demonstrate periapical lesions located in cancellous bone until mineral loss resulted in a lesion of 1 to 7 mm.[18,20] This suggests the prevalence of periapical inflammatory disease is even higher because all teeth with inflamed or necrotic pulps will not result in a periapical radiolucency. Different anatomical locations within the oral cavity have variable likelihoods for identification of a radiolucent lesion based on the thickness of the cortical plate and volume of bone.[19] Additionally, the actual size of a radiolucent defect is larger clinically than can be appreciated with 2D radiographic imaging.[230] In less common clinical situations, the periodontal ligament (PDL) space can be widened or displaced prior to pulp necrosis due to the cytokine cascade from an inflamed pulp.[2,270] Because of the technical limits of traditional imaging, a 2D radiograph should not be considered a panacea in the diagnostic process.

The interpretation of 2D films and digital radiographs is subjective due to significant intraobserver and interobserver variability.[32,95,248] Three-dimensional cone-beam computed tomography (CBCT) technology is a more accurate way of identifying periapical inflammatory disease,[10,57,75,150,193,259] but the interpretation of 3D images is also subjective and based on the experience of the clinician and quality of the images.[16,190,202,254] Moreover, clinicians must be able to identify all of the incidental findings that may be discovered with these larger data sets.[67,185,210] The American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology published a Joint Position Statement in 2015 providing 14 scientifically based guidelines for the use of CBCT in endodontic treatment. This position paper and other reports recommended the judgment of the treating clinician should determine the need for a CBCT and suggested that patient history and clinical examination should demonstrate that the benefits of the scan outweigh the potential risks.[1,151] “CBCT should not be used routinely for endodontic diagnosis or for screening purposes in the absence of clinical signs and symptoms. Clinicians should use CBCT only when the need for imaging cannot be met by lower dose two-dimensional radiography.”[1] Furthermore, when indications arise for the use of CBCT a

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limited field of view is recommended to minimize the amount of radiation and maximize the image resolution. A CBCT can serve as a valuable tool to differentiate subtle radiographic changes and facilitate the development of a more comprehensive differential diagnosis compared to conventional radiography (Fig. 3.3).[57,192]

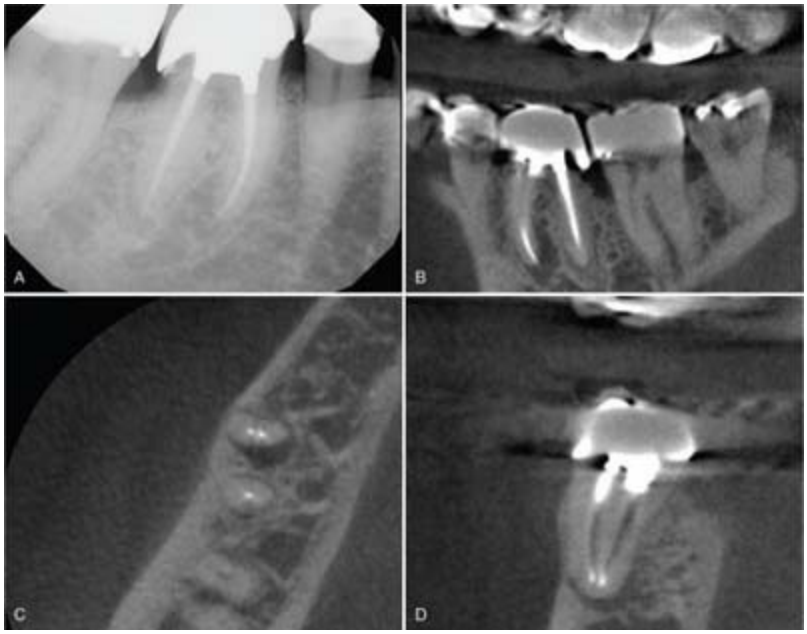

FIG. 3.3 A limited field CBCT is a useful adjunct when the findings on a periapical image are not conclusive. **A,** A subtle radiolucent area is present at the mesial root apex of the mandibular first molar. **B,** Sagittal, **(C)** axial and **(D)** coronal CBCT images reveal a more prominent loss of density at this root apex.

A) CBCT image shows radiopaque crown and comparatively less opaque two root canals in mandibular first molar.

B) Sagittal view shows first molar with radiolucent crown. One root canal is radiolucent, whereas other root canal is radiopaque.

C) Axial view shows teeth arranged diagonally. Two teeth have tiny dense dots at the center.

D) Coronal view shows radiolucent root canals and crown. Some of the regions at the neck region and edges appear radiopaque.

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Differential diagnosis of periapical pathosis

In many cases, the radiographic findings associated with noninflammatory pathologies can very closely mimic the presentation of the more common periapical cyst or periapical granuloma, and there is no way to definitively determine the clinical importance of a lesion based on radiographic features alone.[17,137,139,141,235,243] However, accurate interpretation of the bone abnormalities in conjunction with a thorough review of the medical history, dental history, pulp testing, and clinical examination can yield a differential diagnosis that could result in earlier treatment and potentially a better prognosis. Given the limitations of both pulp testing and radiographic interpretation, the diagnosis of periapical inflammatory disease is associated with a variable level of uncertainty. As a result, using a repeatable method when evaluating radiographic abnormalities can aid the clinician in developing a comprehensive differential diagnosis.

Multilocular radiolucencies

Periapical cysts and periapical granulomas have a wide spectrum of radiographic presentations with some causing extensive bone destruction and others resulting in minimal radiographic abnormalities (Fig. 3.4). Irrespective of size, the presentation of periapical inflammatory disease should be radiographically unilocular and confirmed with a lack of responsiveness to pulp testing. If a multilocular radiolucency is identified on a periapical image, then a panoramic radiograph or CBCT is recommended to confirm the multilocular growth pattern and extent of the lesion. The differential diagnosis of multilocular radiolucencies is extensive, but the most common are odontogenic keratocyst (OKC), ameloblastoma, and central giant cell lesion (CGCL) (Box 3.1).

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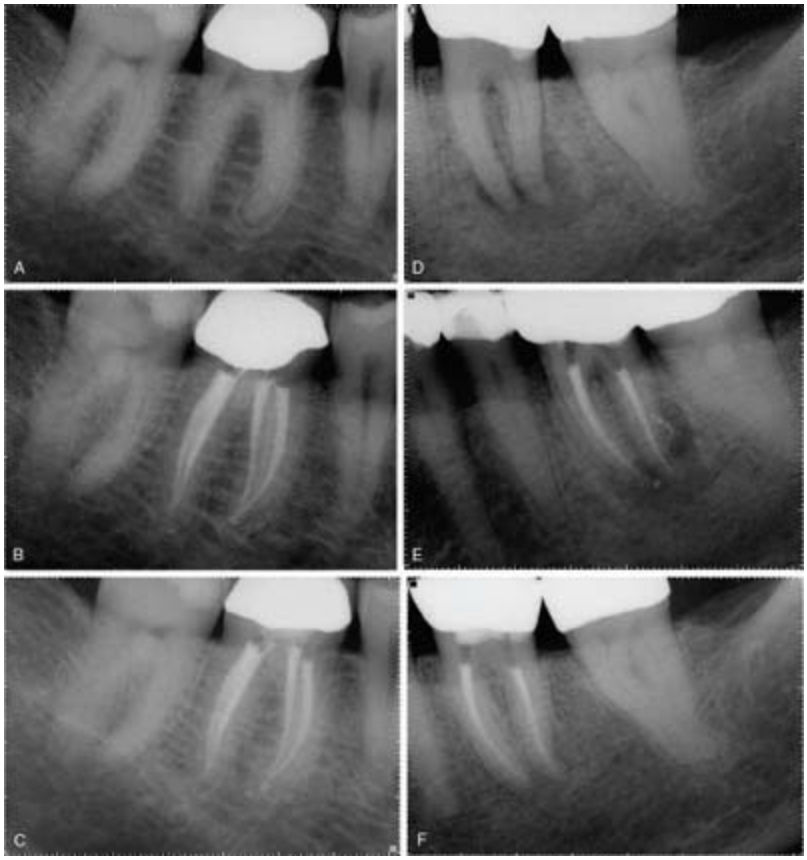

FIG. 3.4 Periapical inflammatory disease presents with a wide spectrum of radiographic changes. **A,** Preoperative periapical image of a mandibular first molar with a necrotic pulp and asymptomatic apical periodontitis. There is a widened PDL space and subtle radiolucency at the mesial root apex. **B,** Nonsurgical root canal treatment was completed. **C,** Evidence of bone healing at 6-month recall. **D,** Preoperative periapical image of a mandibular first molar with a necrotic pulp and asymptomatic apical periodontitis. This lesion is considerably larger than in image **A** and involves the mesial and distal root apices with coronal extension along the distal aspect. **E,** Nonsurgical root canal treatment was completed and a lateral canal is present in the apical half of the root. **F,** Evidence of bone healing at 6-month recall.

A) Radiograph shows central tooth with radiopaque crown and root with medium density. The apex of one of the root canals shows a radiolucent space close to adjacent lamina dura.

B) Radiograph shows central tooth with filled root canals. The tissue between the bifurcated region appears less dense. Beside this, the neck region at the one edge is radiolucent.

C) Radiograph is same as that of B, except that the tissue at the central of bifurcated region and neck is dense.

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D) Radiograph shows a large dark patch at the root tip. The crown of tooth is radiopaque.

E) Radiograph shows bifurcated root of tooth with partially filled (up to three-fourth) root canals and thin tissue at root tip.

F) Radiograph is same as that of E, except that tissue is thick at root tip.

Box 3.1

Uncommon Multilocular Radiolucencies

Ameloblastic fibroma
Odontogenic myxoma
Central odontogenic fibroma
Calcifying epithelial odontogenic tumor
Orthokeratinized odontogenic cyst
Lateral periodontal cyst
Calcifying odontogenic cyst
Central hemangioma/Arteriovenous malformation
Aneurysmal bone cyst
Cherubism
Hyperparathyroidism
Intraosseous mucoepidermoid carcinoma

Odontogenic keratocyst

The OKC is a developmental cyst that accounts for 3% to 11% of all odontogenic cysts.[42] In 2005, the World Health Organization reclassified the OKC as a neoplasm with the designation keratocystic odontogenic tumor because of molecular genetic changes found in other neoplasms, but in 2017 the name was changed back to the original OKC.[69,238] As a result, both names will appear in the literature and it is important to distinguish OKCs from lesions of inflammatory origin due to substantial growth potential and recurrence risk.

The OKC can be identified at any age, but most are discovered as a solitary lesion between the ages of 10 and 40.[42] If multiple OKCs are present or a single OKC is diagnosed before the age of 10, nevoid basal cell carcinoma

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syndrome would be suspected.[81] Histologically, the basal layer of the cyst lining has palisaded cuboidal cells, often with hyperchromatic nuclei, and the surface of the cyst exhibits parakeratin production. The OKC has a thin, noninflamed, stratified squamous epithelial cyst lining consisting of six to eight layers of cells that is easily dislodged from the underlying connective tissue during surgical treatment increasing the likelihood for recurrence. The growth pattern also influences the reported recurrence rates because a multilocular presentation is more difficult to completely remove with curettage.[121] Most reports indicate a recurrence rate of approximately 30%.[23,49] However, the rate of recurrence is variable, depending on the study protocol, and recurrence can occur more than 5 years after the original surgical intervention, suggesting long-term follow-up is mandatory.[23,81]

Radiographic features.

The OKC presents as a well-defined radiolucency and can be found at any location in either jaw (Fig. 3.5).[6,91,216] They can occur in the periapical region, but there is an uneven distribution of OKCs throughout the maxilla and mandible with the highest incidence in the posterior body and ramus of the mandible (Fig. 3.6).[182,250] When an OKC is associated with an impacted tooth (25% to 40% of cases), it should not be confused with a lesion of endodontic origin due to its location surrounding the crown. This presentation would be indistinguishable from that of a dentigerous cyst (Fig. 3.7). Smaller OKCs are most often asymptomatic and commonly present as a unilocular radiolucency while larger OKCs tend to have the classic multilocular appearance.[250] Larger OKCs may present with pain and swelling, but many are asymptomatic with no evidence of bone expansion.

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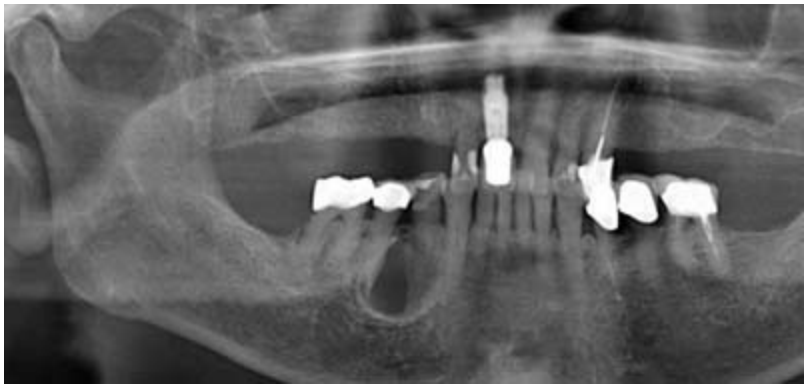

FIG. 3.5 Although most odontogenic keratocysts occur as multilocular radiolucencies at the angle of the mandible, they can be located at any position in either jaw. A cropped panoramic radiograph reveals a corticated odontogenic keratocyst in the mandibular right premolar region.

Radiograph shows panoramic view of teeth with a hollow cavity to the right. The teeth are not properly aligned at the center. Crowns of some teeth are extremely radiopaque.

Source: (Courtesy Dr. Christel Haberland.)

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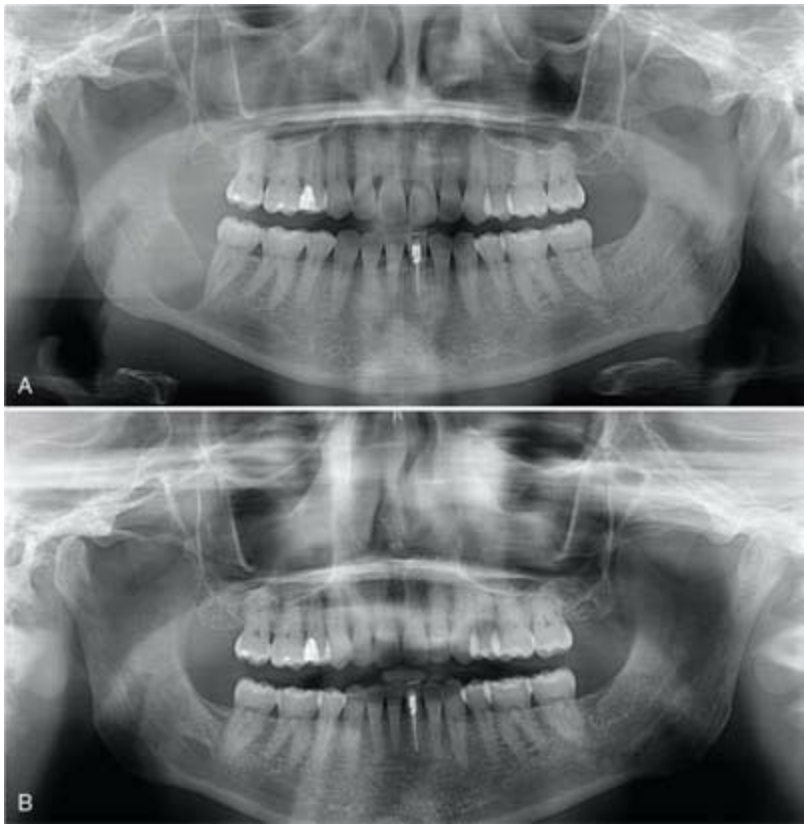

FIG. 3.6 **A,** A cropped panoramic radiograph shows a well-defined unilocular odontogenic keratocyst at the right angle of the mandible and distal root of second molar. The lesion was surgically removed and good bone healing was evident at a 3-year recall with no indication of recurrence **(B)**.

A) Radiograph shows light patches below teeth at the center. Tissue is less dense toward the left and right in the lower jaw.

B) Radiograph is similar to A, except that there is a dense tissue below teeth.

Source: (Courtesy Dr. Christopher Bacsik.)

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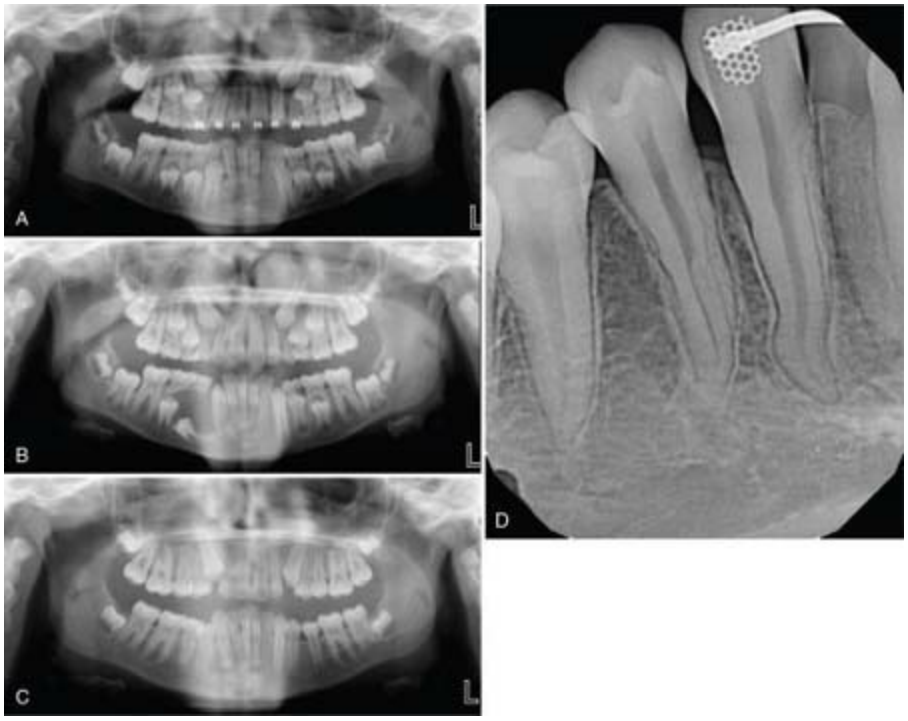

FIG. 3.7 **A,** A panoramic radiograph of a 10-year-old female with a mixed dentition. **B,** Serial panoramic images were obtained for orthodontic reasons and a well-defined expansile lesion developed in the mandibular right premolar region over 9 months. This type of radiolucency should not be confused with an inflammatory etiology due to its location around the crown of an unerupted tooth. This odontogenic keratocyst was treated by unroofing the cyst without removal of the entire lesion. **C,** Panoramic image with no evidence of recurrence at 2 years. **D,** Periapical image with no evidence of recurrence at 6 years.

A) Radiograph shows open mouth with irregular arrangement of teeth. Some teeth arrange at the root apex of the other teeth except the central part.

B) Radiograph is similar to A radiograph, except that there a hollow cavity is created adjacent to the center.

C) Radiograph shows open mouth with almost regular arrangement of teeth. The teeth at the center in the mandible appear slightly clustered.

D) Radiograph shows four teeth with a rectangular framework and a white strip on the third tooth. Part of the crown of fourth tooth is broken at one side.

Ameloblastoma

Ameloblastoma is the most common odontogenic tumor. It presents over a

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wide age range with an equal frequency after the age of 20, but it is uncommon before 20 years of age and rare before the age of 10.[122] These benign tumors are slow growing but can become large and disfiguring if left untreated. There are six histologic subtypes, but these differences have no impact on the treatment options or recurrence rate.[31] Ameloblastomas will often infiltrate normal bone resulting in extension beyond radiographic margins and higher recurrence rates. A more aggressive surgical resection with a larger margin will result in a lower recurrence rate, but this may not be possible due to the size of the tumor or its location near important anatomical structures. Marginal resection is the most common treatment, but recurrence rates of 15% have prompted some surgeons to recommend a 1 to 2 cm resection margin due to the infiltrative growth pattern. However, when the tumor presents near vital structures, a resection may not be possible and the resulting curettage is associated with a recurrence rate that ranges from 50% to 90%.[94] As the molecular pathways involved in the pathogenesis of ameloblastoma are better understood, it may be possible to block these mechanisms with targeted chemotherapeutics, preventing the need for surgery.[109,219] As with OKCs, recurrence can occur many years following the initial surgical intervention and long-term follow-up is mandatory.

Radiographic features.

Ameloblastoma can occur at any location in the maxilla or mandible, but 66% are found in the mandibular posterior region and only 15% present in the maxilla.[179] Smaller lesions are often asymptomatic and may present as a unilocular radiolucency found on routine radiographic examination. A small, unilocular ameloblastoma in the periapical region may be indistinguishable from a periapical cyst or granuloma.[77,97] Larger ameloblastomas often result in a painless swelling, and expansion of the cortical plate may help differentiate an ameloblastoma from both inflammatory lesions and OKCs (Fig. 3.8). As with OKCs, many ameloblastomas are associated with an impacted tooth and should not be confused with a lesion of endodontic origin. Ameloblastomas may cause resorption of adjacent teeth, and irregular scalloping of the margin is highly suggestive of the diagnosis.[48] However, when an ameloblastoma presents as a typical nonexpansile multilocular radiolucency, it cannot be distinguished from any other lesion with this

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growth potential based on radiographic findings alone.

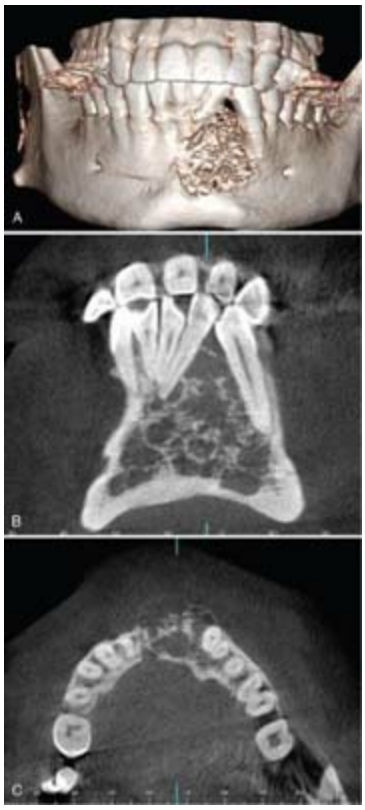

FIG. 3.8 **A,** 3D volumetric rendering of an ameloblastoma between roots of mandibular canine and mandibular lateral incisor with destruction of the buccal cortical plate. **B,** Sagittal CBCT image reveals a low-density lesion with numerous septae displacing the roots of teeth #22 and #23. **C,** Axial CBCT image exhibiting a perforation of the buccal cortical plate and buccal-lingual expansion that is common with ameloblastomas. This lesion would be characterized as a multilocular radiolucency on a periapical image.

A) 3D image represents the panoramic view of jaw with a triangular chamber of abnormal cells below the root of teeth at the center.

B) CBCT image shows a big cavity with chambers of thin tissues below the teeth in the lower jaw.

C) CBCT image shows lower jaw, in which teeth form an arch shape and has roughly circular patch with septae.

Source: (Courtesy Dr. Bruno Azevedo.)

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Central giant cell lesion

Long recognized by the term *central giant cell granuloma*, the most recent World Health Organization classification now designates this entity as CGCL.[69] Like OKCs and ameloblastomas, the CGCL will often present as a multilocular radiolucency. It has an unknown etiology that may be a reactive process, but a benign neoplastic origin has not been ruled out. The diagnosis of CGCL is made over a wide age range, with the majority discovered before the age of 30.[172,265] Most case series report a female predilection, and 70% of CGCLs are located in the mandible with a tendency to cross the midline (Fig. 3.9).[143] Histologically, the CGCL is identical to the brown tumor of hyperparathyroidism, and the distinguishing microscopic feature is the presence of multinucleated giant cells. The number and size of the giant cells vary considerably throughout the lesion, and there is controversy about the prognostic significance of this finding, with some studies suggesting a more aggressive behavior.[143,265] Curettage is the most common treatment for CGCL, but a more extensive surgery may be needed for aggressive or recurrent lesions. Alternative treatments for CGCL include, but are not limited to, intra-lesional corticosteroids, bisphosphonates, and imatinib.[54] The overall recurrence rate for CGCL is variable, with most studies reporting a rate near 20%.[53]

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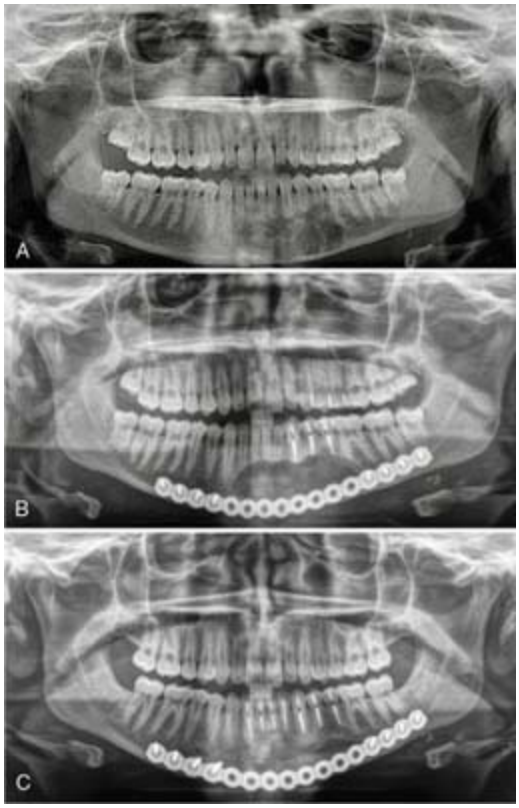

FIG. 3.9 **A,** Central giant cell lesion extending from left mandibular premolar region across the midline in a 17-year-old female. This radiolucency cannot be differentiated from other multilocular lesions without a biopsy (see Box 3.1). **B,** The left mandibular premolars and canine had nonsurgical endodontic treatment prior to removal of the central giant cell lesion and the mandibular incisors had nonsurgical endodontic treatment after removal of the lesion, when it was determined intraoperatively the blood supply to these teeth had been compromised. **C,** Eighteen-month recall with evidence of increased bone density, but incomplete healing.

A) Radiograph shows panoramic view of teeth with sore tissues below the root of teeth near the center.

B) Radiograph shows large bulge below the teeth extending from incisors to premolars. Below the bulge, there is a white chain made of circular rings extending from the third molar to first molar on the other side.

C) Radiograph shows a slightly dense tissue in place of the bulge with the same chain extending from the third molar to second molar on the other side.

Radiographic features.

The characteristic presentation of CGCLs can be subdivided into two

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categories.[44] Nonaggressive CGCLs tend to be asymptomatic and are often discovered on routine radiographs or due to a subtle expansion. These lesions are smaller and grow more slowly than the aggressive subtype. Additionally, nonaggressive CGCLs should not cause perforation of the cortical plate or resorption of roots. Aggressive CGCLs are more likely to exhibit pain and rapid growth.[53] Aggressive CGCLs may result in perforation of the cortical plate, resorption of roots, paresthesia, and extension into soft tissues following perforation. These lesions are usually larger at the time of diagnosis and exhibit a higher recurrence rate.

Most commonly, a CGCL will have a well-defined but noncorticated border. The size can vary from several millimeters to more than 10 cm. As a result, the amount of expansion and thinning of the cortical plate will be highly unpredictable as will the number and size of the loculations (Fig. 3.10). When a smaller lesion is discovered in the periapical region, it can look identical to a periapical cyst or periapical granuloma and larger CGCLs may resemble both OKC and ameloblastoma.[51,226]

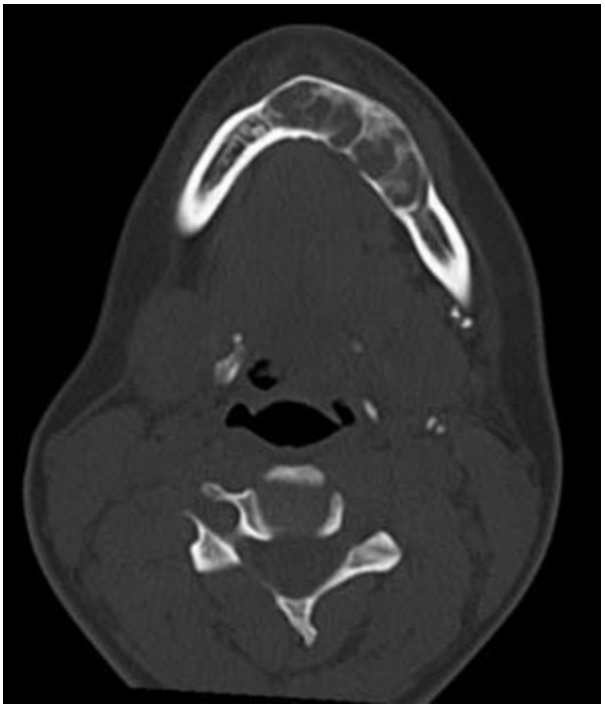

FIG. 3.10 Axial view of CT showing the extent of the central giant cell lesion of the same patient from Fig. 3.9. This plane reveals a multilocular growth pattern with thinning of both the buccal and lingual cortical plates.

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CT scan shows an arc-shaped lower jaw at the top of a large irregular structure. The arc shows multiple vesicles with thin tissue.

Radiopacities in the periapical region

Although a multilocular radiolucency is consistent with a noninflammatory etiology, any of the multilocular radiolucencies can also present as a unilocular lesion in its earlier stages prior to reaching full growth potential. As a result, other radiographic features need to be considered when forming a differential diagnosis. Endodontic lesions of inflammatory origin most often present as radiolucent lesions with no indication of radiopacity.[21] As a result, if any evidence of radiopacity is identified, suspicion of a noninflammatory etiology should be elevated, especially if a limited restorative history is present or pulp testing is inconclusive. Exceptions to this rule would include condensing osteitis and maxillary sinusitis of endodontic origin (MSEO).

Condensing osteitis

Condensing osteitis is an area of bone sclerosis in the periapical region that results from an inflammatory stimulus secondary to chronic pulpal inflammation or necrosis. It is most commonly diagnosed in children and young adults, but it can occur in older individuals. Compact bone replaces the fatty marrow, and there are variable levels of fibrosis consistent with an osteoblastic response to a low-grade stimulus.[99] Treatment is focused on the removal of the inflammatory stimulus, and Eliasson et al. reported 85% of these lesions resolve to some extent following root canal treatment or extraction of the compromised tooth.[68] This study is limited by a small sample size, and it is should be noted that regression of the dense bone did not correlate with the quality of the root canal fillings.

Radiographic features.

The true incidence of condensing osteitis in the population is unknown, as there is a small amount of literature on the condition, and historically it has likely been confused with idiopathic osteosclerosis due to similar radiographic appearances and clinical presentation. Condensing osteitis most commonly presents in the premolar and molar regions of the mandible.[76] The

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involved tooth exhibits a widened PDL space and is associated with a localized radiodense zone apical to a carious or heavily restored tooth (Fig. 3.11). The sclerotic bone is asymptomatic, but the involved tooth may be painful due to pulpal inflammation or necrosis.

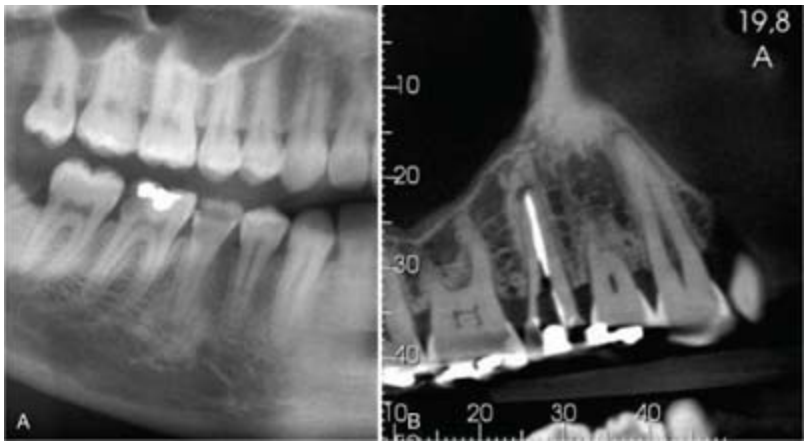

FIG. 3.11 **A,** Cropped panoramic image of condensing osteitis exhibiting a widened PDL space with a radiodense rim at the apex of a carious tooth #29. **B,** Sagittal CBCT image of maxillary premolar with previous endodontic treatment. A homogeneous high-density lesion is present with circumferential loss of density at the root apex suggesting an inflammatory etiology.

Set of two radiographs marked A and B depict

Source: (Courtesy A, Dr. Christel Haberland; B, Dr. Bruno Azevedo.)

Maxillary sinusitis of endodontic origin

MSEO is an inflammatory disease of microbial origin caused by a necrotic pulp or a root canal–treated tooth with posttreatment disease and may account for 10% of sinusitis cases.[169] The teeth most closely associated with the maxillary sinus include the second molar, first molar, and second premolar. Eberhardt et al. reported that the distance from the MB root of maxillary second molars to the sinus floor is less than 1 mm, while Tian et al. measured similar distances between root ends and the sinus floor but also found that this difference increases with age.[65,249] More than 40% of maxillary molars have a root that protrudes into the sinus.[125] Because of the close anatomical proximity of some root ends to the maxillary sinus and the thin plate of bone

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separating the roots from the sinus lining, an endodontic infection can cause bone destruction leading to a communication through the floor of the sinus. This communication can result in the accumulation of inflammatory mediators elevating the sinus lining or triggering an osteoblastic reaction. Other potential bacterial pathways as an etiology for sinusitis include periodontal disease, root fractures, extractions, and the placement of dental implants.

The sinus component is often asymptomatic and, although the pulpally involved tooth may be painful, the typical symptoms of an endodontic infection may not be obvious. Because MSEO results from a necrotic pulp or a tooth with posttreatment disease, there will be no thermal sensitivity. Swelling and percussion symptoms are less common due to the release of pressure into the sinus. Headache, facial pain, unilateral nasal obstruction, postnasal drip, and nasal discharge are clues to the presence of MSEO, especially when these symptoms are concurrent with a bacterial etiology of endodontic origin.[268,269]

In spite of a reported prevalence ranging from 1.5% to 14%, MSEO often goes undiagnosed, and the literature suggests dental infections contribute to a large percentage of maxillary sinusitis cases.[33,160,179,191] If MSEO is not diagnosed, patients may be erroneously prescribed multiple rounds of antibiotics or undergo unnecessary sinus surgeries due to chronic sinus infections. Treatment involves the elimination of the bacterial etiology with root canal treatment, endodontic surgery, or extraction.[183] Any attempt to directly treat the sinus with a surgical approach will be ineffective, as the source of the infection will not be directly addressed. Likewise, systemic antibiotic therapy may reduce symptoms temporarily, but will not result in a long-term cure.

Radiographic features.

MSEO is often difficult to identify with conventional imaging due to the superimposition of root ends, sinus, cortical plates, and the zygoma (Fig. 3.12). As a result, a high percentage of MSEO cases are missed with routine periapical radiographs.[184,246] A panoramic radiograph may exhibit a sessile, slightly radiopaque dome-shaped elevation. Limited field CBCT will detect a greater percentage of cases of MSEO than other imaging modalities, and two

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distinct patterns can be identified.[160]

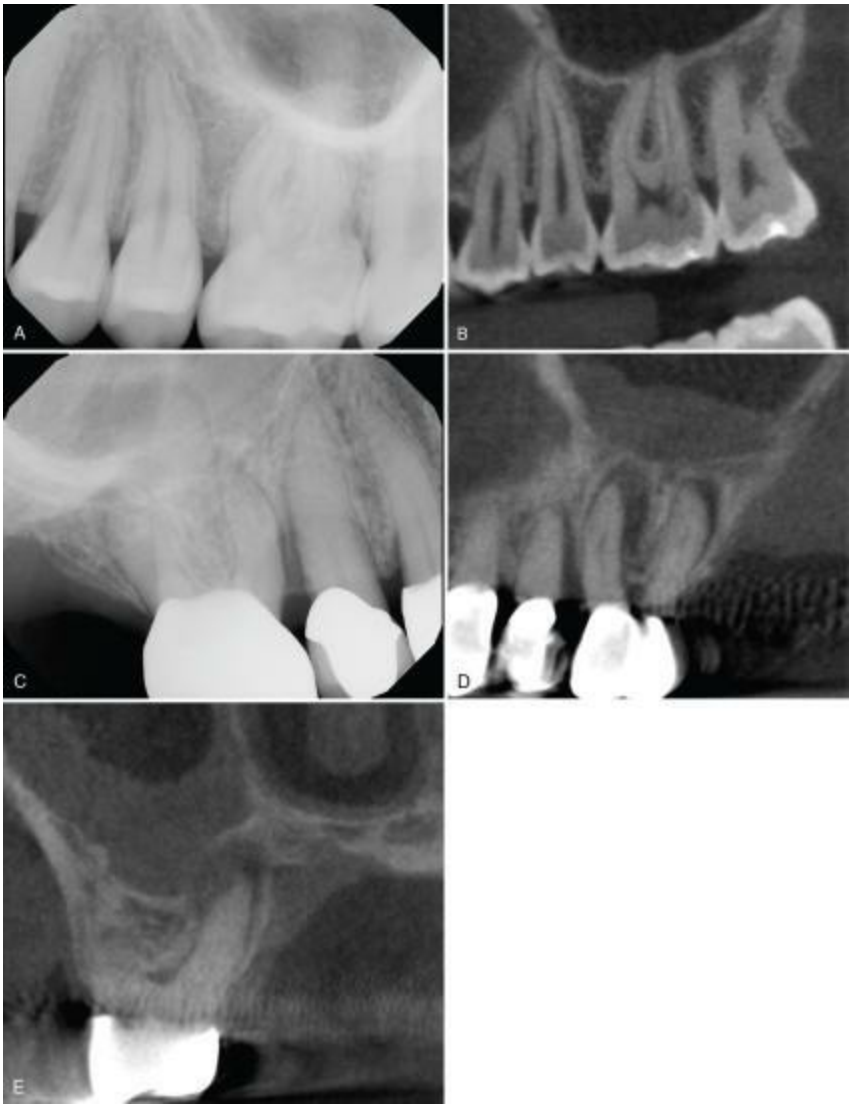

FIG. 3.12 **A,** Periapical image of #14 demonstrating cervical resorption and superimposition of the maxillary sinus over the palatal root apex. There is no way to determine the presence of inflammatory changes in the sinus based on this image. **B,** Sagittal CBCT image obtained because of resorption in first molar. Teeth in the field of view were asymptomatic and responded normally to pulp testing. There is no indication of mucosal thickening in the sinus. **C,** Periapical image of necrotic #3 with well-defined radiolucencies at the apices. **D,** Sagittal CBCT image confirms areas of low density at the root apices and reveals resorption of the maxillary sinus floor with localized mucosal thickening. **E,** Coronal CBCT image illustrates collateral edema into the sinus with a perforation of the sinus floor near the palatal root apex.

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Set of five radiographs marked A through E depict evaluation and results of superimposition of the maxillary sinus over the palatal root apex.

Periapical osteoperiostitis.

The inflammatory process from an odontogenic infection can expand the sinus periosteum and displace it superiorly into the sinus. Periapical osteoperiostitis is the osteoblastic reaction that results in new bone formation under the periosteum as it is elevated vertically, resulting in a dome-shaped radiopacity on the sinus floor.[246]

Periapical mucositis.

Periapical mucositis occurs when odontogenic infections displace the sinus lining without the reactive osteoblastic changes. This will also present as a dome-shaped lesion radiographically, but because the mucosal thickening and tissue edema is limited to the soft tissues, periapical mucositis is more difficult to identify than periapical osteoperiostitis (Fig. 3.13). This type of radiographic change has also been termed *antral pseudocyst*.[90]

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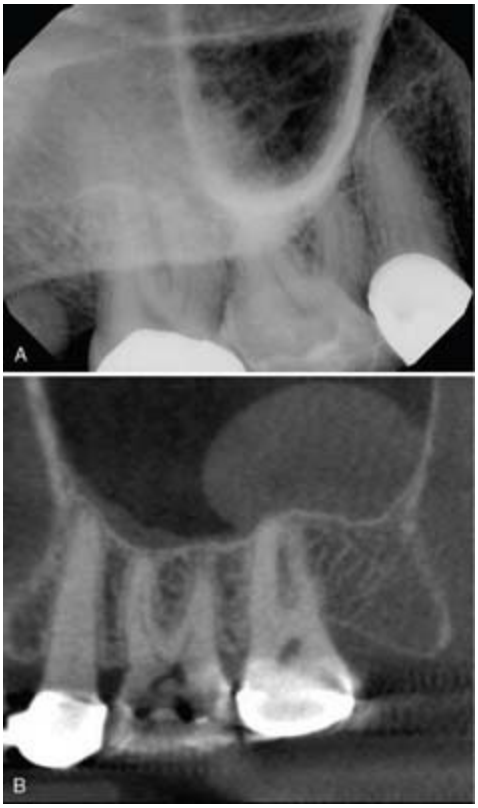

FIG. 3.13 **A,** Periapical image of #2. This tooth is nonresponsive to thermal testing and painful to percussion. Radiographically there is a slightly increased density in the maxillary sinus near the palatal root apex, but superimposition of normal anatomical structures limits the diagnostic accuracy of the image. **B,** Sagittal CBCT image reveals a homogeneous dome-shaped elevation into the maxillary sinus.

Set of two radiographs marked A and B depict antral pseudocyst.

Osteomyelitis with proliferative periostitis

Proliferative periostitis is a reactive bone formation in response to the presence of inflammation similar to periapical osteoperiostitis that occurs with MSEO. However, the reactive bone is deposited in parallel rows expanding the surface of the involved jaw, usually in a child or adolescent.[179] It is thought that subperiosteal spread of an inflammatory process elevates the periosteum, stimulating new bone formation.[266] Dental caries with a secondary odontogenic infection is the most common cause. Like condensing osteitis, most cases arise in the premolar and molar area of the mandible, but unlike condensing osteitis, there is obvious expansion, most often of the

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lower border of the mandible and buccal cortical plates.[128] Removal of the inflammatory stimulus via appropriate endodontic treatment or extraction should result in bone healing over a 6 to 12 month time frame, but biopsy should be considered if bone expansion is present and/or a dental source of infection cannot be identified with clinical examination and pulp testing.

Radiographic features.

Because of superimposition of the lesion over normal bone, proliferative periostitis may be difficult or impossible to visualize with conventional intra-oral radiography (Fig. 3.14). A CBCT or an occlusal radiograph will demonstrate a variable number of layers of reactive bone.[252,274] The boney sheets are typically parallel to each other and the cortical plate. Numerous small radiolucent areas may be identified within the body of the lesion.[66]

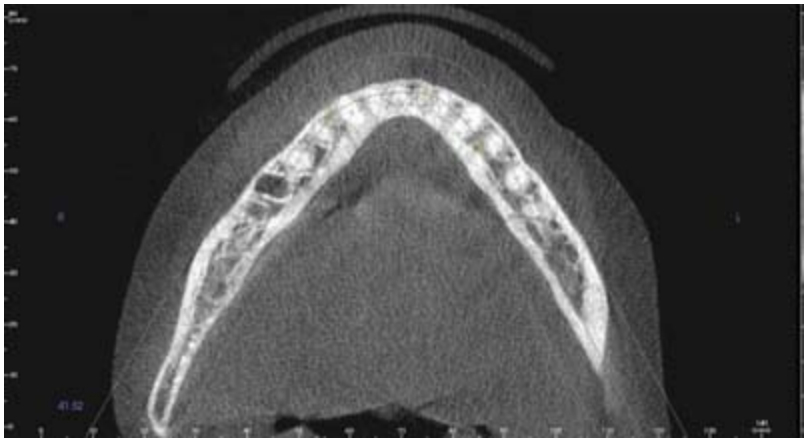

FIG. 3.14 Axial CBCT image of mandibular arch demonstrates irregular medullary bone destruction and cortical defects from tooth #25 to tooth #31. There is new bone formation along the inferior facial cortex consistent with osteomyelitis and proliferative periostitis.

CBCT images shows teeth forming an inverted U-shape. A radiolucent cavity in place of the tooth is shown toward the center with a cloudy tissue on one side. The left side of the radiograph shows extremely thin tissue.

Source: (Courtesy Dr. Laurence Gaalaas.)

Although a radiopacity may be the result of an osteoblastic reaction to an inflammatory stimulus, if pulp testing and clinical history do not indicate an endodontic etiology, other disease entities should be considered in the

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differential diagnosis. These radiopacities may occur for idiopathic reasons, be developmental in nature, be the result of an underlying bone disorder, or represent variations of normal.

Idiopathic osteosclerosis

Idiopathic osteosclerosis is typically an incidental radiographic finding characterized by an increased bone density of unknown etiology. The prevalence is estimated to be approximately 5%, but due to inconsistent nomenclature in the literature and the similarity in clinical presentation to condensing osteitis, the true prevalence is unknown.[93,129,198] Idiopathic osteosclerosis is most commonly found in adolescents and may remain unchanged or continue to enlarge slightly until normal growth and development is complete.[198] At that point, the majority of lesions will remain unchanged, but a small number may resolve.[104] The diagnosis of idiopathic osteosclerosis is based on clinical presentation of the lesion, pulp testing of teeth in the area, and restorative history. If any of the clinical findings are suspicious, a biopsy could be done, but typically the diagnosis can be made based on the clinical characteristics and no treatment is indicated.

Radiographic features.

Idiopathic osteosclerosis is identified as an asymptomatic, well-defined, nonexpansile radiopacity (Fig. 3.15). The size of the lesion varies from a few millimeters up to 2 cm, and the outline is often irregular. Like condensing osteitis, it is most commonly located in the first molar region, and there is an overwhelming predilection for the mandible.[198,272] It typically presents as a solitary lesion of uniform radiodensity, but multiple radiopacities have been reported in the same patient.[272] Idiopathic osteosclerosis usually occurs near a root apex and root resorption is possible, but uncommon.

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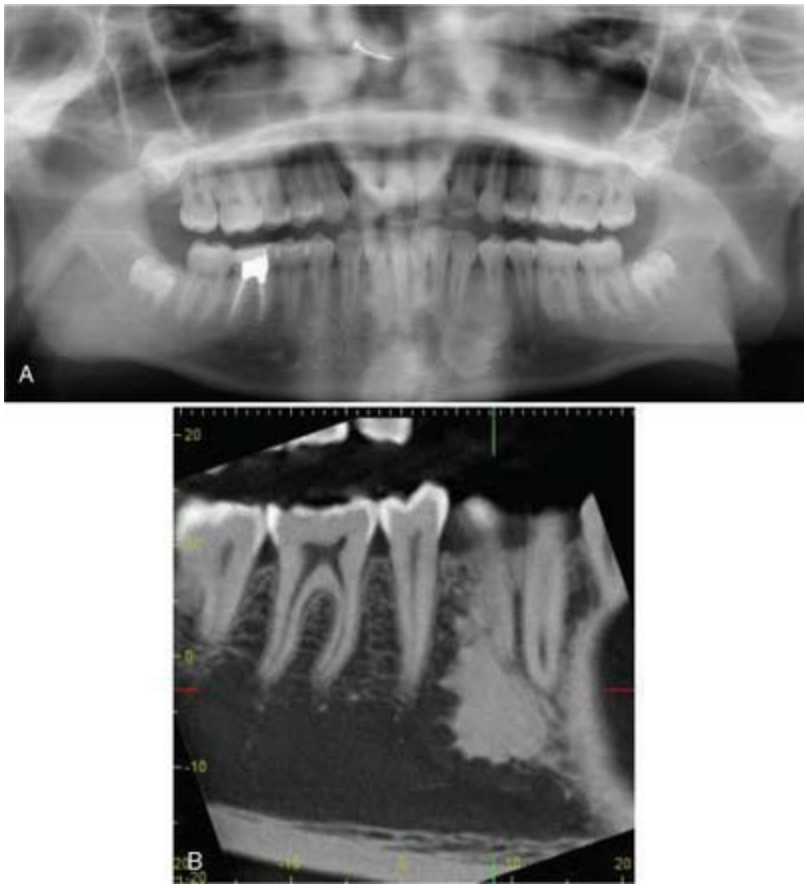

FIG. 3.15 Idiopathic osteosclerosis. **A,** A cropped panoramic image with a well-defined alteration in the trabecular pattern apical to teeth #20 to 22 in a 15-year-old female. Peripherally there is slight cortication with no evidence of root resorption. **B,** Sagittal CBCT image showing a homogeneous area of hyperdensity near the canine and premolar root apices. The lesion has an irregular outline, but there is no indication of root resorption.

A) CBCT image shows dense tissue below the teeth at the center. The dense tissue is surrounded by radiolucent tissue on the left and right.

B) CBCT image shows clustered dense tissue near the premolar and canine. Teeth shows radiolucent canals.

Source: (Courtesy A, Dr. Laurence Gaalaas; B, Dr. Bruno Azevedo.)

Odontoma

Odontomas are common developmental abnormalities and should not be considered neoplasms. They are composed of tissues found in normal teeth, including enamel, dentin, and variable amounts of pulp and cementum.

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Compound odontomas are made up of multiple structures that resemble teeth and are most often located in the anterior maxilla, while complex odontomas are composed of an unorganized aggregate of enamel and dentin and are most often diagnosed in the molar regions.[37] However, both types will occasionally be found in any maxillary or mandibular site and in the periapical areas.[200] If the diagnosis can't be made based on radiographic findings, surgical removal has an excellent prognosis.

Radiographic features.

Most odontomas are diagnosed before the age of 20 as asymptomatic findings on routine radiographs.[245] Often they are identified when a normal tooth fails to erupt. They are usually smaller than a tooth, but larger odontomas of greater than several centimeters have been reported.[245] Compound odontomas resemble teeth radiographically and are surrounded by a radiolucent border (Fig. 3.16). Complex odontomas have the same radiodensity as teeth and are also associated with a radiolucent border but lack the shape of normal teeth (Fig. 3.17). An early odontoma could resemble a periapical radiolucency if it appears near a root apex prior to calcification.

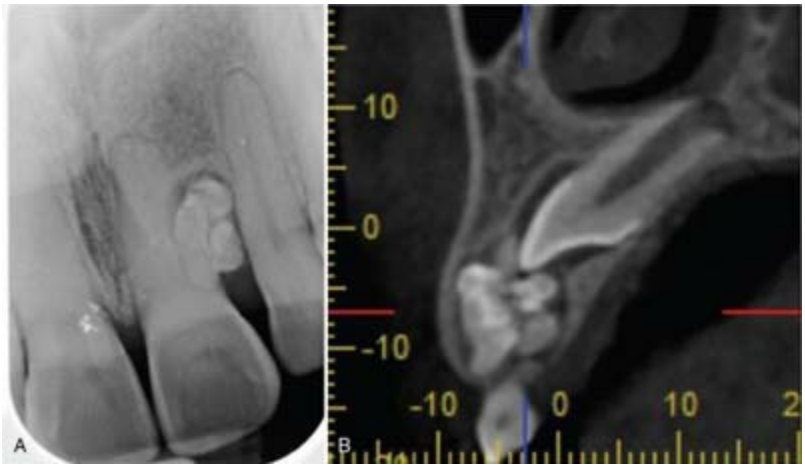

FIG. 3.16 **A,** Periapical image of a compound odontoma in a 35-year-old female. Multiple radiopacities resembling teeth are located interproximal to the maxillary central and lateral incisors. **B**, CBCT image demonstrating multiple high-density toothlike structures in association with a corticated low-density zone resembling dental follicle.

Set of two radiographs marked A and B depict changes observed in compound

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odontoma.

Source: (*A,* Courtesy Dr. Christel Haberland. *B,* Courtesy Dr. Bruno Azevedo.)

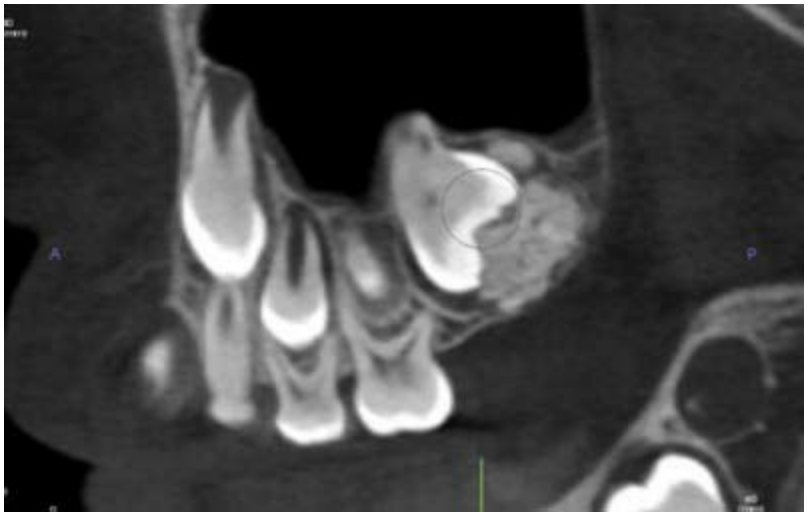

FIG. 3.17 Complex odontoma. Sagittal CBCT image with a high-density globular mass near the crown of a maxillary first molar. The mass exhibits the density of dentin.

CBCT image shows tooth-like additional structures below the teeth. Below the first molar a thick proliferated tissue is present.

Source: (Courtesy Dr. Bruno Azevedo.)

Paget disease of bone

Paget disease is a common metabolic bone disorder characterized by a highly variable and mixed radiographic presentation due to the abnormal and haphazard deposition and resorption of bone. A defect in osteoclasts, and to some degree osteoblasts, results in thickened, enlarged, and weakened bones.[224] It is rare before the age of 40 and tends to affect older white adults with a prevalence that is geographically variable.[179] The highest incidence is found in people of British ancestry, and the prevalence in the United States is estimated to be 1% to 2%.[179] Paget disease has an unknown etiology, and both genetic and environmental factors may be involved. A family history has been reported in as high as 40% of cases, and the decline in overall

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prevalence over the last 25 years suggests a possible link to a paramyxovirus.[46]

Paget disease is often asymptomatic at the time of diagnosis, but bone pain is not uncommon.[154] Test results indicate an elevated level of serum alkaline phosphatase with normal blood calcium and phosphorus levels. However, alkaline phosphatase is not always abnormal in patients with limited disease. Asymptomatic patients with limited involvement require no treatment and bisphosphonates are used for symptomatic patients to reduce bone turnover and decrease pain. Paget disease tends to be a chronic and slowly progressive disease, but malignant transformation to osteosarcoma is possible.[41]

Radiographic features.

Paget disease typically arises from several bones at the same time. The pelvis, femur, lumbar vertebrae, tibia, and skull are commonly involved.[205,224] The jaws are involved in less than 20% of cases and the maxilla is twice as likely to be affected compared to the mandible. Maxillary involvement is characterized by an enlargement of the midface, and symptoms may include a deviated septum, nasal obstruction, and sinus obliteration. Additionally, enlarged alveolar ridges resulting in space between teeth can occur in either jaw.

Early in the disease process there is decreased radiodensity with a coarse trabecular pattern (Fig. 3.18). During this early resorptive stage, the lesions are very vascular resulting in significant bleeding during oral surgical procedures. Following the resorptive changes, patchy areas of confluent bone sclerosis become more prominent.[39] These sclerotic areas have classically been described as having a “cotton wool” radiographic appearance and may exhibit poor wound healing and an increased risk of osteomyelitis following surgery.[22] Teeth in affected areas may develop hypercementosis with radiographic features similar to cemento-osseous dysplasia.

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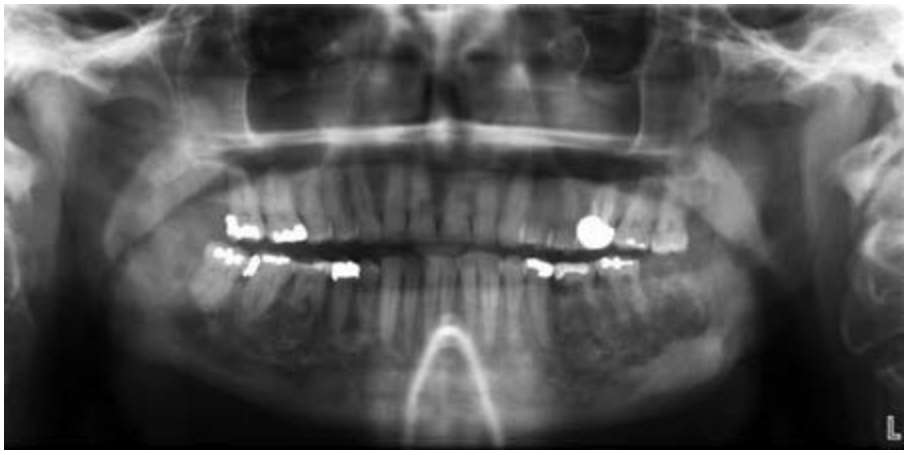

FIG. 3.18 Panoramic radiograph demonstrating Paget disease of bone. A multifocal presentation involving both right and left mandibular molar areas. The coarse trabecular pattern and mixed radiolucency is common to Paget disease. There is significant root resorption, but the typical hypercementosis is absent.

Radiograph shows inverted U-shape radiopaque outlining below the teeth at the center with slightly radiolucent tissue inside.

Source: (Courtesy Dr. Carl Allen.)

Exostoses and tori

An exostosis is a boney outgrowth from the cortical plate. There is a high degree of uncertainty in the literature about the cause and prevalence of exostoses, and they should be considered variations of normal anatomy that have a multifactorial etiology.[86,108,171,189] An exostosis is a relatively common finding on routine clinical examination, but there is minimal literature on the topic and the prevalence within the population is unknown. The most common exostoses are the torus mandibularis and torus palatinus.[179] Exostoses are usually asymptomatic unless trauma results in localized inflammation or ulceration. Treatment is usually unnecessary with the classic clinical presentation. Removal of sizable exostoses may be needed for normal function or for prosthetic reasons.

Radiographic features.

In some cases, the bone density will be high enough that an exostosis will be seen as a radiopacity on dental radiographs. It is rare to see a palatal torus on dental radiographs, but it is not uncommon to visualize a mandibular torus

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superimposed on the roots (Fig. 3.19).

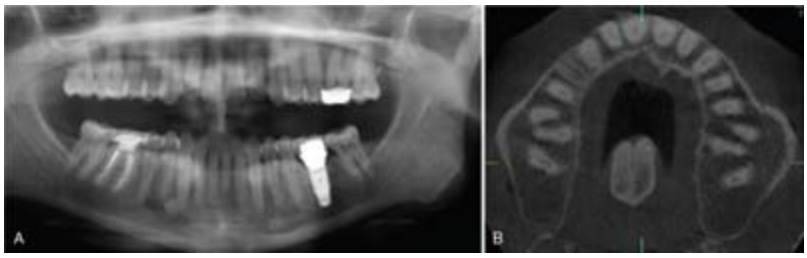

FIG. 3.19 **A,** A radiodense ridge superimposed over the coronal portion of the roots extending from the implant in the #19 position to the mesial of tooth #30. This represents a prominent lingual exostosis and should not impact endodontic diagnosis but could interfere with visualization of the canals radiographically. This panoramic image also demonstrates well-defined uniform radiopacities inferior to tooth #21 and tooth #27 consistent with idiopathic osteosclerosis. **B,** Axial CBCT image of a maxillary torus characterized by a well-defined high-density area in the midline.

A) Panoramic radiograph shows a radiopaque tooth on the left. The space between upper and lower rows of teeth has fibrous tissues at the center. Crown and root canals of one tooth are radiopaque on the right in the lower jaw. The upper jaw shows tooth with radiopaque crown.

B) Radiograph of maxillary arch shows two fused tooth-like structures at the center below the teeth.

Multifocal presentations

Although it is not common to have asymptomatic radiolucencies resulting from simultaneous pulpal necrosis in multiple teeth, it can’t be ruled out on radiographic examination alone. Pulp testing and dental history are important if multiple necrotic pulps are suspected (Fig. 3.20), but a multifocal presentation also raises concerns about a nonbacterial etiology.

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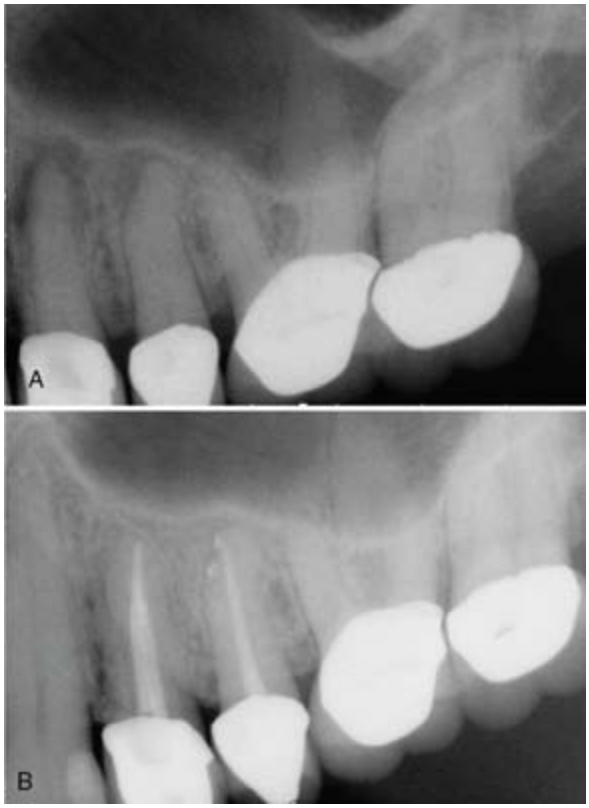

FIG. 3.20 **A,** Multiple periapical radiolucencies suggest the possibility of a noninflammatory etiology. However, teeth #12 and #13 did not respond to cold testing and both teeth were tender to percussion and palpation. The diagnosis for tooth #12 and tooth #13 was pulpal necrosis and symptomatic apical periodontitis. Nonsurgical root canal treatment was completed in both teeth. **B,** A 6-month recall demonstrated bone healing at the root ends of both teeth.

A) Radiograph shows large dark blotch next to roots of teeth. Two teeth on the right have radiolucency at the root tip.

B) Radiograph shows two teeth on the right with filled root canals.

Periapical cemento-osseous dysplasia

Periapical cemento-osseous dysplasia (PCOD) is the most common multifocal radiolucency to mimic lesions of endodontic origin and has been known by a variety of terms in the literature including, but not limited to, periapical cemental dysplasia, cementoma, and periradicular fibrous dysplasia.[178,227] Like the other fibro-osseous lesions (Box 3.2), PCOD is characterized by the replacement of bone with fibrous and mineralized tissue. PCOD occurs in the anterior mandible and has a predilection for black

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females.[8,179] The diagnosis is most often made between 30 and 50 years of age and it is uncommon prior to the age of 20. The teeth in the area are usually asymptomatic, and the diagnosis is most often made from routine radiographs.[227] The teeth are often unrestored and should respond normally to pulp testing. Treatment is not necessary unless the area is symptomatic.[159] If there is uncertainty about the diagnosis, a biopsy can be obtained, but this comes with an increased risk for infection, bone necrosis, or a disruption in the pulpal blood supply.[15]

Box 3.2

Fibro-Osseous Lesions

Periapical cemento-osseous dysplasia
Fibrous dysplasia
Focal cemento-osseous dysplasia
Florid cemento-osseous dysplasia
Ossifying fibroma

Radiographic features.

The radiographic presentation of PCOD is dynamic, variable, and changes as the patient ages.[130,218] Early lesions are mostly radiolucent and will have a strong resemblance to the osteolytic changes that result from pulpal necrosis (Fig. 3.21). As a result, misdiagnosis of PCOD will commonly result in unnecessary root canal treatment. As the lesions change with time, they will take on a mixed radiolucent radiopaque appearance radiographically, and late stage PCOD is characterized by well-circumscribed radiopacities with a well-defined radiolucent border (Fig. 3.22). A multifocal presentation is most common, but solitary lesions that are indistinguishable from periapical cysts or periapical granulomas have been reported.[179] The PDL space is usually unaltered, and even late stage lesions will rarely exceed 1.0 cm.

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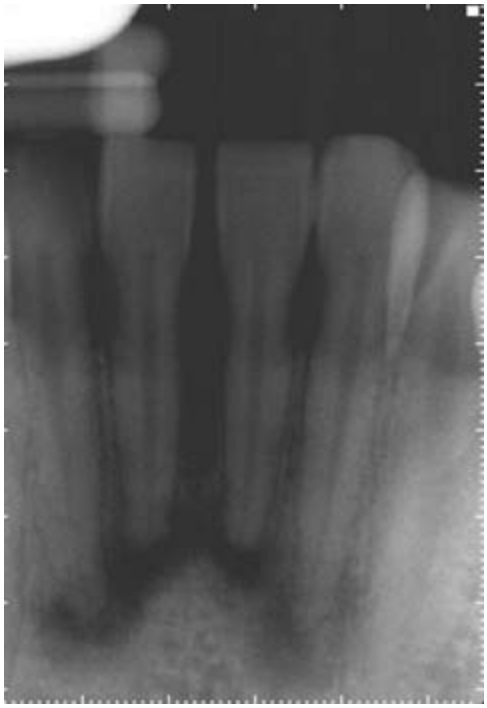

FIG. 3.21 Multiple well-defined radiolucencies in the anterior mandible. The patient reported no history of trauma and was asymptomatic. These teeth had no significant dental history and responded to both cold and electric pulp testing. Based on this clinical presentation, a diagnosis of periapical cemento-osseous dysplasia was made and no treatment was indicated.

Radiograph shows radiolucent root canals in teeth. Beside this, root tips show a dark patch.

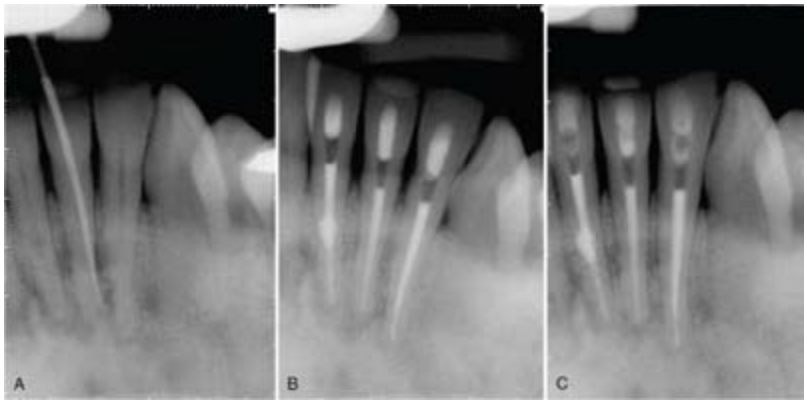

FIG. 3.22 **A,** A 55-year-old female presented with pain, a sinus tract facial to #24, and a history of biopsy in the area several years prior. Teeth #23, #24, and #25 did not respond to cold or electric pulp testing. Radiographically the mixed radiolucent and radiopaque lesions were

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consistent with periapical cemento-osseous dysplasia, but the sinus tract and pulp testing indicated pulpal necrosis in teeth #23, #24, and #25. Pulpal necrosis was likely due to a disruption of the pulpal blood supply at the time of the biopsy. **B,** Nonsurgical root canal treatment was completed and there was no indication of vital pulp tissue in the canals. The previous biopsy report was obtained prior to root canal treatment, and the diagnosis of periapical cemento-osseous dysplasia was confirmed. **C,** At the 6-month recall the sinus tract had healed and the patient was asymptomatic. Because the radiographic lesions were not the result of a bacterial etiology, there was no expectation to see a significant change in the bone at the 6-month recall.

A) Radiograph shows two teeth with radiolucent root canals. Tooth at the center of both teeth undergoes root canal treatment.

B) Radiograph shows three teeth with filled root canals. At the neck region, root canals are radiolucent. The tooth on the right shows a circular mass globule at the center of root canal.

C) Radiograph similar as that of B, except that there are oval radiolucent patches below crown in the first and third teeth.

Florid cemento-osseous dysplasia

Florid cemento-osseous dysplasia (FCOD) most commonly exhibits bilateral, multifocal involvement of the mandible, and all four quadrants can be affected. However, there is no abnormality in blood chemistry.[27,56,233] The lesions can occur in the anterior mandible, but unlike PCOD, the lesions are not limited to the anterior region. Similar to PCOD, there is a predilection for black females, but patients tend to be older at the time of initial diagnosis.[12,56] FCOD can be asymptomatic and discovered on routine radiographs, but pain and exposed bone are also possible.[56,158,222] Because the bone lesions possess a diminished vasculature compared to normal bone, these areas are susceptible to osteomyelitis and the formation of boney sequestra. Tooth extraction and root canal treatment have been reported to initiate secondary infection and symptoms in FCOD.[221] Generally, no treatment is indicated in the asymptomatic patient and the diagnosis can be made based on radiographic findings, clinical examination, and pulp testing.[55] If secondary infection occurs with symptoms, treatment options include antibiotics and surgery.

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Radiographic features.

FCOD exhibits a similar radiographic maturation to that of PCOD. Early lesions are radiolucent and, as they evolve, a mixed radiolucent and radiopaque presentation is common (Fig. 3.23). As patients age, FCOD lesions become more uniformly radiopaque with a thin radiolucent border. These lesions may be well defined, but they are often ill defined, blending with the normal bone.[56,233] An axial computed tomography (CT) will show high-density masses surrounded by a low-density layer with possible cortical expansion but no perforation of the cortical plate.[12,27] Unlike PCOD, FCOD can fuse with the root apex and blend with the normal bone.[12] A single periapical radiograph may suggest the diagnosis, but a panoramic image would be needed to demonstrate the multiquadrant involvement characteristic of the process.

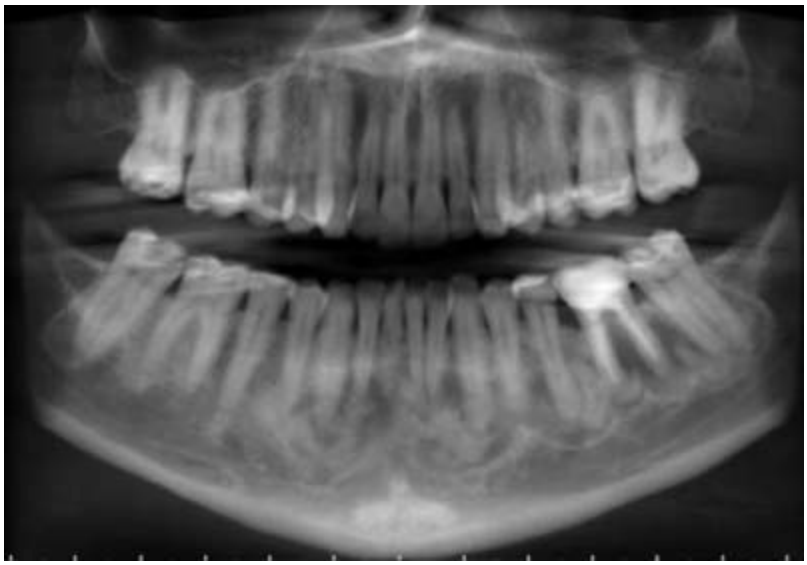

FIG. 3.23 Florid cemento-osseous dysplasia. A mixed radiolucent and radiopaque presentation with multiquadrant involvement. These teeth should respond normally to pulp testing unless there is a history of previous endodontic treatment. If a radiolucent area is present near the root apex of a previous root canal, it may be impossible to determine if the bone loss is due to a bacterial etiology or a manifestation of florid cemento-osseous dysplasia.

Radiograph shows mixture of radiolucent and radiopaque tissues in the jaw bone next to teeth.

Source: (Courtesy Dr. Bruno Azevedo.)

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Hyperparathyroidism

Parathyroid hormone (PTH) is normally produced due to decreased levels of serum calcium. Primary hyperparathyroidism results from an uncontrolled overproduction of PTH and is most often due to a parathyroid adenoma. Primary hyperparathyroidism is usually asymptomatic and can be found with routine testing. These patients have an elevated risk for kidney stones due to elevated levels of serum calcium. Additionally, there is an increased likelihood for the development of duodenal ulcers. Primary hyperparathyroidism is treated by the surgical removal of the parathyroid adenoma or hyperplastic tissue responsible for the excess PTH production.[179,201]

Secondary hyperparathyroidism develops due to chronic overproduction of PTH in the presence of low serum calcium resulting from chronic renal disease. A dietary and pharmacological approach is initially utilized to treat secondary hyperparathyroidism. If this is not successful, the parathyroid glands can be removed or a renal transplant may restore normal vitamin D metabolism.[47]

Radiographic features.

Hyperparathyroidism is characterized by a generalized loss of lamina dura around roots of teeth. Alterations in the trabecular pattern of bone resulting in a “ground glass” appearance tends to be more striking in patients with secondary hyperparathyroidism, and this may be associated with significant bone enlargement clinically.[206] Secondary hyperparathyroidism has also been associated with pain and increased cortical bone density.[3] Additionally, lytic lesions resembling periapical inflammatory disease can occur (Fig. 3.24).[120,155]

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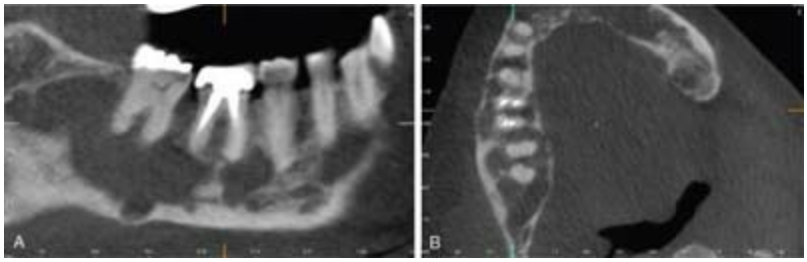

FIG. 3.24 Hyperparathyroidism. **A,** Sagittal CBCT image with multiple low-density lesions and changes in mandibular bone density. **B,** Axial CBCT image showing a thinning of the cortical plates in association with the low-density lesions.

A) CBCT image shows dark patches below the roots in the mandibular quadrant. The molar at the end has radiopaque enamel. The next molar shows radiopaque crown and partially radiopaque root canals.

B) CBCT image shows mandibular arch with radiolucency in the cortical plates. The cortical plates at the right are expanded.

Source: (Courtesy Dr. Marcel Noujeim.)

The brown tumor of hyperparathyroidism may develop with long-standing disease, especially in female patients with primary hyperparathyroidism.[3] It presents as a well-defined unilocular or multilocular radiolucency. These lesions can be single or multiple and may be located in the mandible, clavicles, ribs, and pelvis.[179,201] A single, well-defined, unilocular brown tumor of hyperparathyroidism on a periapical radiograph would be indistinguishable from a periapical cyst or a periapical granuloma.

Langerhans cell histiocytosis

Langerhans cell histiocytosis (LCH) has historically been known by several designations (histiocytosis X, eosinophilic granuloma), and there is controversy about its status as a reactive process or a true neoplasm because LCH also contains a diverse inflammatory infiltrate of eosinophils, lymphocytes, and plasma cells. Additionally, LCH is characterized by a wide spectrum of clinical presentations not typical of a neoplastic process, ranging from single organ involvement that may not require treatment to disseminated and life-threatening systemic disease. However, genetic studies have shown a mutation in an oncogene associated with abnormal cell proliferation in more than 50% of LCH cases, suggesting a true neoplasm.[14]

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Langerhans cells normally act as antigen presenting cells to T lymphocytes and are located in skin and mucosa. Historically, LCH was thought to arise from these cells due to the presence of Birbeck granules using electron microscopy and immunoreactivity to specific cell surface markers. More recent research has shown that Birbeck granules are not exclusive to Langerhans cells. Currently, there is debate regarding the actual cell of origin that results in LCH with some suggesting that immature myeloid dendritic cells of bone marrow origin are the causative cells.[7,25] Because of its diverse presentation, standardized treatment protocols are limited and ultimately treatment and prognosis are determined by the location and extent of the disease.

Radiographic features.

Patients present over a wide age range, but the majority are diagnosed before the age of 15.[179] LCH typically presents as single or multiple punched out radiolucencies with well-defined borders, but an ill-defined margin is also possible.[50,112] A solitary well-defined lesion on a periapical radiograph may be indistinguishable from a periapical cyst or periapical granuloma, but the LCH lesion may be more destructive.[196] LCH can have a widespread distribution and affect any bone, but jaw involvement is more common than soft-tissue involvement in the maxillofacial region, and the mandible is affected three times more than the maxilla.[112,179] LCH can result in the loss of alveolar bone and the loosening of teeth, resembling severe periodontal disease (Fig. 3.25).

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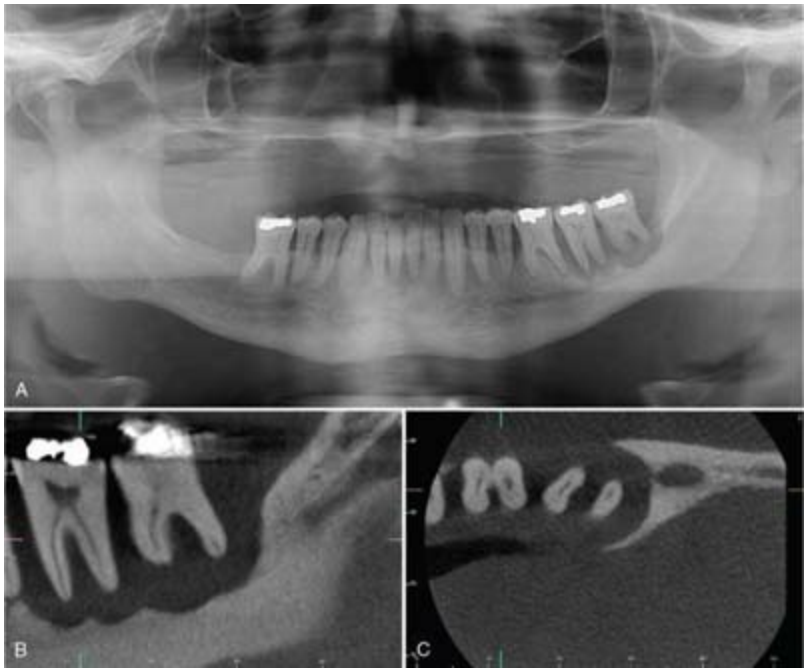

FIG. 3.25 Langerhans cell histiocytosis. **A,** Reformatted panoramic image with destructive radiolucent area in mandibular molar region demonstrating "floating" teeth in the #17 and #18 area. **B,** Sagittal CBCT image confirming the aggressive nature of the lesion. **C,** Axial CBCT image revealing destruction of both buccal and lingual cortical plates.

A) Panoramic radiograph shows teeth in the lower jaw with a trabecular pattern below the roots.

B) CBCT image shows extremely low-density bones around and next to roots.

C) CBCT image shows poorly aligned teeth with perforated buccal and cortical plates.

Source: (Courtesy Dr. Marcel Noujeim.)

Ill-defined pathologies

At first glance, the peripheral demarcation of a radiolucency may not seem important because periapical cysts and periapical granulomas are the most common ill-defined and well-defined radiolucencies. The differential diagnoses for ill-defined radiolucencies is more ominous and as a result, if pulp testing is not confirmed by the clinical findings at the time of root canal treatment, further investigation and referral would be indicated (Box 3.3). For example, a necrotic pulp would be expected in a tooth with a significant

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caries history that is nonresponsive to pulp testing and painful to percussion. However, if vital pulp is identified and the patient's symptoms do not resolve following root canal treatment, a biopsy would be recommended if the radiolucent area is ill defined and there is evidence of root resorption, tooth mobility, bone expansion, or perforation of the cortical plates.

Box 3.3

Uncommon Ill-Defined Radiolucencies

- Osteosarcoma
- Chondrosarcoma
- Ewing sarcoma
- Fibrosarcoma
- Odontogenic carcinomas
- Salivary gland neoplasms
- Osteoradionecrosis
- Desmoplastic fibroma

Osteomyelitis

Osteomyelitis is an uncommon acute or chronic inflammatory process that spreads through the bone away from the initiating site of infection. In acute cases, symptoms usually present within 2 weeks of the infection while it may take more than 6 weeks to develop symptoms with chronic osteomyelitis. Most commonly, a polymicrobial bacterial infection composed of organisms indigenous to the oral cavity serves as the precipitating factor leading to inflammatory bone destruction and the formation of bone sequestra, which are fragments of necrotic bone that are displaced from the viable bone.[140,242] Less commonly, a fungal or mycobacterial infection initiates the osteomyelitis. Predisposing factors include chronic systemic diseases, an immunocompromised state, or any condition that impacts the vascularity of bone. The incidence of chronic osteomyelitis may be increasing due to an increased prevalence of diabetes and peripheral vascular disease in the aging population as well as the increased use of fixation devices in orthopedic surgery.[106] The mandible is more commonly involved due to its more limited vascular supply. Osteomyelitis can occur at any age, and there is a strong

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male predilection.[140]

Acute osteomyelitis.

Acute osteomyelitis is characterized by the spreading of infection and inflammation through the bone. Patients typically have the signs and symptoms of acute infection, including fever, lymphadenopathy, tenderness to palpation, and elevated white blood cell counts.[242] Paresthesia of the lower lip is possible due to nerve compression, and the extent of swelling is dependent on how the infection spreads.[140] Treatment consists of surgical intervention to remove the source of infection, promote drainage, and remove any infected or necrotic bone. Antibiotics and multiple surgeries may be needed to resolve the infection in some cases, and the emergence of antibiotic-resistant bacteria has served to complicate treatment in an increasing number of cases.[64]

Radiograph changes associated with acute osteomyelitis can be subtle as the infection spreads through the medullary spaces.[124] Periapical images may reveal an ill-defined radiolucency with widened PDL spaces and the loss of lamina dura. As expected, a CBCT will often show the spread of the infection more clearly than conventional dental imaging and, although not practical in the dental setting, magnetic resonance imaging (MRI) can demonstrate edema and inflammatory changes in the marrow spaces characteristic of acute osteomyelitis.[11,13,124] If the infection spreads under the periosteum, there can be an osteoblastic reaction leading to new bone formation with radiopacity or an ill-defined lesion of mixed radiodensity.[105,242]

Chronic osteomyelitis.

Chronic osteomyelitis is the result of an unresolved acute infection causing the inflammatory response to form a layer of granulation tissue around the bacteria. The granulation tissue forms a space that is difficult to access with antibiotics and also serves as a reservoir for bacteria. As seen in acute osteomyelitis, pain is a common presenting symptom.[242] However, swelling and drainage are more likely to be present with chronic osteomyelitis. In advanced cases, tooth mobility and significant bone loss are possible prior to pathologic fracture. Management of chronic osteomyelitis is more difficult due to the pockets of dead bone and infection that are isolated behind the

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granulation tissue wall.[132] Surgery to remove all infected and necrotic bone is mandatory, and jaw resection may be necessary in some cases. Systemic or intravenous (IV) antibiotics are used in conjunction with surgery. Hyperbaric oxygen has been used in extreme cases, but this is typically not needed if all infected bone has been removed.

The radiographic presentation of chronic osteomyelitis is variable, depending on the duration and extent of the infection and the differing amounts of bone destruction.[273] Chronic osteomyelitis is often characterized by an irregular, ragged, and ill-defined radiolucency. The radiolucent area may contain sequestra and zones of increased density resulting in a mixed radiolucent and radiopaque appearance (Fig. 3.26).[273] Large areas of bone necrosis are possible if blood supply is compromised, and the destructive "moth eaten" nature of some lesions suggests the potential for a malignant neoplasm (Fig. 3.27).[105,116]

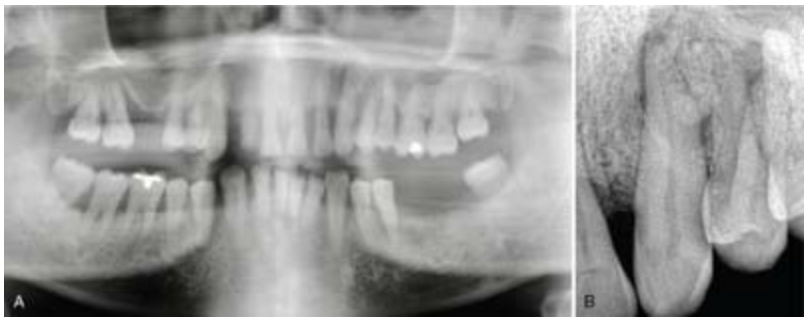

FIG. 3.26 **A,** 50-year-old male with a complaint of increasing pain and swelling from the maxillary left canine region over the last year. Examination revealed a palatal swelling and radiographically there was an ill-defined radiolucent lesion near teeth #11 and #12. **B,** A periapical image with a mixed radiolucent and radiopaque lesion is present, but the margins are more well-defined than on the panoramic image. A biopsy was done to rule out a salivary gland neoplasm due to the palatal swelling and destructive nature of the radiolucency. The biopsy revealed both acute and chronic inflammation with sequestrum formation and bacterial colonies consistent with a diagnosis of osteomyelitis.

A) Panoramic radiograph shows poorly-aligned teeth with space in lower left quadrant.

B) Radiograph shows upper teeth with perforated cells.

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Source: (Courtesy Dr. Laurence Gaalaas.)

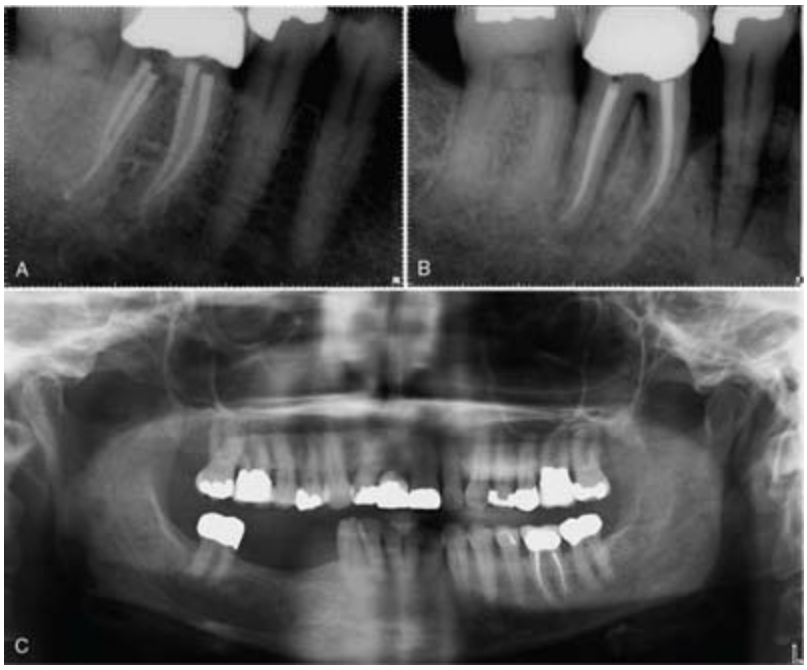

FIG. 3.27 **A,** Nonsurgical root canal treatment completed in tooth #30 due to pulpal necrosis. **B,** A 1-year recall due to increasing pain in the area. Radiographs revealed an ill-defined radiolucency associated with teeth #28, #29, and #30. These teeth exhibited abnormal mobility and there was significant soft-tissue inflammation, gingival bleeding, and exposed bone. The destructive nature of the bone loss and clinical presentation suggested malignancy, but a biopsy revealed sheets of chronic inflammatory cells and necrotic bone consistent with osteomyelitis. **C,** A panoramic image reveals complete osseous healing at 4-year recall.

A) Radiograph shows molar with radiopaque crown and filled root canals.

B) Radiograph shows three teeth. First tooth has radiopaque edges at the top. Second tooth has radiopaque crown and filled root canals. Some part of the crown is radiopaque in third tooth and its root canal is radiolucent. All three teeth are poorly fitted.

C) Panoramic radiograph shows properly fitted teeth. Most of the teeth are missing in lower right quadrant.

Medication-related osteonecrosis of the jaw

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Bisphosphonate osteonecrosis of the jaw was first reported when an association was identified between the use of intravenous bisphosphonates and an increased frequency of jaw necrosis.[162,213] However, since these initial reports, other drug classes have been implicated and the name was changed to reflect this in 2014.[214] New medications are frequently being added to the list of causative agents (Table 3.1), and osteonecrosis has been reported in other osseous sites including the small bones of the inner ear and femoral head.[92,166,211]

Table 3.1

Other Medications Implicated in Pathogenesis of Medication-Related Osteonecrosis of the Jaw

Generic Name	Trade Name
Imatinib	Gleevec
Regorafenib	Stirvarga
Axitinib	Inlyta
Pazopanib	Votrient
Cabozantanib	Cometriq
Dasatinib	Sprycel
Methotrexate	Otrexup, Trexall
Corticosteroids	
Adalimumab	Humira
Infliximab	Remicade
Rituximab	Rituxan
Romosozumab	Evenity
Aflibercept	Zaltrap
Everolimus	Afinitor
Temsirolimus	Torisel
Sirolimus	Rapamune
Radium 223	Xofigo
Raloxifene	Evista

According to the American Association of Oral and Maxillofacial Surgeons position statement,[214] patients may be considered to have medication-related osteonecrosis of the jaw (MRONJ) if all of the following

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characteristics are present:

1. Current or previous treatment with antiresorptive or antiangiogenic agents
2. Exposed bone or bone that can be probed through an intraoral or extraoral fistula in the maxillofacial region that has persisted for more than 8 weeks
3. No history of radiation therapy to the jaws or obvious metastatic disease to the jaws

Antiresorptive medications.

The exact mechanisms leading to MRONJ are unknown and the pathogenesis is likely multifactorial. Osteoclasts are involved in normal bone healing by signaling osteoblasts and influencing angiogenesis. As a result, any medication that impacts osteoclast number or function may influence bone resorption, new bone formation, and the local vasculature. Most cases arise following dentoalveolar surgery, suggesting an inflammatory or infectious etiology could be a contributor to the initiation of MRONJ.[260,271] Periodontal disease and periapical inflammation from a necrotic dental pulp are recognized risk factors for MRONJ, but the risk of developing osteonecrosis in patients exposed to antiresorptive medications following root canal treatment is unknown.[80,214,260,271] Other risk factors for the development of MRONJ include IV bisphosphonate use in cancer patients, corticosteroids, chemotherapy, diabetes, alcohol consumption, poor oral hygiene, and bisphosphonate use for more than 3 years. Smoking and advanced age may increase risk, but the current literature is not definitive.[145,261] In spite of these recognized risk factors, there is no way to reliably predict the development of MRONJ in any individual patient. The use of serum C-telopeptide cross-link of type 1 collagen (sCTX) has been advocated as a biomarker of bone metabolism and a predictor for the development of MRONJ.[163] However, several variables impact sCTX levels, and a systematic review concluded that current evidence does not support the use of sCTX levels as a predictor for the development of MRONJ.[70]

Bisphosphonates suppress osteoclast activity by increasing apoptosis. These medications have an extremely long half-life and exert an effect for

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many years or decades.[110] MRONJ risk is much higher for cancer patients taking IV doses of bisphosphonates compared to either IV or oral doses for the treatment of osteoporosis. However, the true incidence of MRONJ is not known, and patients with a history of antiresorptive treatment should be made aware of the risks and benefits prior to any treatment linked to the development of jaw necrosis.[9]

Denosumab (Prolia, Xgeva) is a human monoclonal antibody that confers an antiresorptive effect by preventing the development and maturation of osteoclasts. This medication is used both for the treatment of osteoporosis and skeletal-related events due to malignancy. Osteoclasts are derived from the monocyte/macrophage lineage and have a short lifespan of approximately 2 weeks. As a result, osteoclasts need to be continually replenished to keep cell counts constant. Blocking the maturation of these rapidly differentiating cells has a swift and significant impact on the number of functional osteoclasts. Unlike bisphosphonates, denosumab is not deposited in bone and the half-life is less than 1 month, resulting in complete clearance of the drug within 4 to 5 months. In spite of these differences, the risk for the development of MRONJ is very similar for cancer patients taking either denosumab or an IV bisphosphonate.[84,111,241]

Antiangiogenesis medications.

Osteoclasts govern the local vasculature during normal bone healing, and the administration of antiresorptive medications may influence the development of MRONJ by exerting an indirect effect on osteoclast-mediated angiogenesis. However, in some cancer patients, a more direct and profound impact on angiogenesis is desired in an attempt to prevent neovascularization in the tumor or metastatic spread. Sunitinib (Sutent) and sorafenib (Nexavar) are multiple tyrosine kinase inhibitors that block vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), resulting in decreased angiogenesis, tumor growth, and cancer progression. Bevacizumab (Avastin) is a monoclonal antibody that also targets and inhibits VEGF to limit tumor growth and metastasis. Unlike other initiators of MRONJ, in which the pathogenesis is likely multifactorial, the role of antiangiogenic drugs in the development of osteonecrosis is more intuitive. VEGF and PDGF are involved in normal bone remodeling and wound repair, and the

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inhibition of their activity leads to a disruption of the normal blood supply, especially in patients undergoing cancer chemotherapy with a compromised immune or healing state. There are multiple case reports linking antiangiogenesis medications to the development of MRONJ.[74,85,115,136,180]

The estimated risk of MRONJ in cancer patients receiving IV bisphosphonates is reported to be 1%, and as stated previously, this is comparable to cancer patients exposed to the RANK-L inhibitor denosumab. The risk for patients receiving VEGF inhibitors is much lower and is estimated to be 0.2% with bevacizumab.[214] However, patients that are treated with combined antiresorptive and antiangiogenic therapies are reported to develop MRONJ at a significantly higher rate, demonstrating the importance of a thorough medical history that includes both current and past medications.[26,43,100]

Treatment options for MRONJ are limited, therefore prevention is key and all patients should have a complete dental screening prior to initiating antiresorptive or antiangiogenesis treatment. For patients undergoing cancer treatment, oral surgery procedures should be completed while allowing several weeks for healing prior to starting medications with the potential to initiate MRONJ. If a cancer patient has already been exposed to antiresorptive or antiangiogenesis medications, it is best to minimize trauma to the bone, and root canal treatment would be preferred over extraction.[110] If oral surgery is necessary in these patients, the use of a drug holiday has been suggested, but there is little evidence in the literature to support this concept.

Radiographic findings.

Most areas involved with MRONJ are painful due to the presence of necrotic and exposed bone, but this is not universal. The mandible is affected more commonly than the maxilla and both jaws can be involved simultaneously.[217] Panoramic radiographs may show increased radiodensity of the crestal bone in both jaws, and CBCT is better able to demonstrate the sclerotic changes and cortical irregularities commonly associated with MRONJ.[102,256] Prior to necrosis, there is increased radiopacity due to the lack of bone resorption. After the osteonecrosis has been initiated, there will most likely be an irregular, “moth eaten”, and ill-defined radiolucent area with or without sequestra.[256] This radiographic appearance can be very similar to that of

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chronic osteomyelitis (Fig. 3.28). Depending on the location and extent of the necrosis, the radiographic appearance could also resemble periodontal disease or mimic a lesion of endodontic origin. Additionally, with a "moth-eaten" and destructive radiographic presentation, malignancy needs to be ruled out.[89]

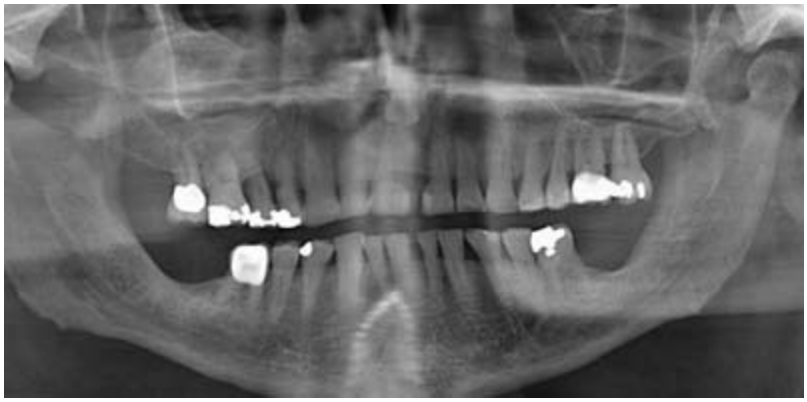

FIG. 3.28 Medication-related osteonecrosis of the jaw. A cropped panoramic radiograph shows a sclerotic rim of bone over the mesial buccal root of tooth #3 and the root apex of tooth #4. There is a well-defined radiolucent area over the midroot of tooth #4 suggesting localized periodontal disease. Purulent discharge and exposed bone were present from teeth #2 to #5.

Panoramic radiograph shows slightly tilted teeth with inverted U-shaped radiopaque region and porous tissue on either side below the teeth. Few teeth show radiolucent root tips.

Source: (Courtesy Dr. Christel Haberland.)

Non-Hodgkin lymphoma

Malignant lymphomas are a heterogeneous group of diseases characterized by a proliferation of lymphoid cells and their precursors. Lymphomas can be divided into two categories: Hodgkin and non-Hodgkin. Hodgkin lymphomas are far less frequent than non-Hodgkin lymphomas and rarely involve extranodal sites. Oral involvement of Hodgkin lymphomas is exceedingly rare and would not mimic lesions of endodontic origin.[267]

Non-Hodgkin lymphomas represent a group of malignancies that most often arise in the lymph nodes and grow as a solid mass, but 40% can arise in

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an extranodal site outside the lymphoid system.[72,186] In the maxillofacial region, non-Hodgkin lymphomas can result from the spread of disease into the area or they can arise de novo in soft tissue or bone.[186] These lymphomas can originate from either B lymphocytes or T lymphocytes. The incidence is increasing and approximately 70,000 cases are diagnosed each year in the United States with B lymphocytes being the most common cell of origin.[72,134] Patients with immunologic abnormalities have an increased risk for the development of non-Hodgkin lymphoma, and several different viruses have been implicated in the pathogenesis.[157,165]

Non-Hodgkin lymphomas are diagnosed over a wide age range and are more common in adults, but younger individuals can also be affected.[134] The early stage of the disease is characterized by a nontender, slow growing mass that is most likely associated with a group of lymph nodes. This emphasizes the importance of a complete and thorough examination of the maxillofacial region prior to any dental treatment. With time, the number of lesions increases and there may be growth outside of the lymph nodes into surrounding tissues.

Chemotherapy directed against the cell of origin is a common treatment strategy and the prognosis is influenced by the extent of the disease at the time of diagnosis.[165] High-grade lymphomas are treated with chemotherapy and radiation if localized, but radiation will not be used if the disease has spread. The treatment for low-grade lymphomas in older individuals is more debatable. Many of these patients may have no treatment initially, as these lymphomas tend to recur following treatment, and survival of 8 to 10 years without treatment is not unexpected. However, 40% of low-grade non-Hodgkin lymphomas transform to high-grade disease requiring aggressive treatment, and the overall cure rate is low.[179]

Radiographic features.

The radiographic and clinical presentation is variable, depending on the aggressiveness of the tumor at the time of diagnosis. Dull, vague pain that mimics a toothache is present in some cases, while others may have significant pain, swelling, or paresthesia. The maxilla is more commonly involved, but mandibular involvement should not be considered rare.[134] Early lesions may be radiographically subtle and difficult to identify with

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panoramic imaging.[117] These lesions can resemble a periapical cyst or periapical granuloma and may demonstrate a loss of lamina dura and widening of the PDL space (Fig. 3.29).[61,82,131,138,170,194,236,220] CT and MRI will more clearly reveal bone marrow involvement, often with less cortical destruction than would be expected given the aggressive nature of the neoplastic process. These findings are not specific for non-Hodgkin lymphoma, and other inflammatory and neoplastic diseases need to be considered in the differential diagnosis.[117,164] As the disease progresses, an ill-defined and destructive radiolucency more consistent with a malignant process is likely to emerge. If the lymphoma remains undiagnosed, it will continue to expand and ultimately perforate the cortical plate.

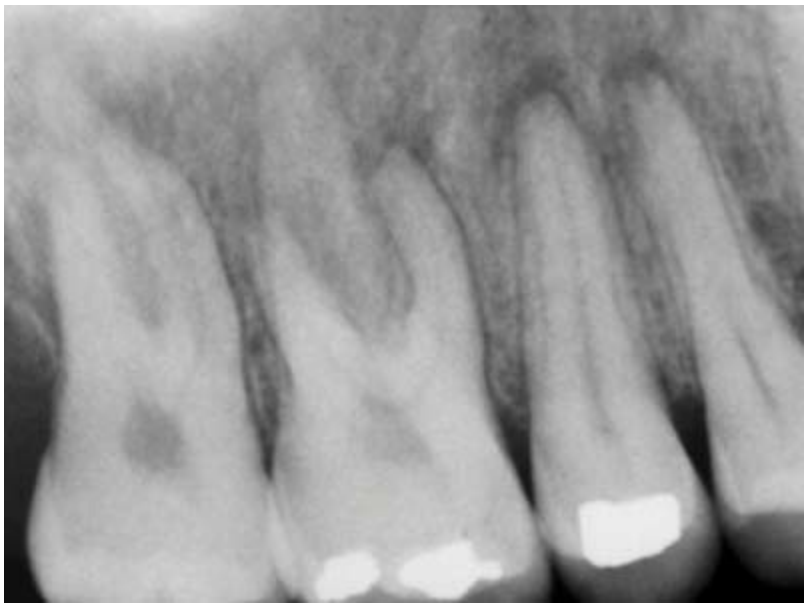

FIG. 3.29 A 29-year-old female with a history of numbness and swelling. These teeth responded normally to pulp testing, but there was a firm and tender buccal swelling from tooth #2 to #6. A diffuse radiolucency was present in the area of the buccal swelling with well-defined radiolucencies at the root apices. Teeth #3 and #4 had limited restorative histories and #2 and #5 were unrestored. Given this clinical presentation a noninflammatory etiology was suspected and biopsy revealed a lymphoma.

Radiograph shows four teeth. The first tooth has radiolucency in the pulp. The second tooth has radiopaque patches at the crown and thin tissue at the root apex. The third tooth has a rectangular radiopaque region at the crown with radiolucency at the root tips of both third and fourth teeth.

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Source: (Courtesy Dr. Michael Ribera.)

Metastatic disease

Metastatic deposits of a primary carcinoma can spread via the bloodstream into the jaws, but this is a rare occurrence.[133] Bones that are more commonly involved include the vertebrae, ribs, pelvis, and skull. Although any malignancy has the potential for metastasis, the most common carcinomas to spread to bone are breast, lung, thyroid, kidney, and prostate. Metastatic disease can be asymptomatic or produce pain, swelling, mobility, and paresthesia. The pain level and presentation can mimic pulpal origin.[63,114] In some cases, metastatic disease can precede the diagnosis of the primary tumor, requiring an extensive workup to determine the location of the primary malignancy.[63,113]

Metastatic disease is more likely to occur in older individuals, corresponding to the increased incidence of primary malignancies in adults. However, metastatic disease has been reported over a wide age range. By definition, a metastatic deposit means an advanced stage of disease progression, and treatment is usually palliative. Bisphosphonates have been used to reduce pain levels and limit the risks of a pathologic fracture, but the overall survival rates are poor.

Radiographic features.

Radiographically, metastatic deposits present most commonly as ill-defined radiolucencies in the mandibular molar region that can be destructive leading to pathologic fracture (Fig. 3.30).[63,207] However, metastatic disease can be very subtle in presentation with only a slight widening of the PDL space or, in some instances, the bone may be radiographically normal.[179] It is possible for breast and prostate metastasis to stimulate an osteoblastic reaction locally, resulting in a radiopaque appearance.[96] Depending on location and growth pattern, metastatic deposits can mimic periodontal disease or an endodontic infection radiographically.[59,87,135,225,255]

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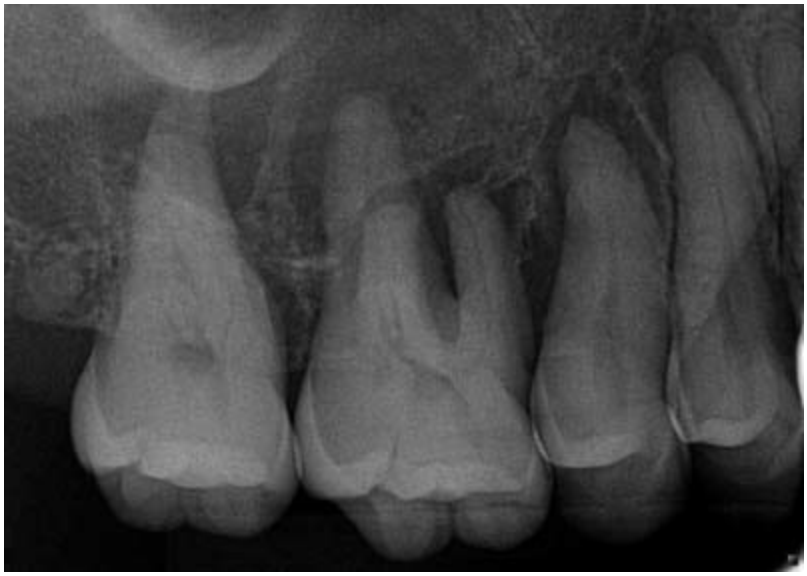

FIG. 3.30 A periapical image demonstrating multiple areas of radiolucency in a 42-year-old female with a chief complaint of achiness and loose teeth in the maxillary right quadrant. These teeth responded to both cold and electric pulp testing. A CBCT revealed an irregular and ill-defined loss of density that perforated the buccal and palatal cortical plates and the maxillary sinus. There was resorption of several root ends. The overall presentation suggested malignancy, and biopsy revealed metastatic breast cancer.

Radiograph shows three teeth with thin tissue in the root and next to the root. The central tooth shows filled root canals. One of the root canals appears abnormal extending into the skin. The third tooth shows radiolucent root canal.

Source: (Courtesy Dr. Scott Doyle.)

Multiple myeloma

Multiple myeloma is an uncommon malignancy of plasma cells that commonly presents with simultaneous involvement in many bones. Multiple myeloma makes up only 1% of all malignancies but is the second most common hematologic malignancy, trailing only lymphoma in incidence.[204] The diagnosis is made using electrophoresis of serum and concentrated urine to demonstrate the presence of M-protein, which is the overproduction of one abnormal immunoglobulin by the malignant plasma cells.[29,203,240]

Multiple myeloma has a mean age at diagnosis of 66 to 70 years and is rarely diagnosed before the age of 40. Most reports suggest there is a slight

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male predilection, but the incidence for blacks is two times greater than for those of European ancestry.[147] Bone pain is the most common symptom, and this may be associated with fatigue if there is underlying anemia.[71,83] Fever and infection can be present due to neutropenia. A nodular tongue is found in nearly 15% of patients because of amyloid deposition from the abnormal aggregation of M-protein. Additionally, the excessive circulating light chain component of the M-protein can lead to renal failure resulting in proteinuria.[29]

Several chemotherapy regimens have been used historically, but autologous stem cell transplantation has become the first-line treatment for patients deemed healthy enough to tolerate the procedure.[29,204] Radiation for isolated bone lesions is considered palliative, and bisphosphonates are utilized to reduce the likelihood for fracture and pain.[29] Newer chemotherapeutic regimens have resulted in better survival rates, but multiple myeloma is difficult to cure, and treatment often seeks to control disease and prolong survival. Thalidomide, lenalidomide, and bortezomib used in combination with dexamethasone has resulted in improved survival.[144] The prognosis is less favorable for older patients with disseminated disease, and the overall 5-year survival approximates 50% in most reports with a median survival of 6 to 7 years.[204] Medical consultation with the patient's oncologist would be recommended prior to any dental care.[240]

Radiographic features.

Radiographically, there are typically multiple radiolucent areas, some with well-defined, punched-out borders and others that present with more irregular and diffuse margins (Fig. 3.31).[71,149] The presence of multiple lesions suggests a noninflammatory etiology, but a solitary lesion on a single periapical radiograph could mimic a periapical cyst or periapical granuloma. As a result, a panoramic radiograph is recommended if multiple myeloma is suspected.[30] Root resorption is not common but has been reported.[257]

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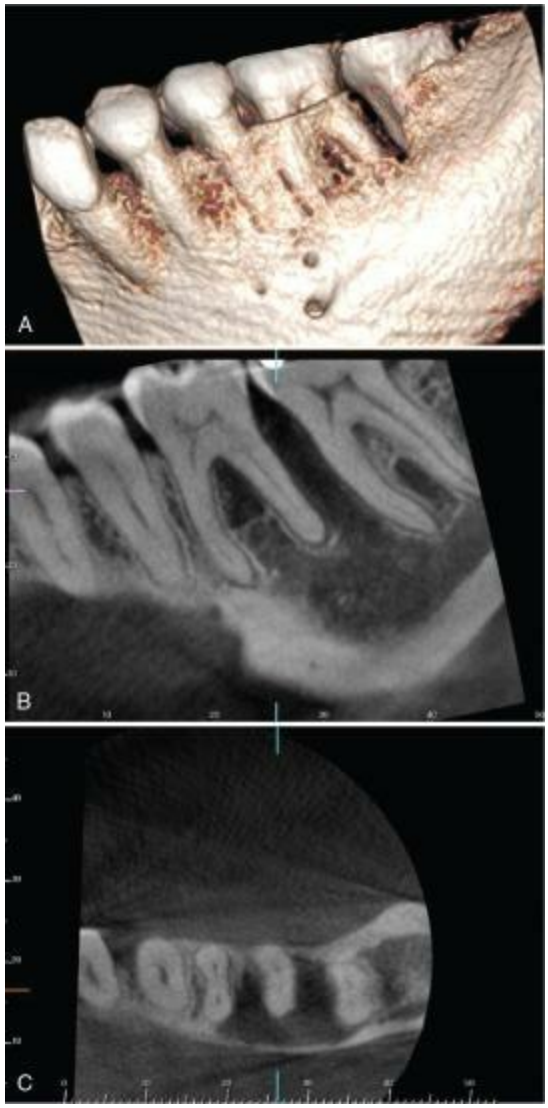

FIG. 3.31 **A,** Volumetric rendering reveals multiple osseous defects throughout the mandible consistent with multiple myeloma. **B,** Sagittal CBCT image showing a well-defined and destructive low-density lesion. **C,** Axial CBCT image exhibiting destruction of the trabecular bone and marked thinning of both buccal and lingual cortical plates.

A) 3D image shows lower jaw with perforated tissue depicted as hollow space in-between the teeth below the gum line.

B) CBCT image shows teeth with radiolucency at the center of root, in-between teeth, and below the root.

C) CBCT image shows teeth arranged horizontally with extremely thin tissues around teeth on the left.

Source: (Courtesy Dr. Marcel Noujeim.)

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Well-defined unilocular periapical radiolucencies

As stated previously, a unilocular, well-defined apical radiolucency most likely represents a periapical cyst or periapical granuloma if the dental history and clinical testing are consistent with an inflammatory etiology. However, many pathologies, with a wide range of clinical significance, can exhibit the identical radiographic presentation as periapical cysts and periapical granulomas, and the dental literature is replete with case reports of noninflammatory pathologies misdiagnosed as periapical inflammatory disease. The astute clinician needs to consider all possibilities when forming a differential diagnosis.[235]

Periapical cysts and periapical granulomas

Pulp necrosis occurs due to the presence of bacteria within the root canal system.[123] Periapical granulomas represent an accumulation of inflamed granulation tissue as a result of a necrotic pulp. Bacteria and byproducts of bacterial metabolism trigger an immune response, most often at the root apex, but this reaction can occur anywhere along the root surface if the pulp space communicates with the PDL via accessory anatomy or a fracture. The affected tooth should be nonresponsive to pulp testing, unless partial necrosis of a multirooted tooth results in variable levels of responsiveness and a more difficult diagnosis.

The exact mechanism of cyst formation is unknown and numerous theories have been proposed.[175,247,251,253] If epithelium near the root apex of a necrotic tooth is stimulated by an inflammatory response, a periapical cyst can result.[152] There is likely a combination of interactions between the resident tissues in the area, including epithelium, bone, extracellular matrix, and the inflammatory cascade.[24] As with periapical granulomas, the affected tooth does not respond to pulp testing and the diagnosis can be more confusing if partial necrosis is present in a multirooted tooth.

Historically, lesions that were larger than 1.5 cm and well demarcated were considered cysts, and it was thought surgery or extraction would be necessary for complete healing.[88,176] However, this concept has evolved and it is generally accepted that there is no way to differentiate periapical cysts from periapical granulomas using conventional imaging (Fig. 3.32), irrespective of the size or cortication of the radiolucency.[38,208] The ability to differentiate

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periapical cysts and periapical granulomas utilizing CBCT has also been the focus of numerous studies with inconsistent findings. Guo and colleagues suggested that CBCT is moderately accurate at differentiating periapical cysts from granulomas, while Simon et al. reported CBCT is an accurate differentiator, but found agreement between the pathologist and radiologist in only 76% of cases.[101,232] Other studies concluded that biopsy remains the gold standard to differentiate periapical cysts from periapical granulomas (Fig. 3.33).[35,212]

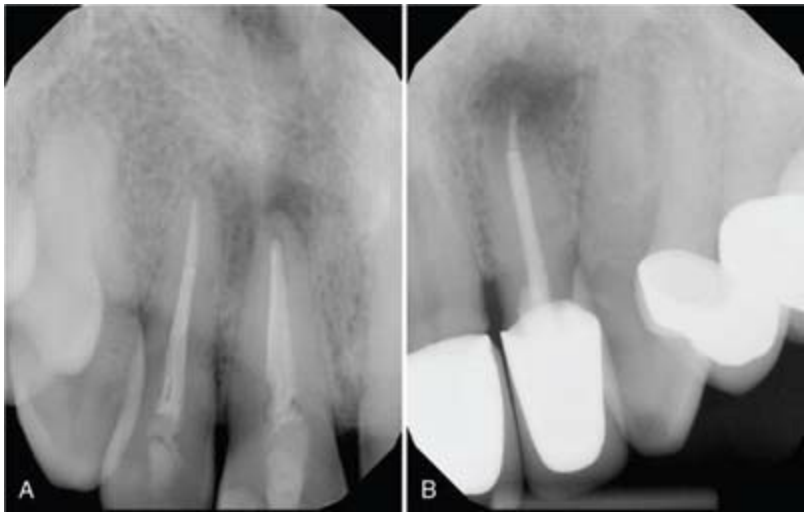

FIG. 3.32 **A,** Well-defined radiolucency at the root apex of tooth #8. The previous root canal filling is short of the apex and exhibits numerous voids. A tissue sample obtained during apical surgery revealed a periapical granuloma. **B,** Well-defined radiolucency at the root apex of tooth #10. Apical surgery was completed, and the biopsy confirmed a periapical cyst. There is no way to differentiate periapical cysts from periapical granulomas using conventional imaging.

A) Radiograph shows left tooth with filled root canal and thin tissue at the root apex.

B) Radiograph shows 10th tooth with extended root canal and nearly circular patch of thin tissue at the root apex.

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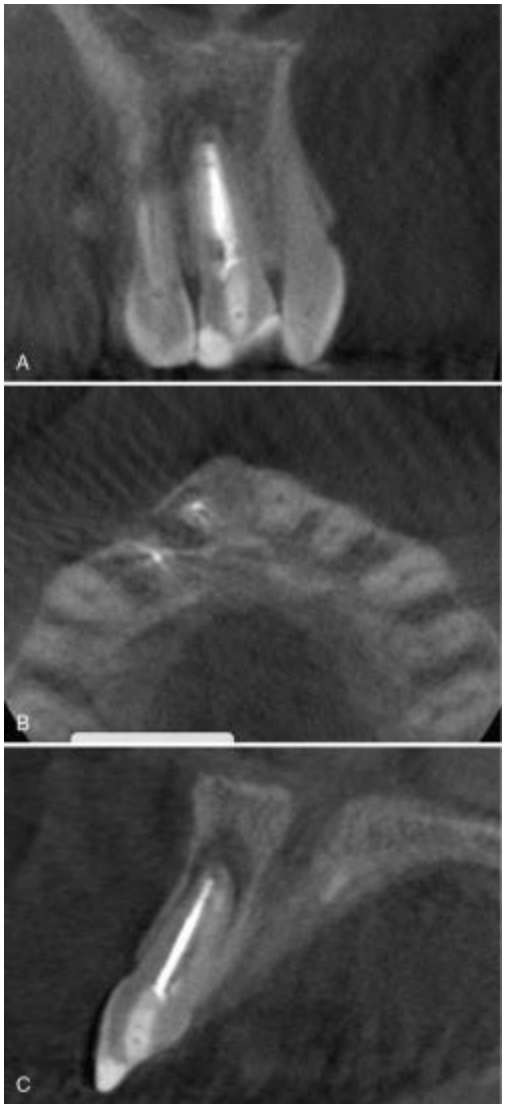

FIG. 3.33 CBCT images of the periapical granuloma from Fig. 3.32, *A*. It is not possible to differentiate periapical cysts from periapical granulomas using CBCT, and biopsy remains the best method to definitively make this distinction. **A,** Sagittal CBCT image of periapical granuloma. **B,** Axial CBCT image of periapical granuloma. **C,** Coronal CBCT image of periapical granuloma.

Three CBCT images, A through C, show sagittal, axial, and coronal sections of poorly anchored tooth due to lesions at the root tip in the periapical region.

Multislice-CT has been reported to have a high degree of accuracy distinguishing periapical cysts from periapical granulomas, but this technology requires significantly more radiation exposure and the quality of the image is not as accurate as CBCT.[45,258] Both ultrasound and MRI may be able to differentiate periapical cysts from periapical granulomas, but clinical

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usage has been limited and these devices are not readily available in the typical dental setting. However, early reports are encouraging and more research is needed to support the use of these technologies.[4,60,153,161]

Because there is no way of knowing whether a radiolucency represents a periapical cyst or a periapical granuloma with imaging alone, it is impossible to know the true incidence of these inflammatory lesions and to subsequently determine if there is a difference in their healing potential following endodontic treatment. Apical surgery or extraction is needed to confirm the diagnosis, and this may lead to a sampling bias of cases that have not healed following routine endodontic treatment. Additionally, in spite of histopathology being considered the gold standard at differentiating periapical cysts from periapical granulomas, this is not without controversy. Some pathologists will make the diagnosis of periapical cyst if an epithelial lining is seen in a histologic section in association with granulation tissue, while other pathologists require visualization of the cyst lining on a basement membrane and an intact luminal surface.

During apical surgery, tissue is curetted from the apex and the sample is typically not removed in a single piece. This may result in a breakdown of the cyst lumen and a decreased likelihood for the diagnosis of a cyst. Moreover, for larger lesions, the entire tissue sample may not be evaluated histologically and slides of representative samples will be viewed by the pathologist. This increases the potential to overlook a small cyst contained in the unviewed tissue. Studies that have utilized intact resected specimens and serial sections to evaluate the entire lesion have reported a lower incidence of cysts compared to many other historical studies.[173,231]

The reported incidence of periapical cysts and periapical granulomas is highly variable in the literature, ranging from 6% to 55%.[17,146,156,174,237] True cysts are completely lined by epithelium and lack any communication with the root canal space. As a result, the healing potential for true cysts may be limited following conventional root canal treatment. In contrast, pocket cysts are in direct contact with the root canal system and may have a greater likelihood for healing following endodontic treatment.[173,231]

Treatment of periapical cysts and periapical granulomas is identical. Both require identification of the offending tooth, allowing removal of the inflammatory stimulus with root canal treatment or extraction. Treatment

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choices will depend on the patient's treatment plan, periodontal status, restorability of the tooth, and patient desires. The diagnosis of periapical inflammatory disease becomes more complicated if the radiolucency is associated with a previously root canal–treated tooth because pulp testing is no longer relevant.

Radiographic features.

Periapical granulomas and periapical cysts present with variable levels of discomfort, from mild awareness to debilitating pain, and the symptoms often correspond to the pain levels identified with percussion and palpation during clinical examination. Many periapical granulomas and periapical cysts are asymptomatic and are discovered on routine radiographs.

In the earliest stages of infection, neutrophils predominate and there can be very little or no bone involvement, consistent with acute dental pain, but no evidence of abnormality on radiographic evaluation. As the inflammatory cascade persists, there is the potential for more bone destruction and a radiolucent area can develop, ranging from a subtle widening of the PDL space to extensive bone loss of more than several centimeters (Fig. 3.34). The radiolucent area can exhibit a well-defined and corticated margin or the inflammatory process can stay within the medullary spaces and lack definition radiographically (Fig. 3.35). Root resorption is not uncommon and the outline and definition of the radiolucency can be altered depending on the angle used to obtain a two-dimensional periapical radiograph. As a result, it is often necessary to obtain multiple radiographs to identify a subtle periapical lesion, but this may not improve diagnostic accuracy in all cases.[36] A CBCT may be a better alternative if there is doubt about the presence of periapical pathology.[142]

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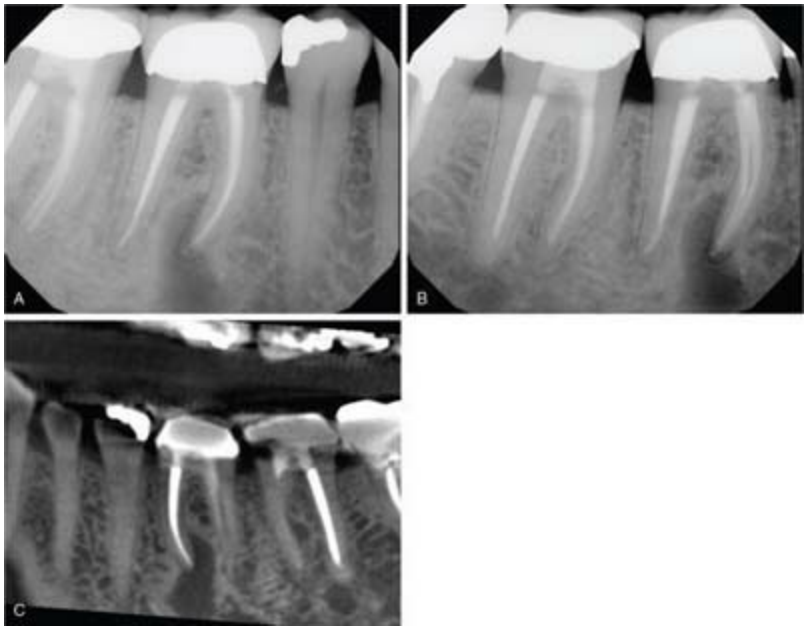

FIG. 3.34 **A,** Periapical image of tooth #30 with a previous root canal and a well-defined radiolucency at the mesial root apex. **B,** Angled periapical image of the same tooth, further demonstrating the extent of the lesion. **C,** Sagittal CBCT demonstrating a loss of density at the mesial root apex of tooth #30. This well-defined lesion was sampled during an apical surgery, and the biopsy showed a periapical cyst.

A) CBCT image shows filled root canals and a large dark parch at the root tip of molar.

B) CBCT image is similar as that of A, except that the dark pitch is thinner.

C) CBCT image shows molar with radiopaque edges of crown and radiolucency in-between. The molar has one radiopaque root canal with a large patch and one radiolucent root canal.

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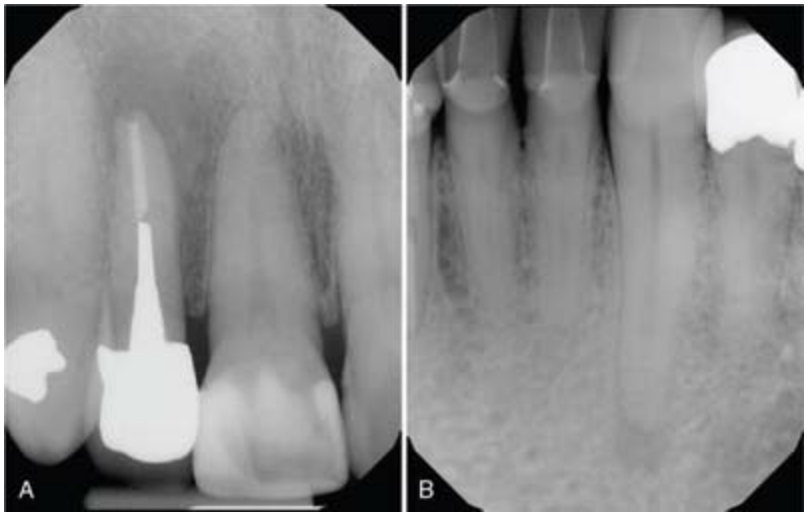

FIG. 3.35 **A,** Biopsy revealed a periapical cyst at the apex of tooth #7. There is a loss of trabeculation at the apex blending into normal bone resulting in an ill-defined radiolucency. **B,** Well-defined periapical granuloma at the root apex of tooth #27 was confirmed with a biopsy when bone loss and symptoms persisted following nonsurgical root canal treatment. Size and radiographic definition of periapical cysts and periapical granulomas are highly variable.

A) Radiograph shows radiopaque crown and broken root canal with a cystic lesion (low-density tissue) at the root apex.

B) Radiograph shows 27th tooth with radiolucent root canal and a square-shaped patch at the root apex.

Lateral radicular cyst

The bacterial etiology of a lateral radicular cyst is identical to that of a periapical cyst or periapical granuloma; the only difference is in the location affected. As a result, pulp testing is critical to determine the causative tooth because several other pathologies can look radiographically identical in this area. Endodontic treatment or extraction is indicated depending on the patient's treatment plan, periodontal status, restorability of the tooth, and patient preference.

Radiographic features.

Lateral radicular cysts are most commonly well-defined radiolucencies, and some may have a corticated border (Fig. 3.36). However, these lesions will exhibit the same growth patterns as periapical cysts and periapical

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granulomas resulting in an ill-defined radiolucency when the inflammatory cascade is confined to the medullary spaces. A CBCT will better demonstrate the radiographic changes when the lesion is small or lacks definition. The differential diagnosis for radiolucencies in the lateral location includes, but is not limited to, the following: OKC, ameloblastoma, and lateral periodontal cyst (LPC).

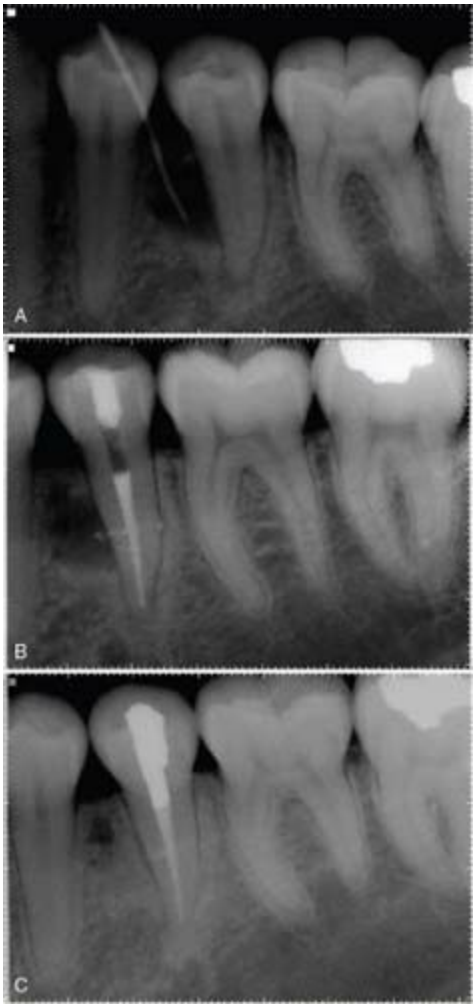

FIG. 3.36 Lateral radicular cyst. **A,** Preoperative image with gutta-percha tracing a draining sinus tract. Patient has a history of biopsy in the area that was diagnosed as a dentigerous cyst. Tooth #20 responds to both cold and electric pulp testing. Options included root canal treatment, extraction, or a second biopsy. The sinus tract suggests a necrotic pulp and the potential for a false-negative pulp test. Also, the diagnosis of dentigerous cyst is not accurate because the tooth has erupted. Nonsurgical root canal treatment was completed. **B,** Several lateral canals can be seen opposite the radiolucent areas and

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the sinus tract resolved within 1 week. **C,** Healing of bone is evident at 18-month recall.

A) Radiograph shows extremely less-density tissue at gum line between teeth. A wedge-shaped structure lies next to the 20th tooth.

B) Radiograph shows 20th tooth with filled root canal and radiolucent region in-between. The gum line on one side of the tooth appears dense.

C) Radiograph shows 20th tooth with filled root canal and normal tissue with small dark patch at one side of the tooth.

Lateral periodontal cyst

The LPC is an uncommon developmental cyst of odontogenic origin thought to arise from rests of dental lamina. Although the LPC is developmental and occurs over a wide age range, diagnosis is uncommon before 30 years of age. The mandibular canine and premolar areas are the predominant sites of involvement, but reports indicate that it can occur in either arch.[40,79,234] LPCs are often discovered as an asymptomatic finding on routine radiographs, but expansion, tenderness to palpation, and pain are possible. Most present as a single lesion, but LPCs can have a multifocal presentation, and a multicystic variant known as a botryoid LPC has also been described.[127,263] Surgical removal is the treatment of choice and recurrence is not likely. However, due to the multicystic nature of the botryoid subtype, a higher recurrence rate is possible.[103,199]

Radiographic features.

Radiographically, the LPC is characterized by a unilocular, well-defined, oval-shaped radiolucency that is indistinguishable from lateral radicular cysts in many cases (Fig. 3.37). However, the adjacent teeth should respond to pulp testing, unless there is pulpal pathology for unrelated restorative reasons. This radiographic presentation is also very similar to early unilocular OKCs or ameloblastoma (Fig. 3.38). Because they are slow growing, most are less than 1 cm at the time of diagnosis. Less commonly, an LPC can be located near a root apex, suggesting a lesion of endodontic origin.[181] The botryoid subtype can present as a multilocular radiolucency, but it is most often unilocular in spite of its multicystic growth pattern.

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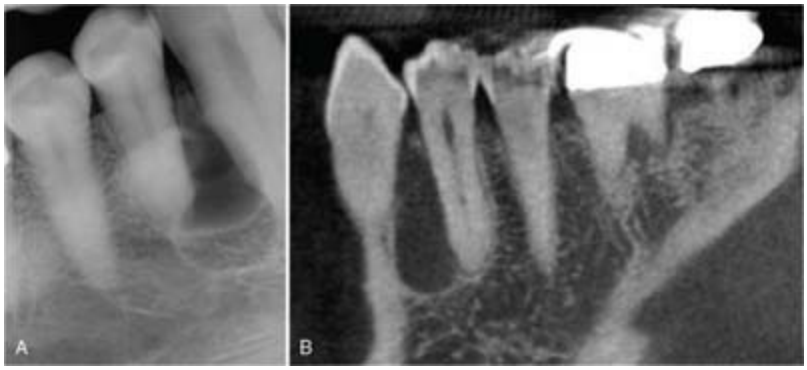

FIG. 3.37 Lateral periodontal cyst. **A,** Periapical image of a well-defined radiolucency near the root apex of mandibular premolar and canine. The periapical image suggests a multilocular presentation that would not be consistent with periapical inflammatory disease from a bacterial etiology. **B,** Sagittal CBCT image revealing the loss of density between the canine and premolar teeth with a normal PDL space at the apex of tooth #21.

A) Radiograph shows thin tissue below the gum line at the root apex between two teeth. All teeth have radiolucent root canal.

B) Radiograph shows teeth surrounded by less-density porous tissues.

Source: (Courtesy Dr. Martin Rogers.)

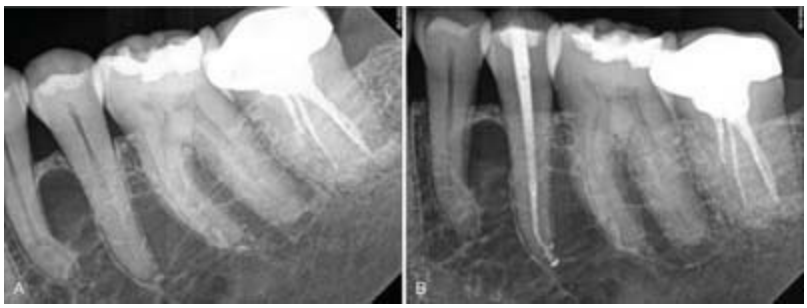

FIG. 3.38 **A,** Pulp testing indicated irreversible pulpitis symptoms from tooth #20 and a well-defined radiolucent area was identified between teeth #20 and #21. **B,** Nonsurgical root canal treatment was completed to address the thermal symptoms, and referral was made for a biopsy that identified an odontogenic keratocyst. Lateral radicular cysts, unicystic ameloblastomas, odontogenic keratocysts, and lateral periodontal cysts can exhibit identical radiographic appearances in the mandibular canine and premolar areas.

A) Radiograph shows lower jaw with thin dark patches between teeth. 20th tooth shows radiolucent root canal.

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B) Radiograph is similar to radiograph A, except that root canal is filled in 20th tooth.

Source: (Courtesy Dr. Karen Potter.)

Focal cemento-osseous dysplasia

Focal cemento-osseous dysplasia is another fibro-osseous lesion characterized by replacement of bone with fibrous and mineralized tissue. As the name implies, focal cemento-osseous dysplasia involves a single site, unlike PCOD, which is more commonly multifocal. Although the exact etiology is unknown, the proximity to root ends and a cementum-like component suggest the cells of origin reside in the PDL.[244] As with PCOD, the majority of cases affect females and are diagnosed after 30 years of age.[130,244] Although there is a predilection for the black population, it is not as strong as is seen with PCOD. The diagnosis could be based on radiographic findings and pulp testing, but other pathologies, with more significant growth potentials, can resemble FCOD, necessitating a biopsy for definitive diagnosis. After the diagnosis has been established, routine follow-up of the affected bone is indicated.

Radiographic features.

Focal cemento-osseous dysplasia usually involves the posterior mandible, is commonly asymptomatic, and is discovered on routine radiographic examination.[159,244] Most lesions are smaller than 1.5 cm and exhibit a variable and mixed radiolucent radiopaque appearance, but some may be completely radiolucent (Fig. 3.39). There is usually a well-defined border, but not always. They tend to occur near root ends and will mimic periapical inflammatory disease if no radiopacity is present within the lesion.[62]

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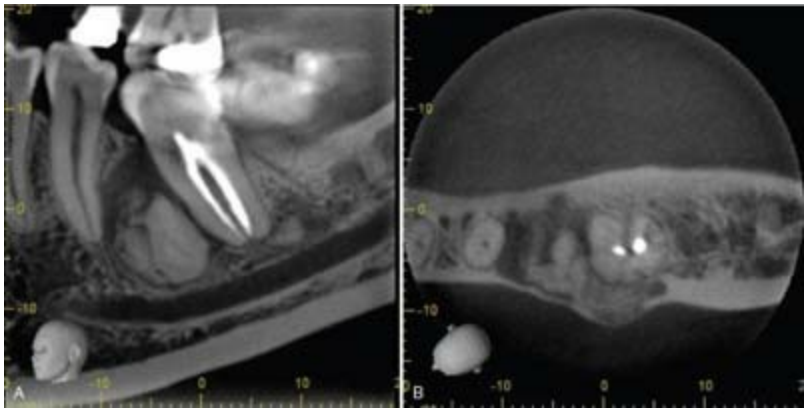

FIG. 3.39 Focal cemento-osseous dysplasia. **A,** Sagittal CBCT image demonstrating a mixed-density lesion with a low-density periphery between the mandibular second premolar and first molar. A second high-density lesion is present near the distal root apex of the first molar. **B,** Axial CBCT image exhibiting an expanded and thinned cortex from a mixed-density central globular mass with a hypodense periphery.

A) CBCT image shows side view of human face at the right corner. Tooth at the center is tilted toward the adjacent tooth that has radiolucent root canal. The gum shows fibrous tissue with a multilocular mass and radiolucent boundary below the root tips.

B) CBCT image shows top view of human skull at the right corner. Teeth arranged horizontal show ill-defined peripheries with fibrous tissues inside.

Source: (Courtesy Dr. Bruno Azevedo.)

Nasopalatine duct cyst

Nasopalatine duct cysts, also referred to as incisive canal cysts, are the most common nonodontogenic cyst in the jaws with a prevalence of approximately 1%.[177,179] They are likely developmental but are most often diagnosed after the age of 30, suggesting a possible traumatic or bacterial etiology. The diagnosis is typically based on radiographic findings, and nasopalatine duct cysts have an excellent prognosis following surgical removal.

Radiographic features.

Nasopalatine duct cysts can occur at any age, but they are more likely found in adults and are rare before 10 years of age.[107,223] Swelling, drainage, and pain are the most common symptoms associated with these cysts. Many are asymptomatic and discovered on routine radiographs. They present as a well-

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defined, round or heart-shaped radiolucency in the maxillary midline or just lateral to the midline (Fig. 3.40). Most are between 1 cm and 2 cm, but they will uncommonly exceed 5 cm and exhibit a more destructive nature.[195,215] Due to superimposition over the root ends of central incisors, pulp testing and clinical correlation are necessary to avoid mistaking nasopalatine duct cysts for lesions of pulpal origin.[78]

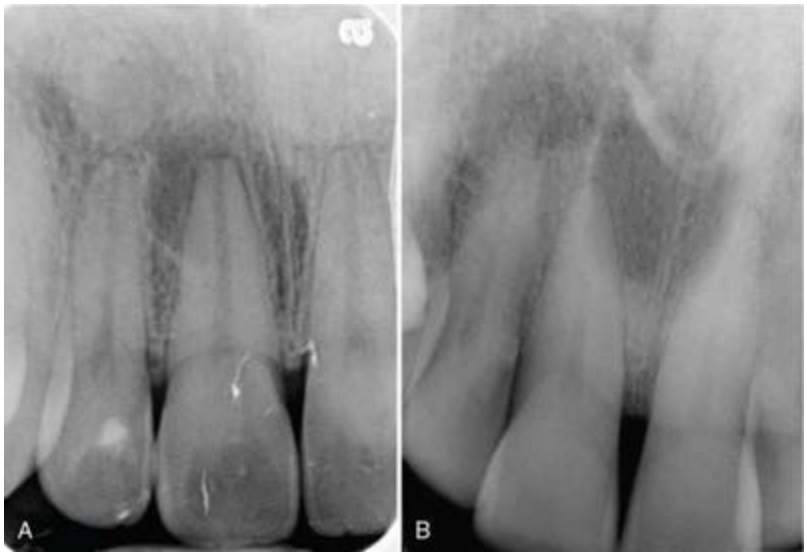

FIG. 3.40 **A,** Periapical image with a well-defined unilocular radiolucency at the root apex of tooth #8. Asymptomatic and no history of trauma. Tooth #8 responded to both cold and electric pulp testing. A biopsy revealed a nasopalatine duct cyst. **B,** The more classic presentation of nasopalatine duct cyst with a rounded radiolucency in the midline.

A) Radiograph shows the central tooth that has a radiolucent circular patch with a vertical radiopaque line inside in the crown. Radiolucent tissue extends from the gum line toward the root apex.

B) Radiograph shows dark patch of abscess between teeth near the root tips.

Source: (Courtesy A, Dr. Christel Haberland; B, Dr. Martin Rogers.)

Stafne defect

Stafne defects are asymptomatic radiolucencies most commonly located near the angle of the mandible, but a less common anterior position has been reported.[34,52,126,239] The exact etiology is unknown and it is often considered

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to be developmental in nature. However, the radiolucent area is usually not diagnosed until middle age, leading some investigators to postulate pressure resorption as the cause. There is a strong male predilection and due to the classic radiographic presentation, the diagnosis of posterior defects is typically made based on radiographic findings alone. No treatment is indicated unless there is uncertainty about the diagnosis, and biopsy most often yields normal salivary gland tissue.

Radiographic features.

Because Stafne defects are asymptomatic they are most often discovered as an incidental finding on panoramic radiographs.[148] They present as radiolucencies on the lingual side of the mandible inferior to the mandibular canal with a well-defined and corticated border (Fig. 3.41).[262] Although the size is variable, most Stafne defects are between 1 cm and 2 cm and, in some instances, the radiolucent area will approximate the root ends of the mandibular teeth, suggesting a periapical cyst or periapical granuloma (Fig. 3.42). Anterior Stafne defects associated with the sublingual gland are less common. Superimposition over anterior root ends and the atypical position suggest periapical inflammatory disease or any other unilocular well-defined radiolucency. In these cases, it is important to correlate clinical and restorative histories with the radiographic findings to ensure appropriate treatment.[58] Additionally, both the axial and coronal planes of a CBCT will demonstrate the lingual concavity and lack of involvement at the root ends.

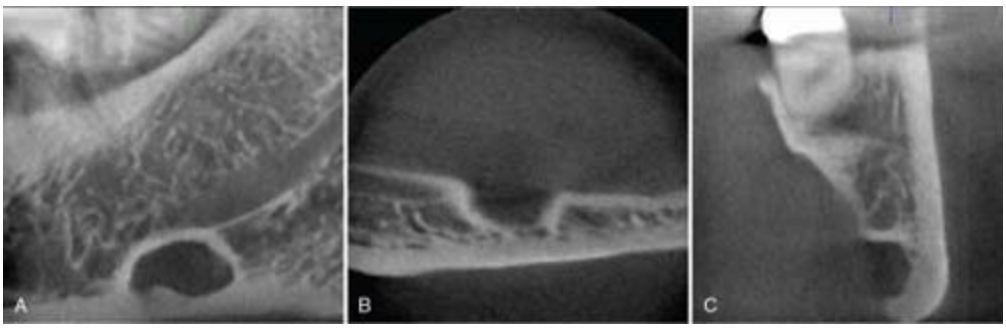

FIG. 3.41 Stafne defect. **A,** Sagittal CBCT image showing a well-defined loss of density inferior to the mandibular canal. **B,** Axial CBCT image demonstrating a lingual concavity. **C,** Coronal CBCT image also exhibiting a lingual concavity inferior to the mandibular canal with no involvement of the root ends.

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A) CBCT image shows a bone cavity in the lower jaw. Surrounding tissue appears fibrous.

B) CBCT image shows top view of teeth with U-shaped cavity at the upper side.

C) CBCT image shows the cavity at the lower end of the jaw.

Source: (Courtesy Dr. Bruno Azevedo.)

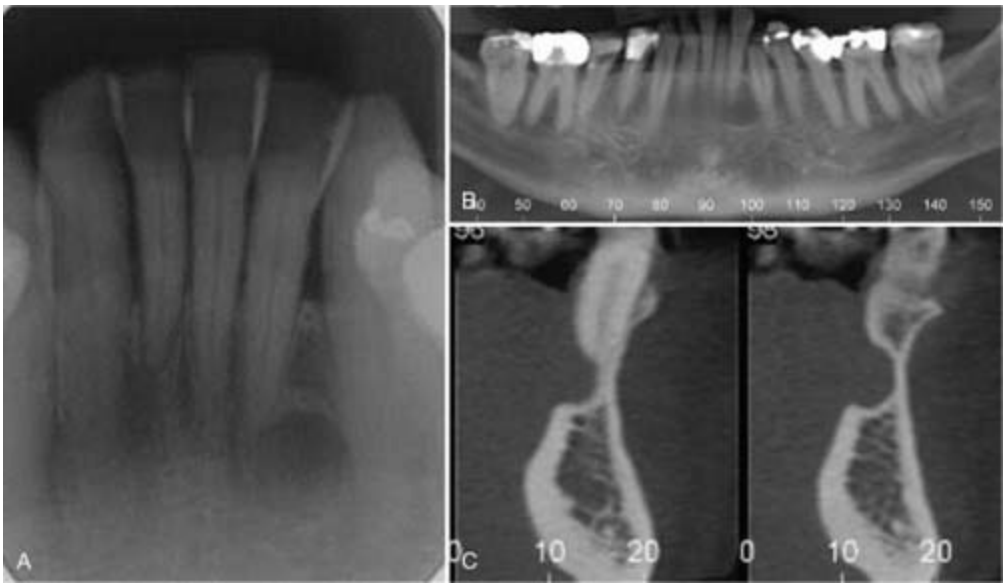

FIG. 3.42 Anterior Stafne defect. **A,** Periapical image exhibiting a well-defined radiolucency near, but not at, the root apex of tooth #23. The lack of restorative history and location of the lesion suggest a noninflammatory etiology and these teeth respond to pulp testing. **B,** Reformatted panoramic image showing a loss of density near the apex of tooth #23. **C,** Coronal CBCT images reveal a concavity in the lingual cortex of bone with no involvement of the root ends.

A) Radiograph shows a dark globular patch near the root tip of tooth. All teeth have radiolucent root canals.

B) Radiograph shows lower jaw with the dark patch almost near the center.

C) A series of two radiographs shows hollow cavities in the lingual cortex of 23rd tooth.

Source: (Courtesy Dr. Bruno Azevedo.)

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Summary

For practitioners, it is important to remember that the great majority of radiographic abnormalities encountered on a daily basis will be due to a bacterial etiology as the result of a necrotic pulp. However, it is imperative that clinicians also understand that developmental, metabolic, and neoplastic etiologies can very closely resemble an inflammatory cause.

A multilocular radiolucency should not be the result of a necrotic pulp, and further diagnostic workup is indicated when this type of change is identified. Most radiographic lesions of inflammatory origin are going to be radiolucent, and the presence of calcification suggests a higher likelihood for a nonpulpal etiology. Furthermore, a multifocal presentation should arouse suspicion about noninflammatory etiologies, especially if the dental history and pulp testing are not consistent with pulp necrosis in multiple teeth. Periapical inflammatory disease can be well defined and corticated radiographically or the lesion can have ill-defined margins when the radiographic change is confined to the medullary spaces. However, rapid cortical expansion or perforation of the cortical plate, extensive tooth mobility, irregular root resorption, and a “moth eaten” appearance suggest a more ominous differential diagnosis.

This chapter categorized lesions based on the most typical radiographic presentations encountered in clinical practice, but many of these pathologies defy classification into a single category and may exhibit a wide variety of radiographic appearances. This review also focused on the most common noninflammatory lesions that present in the periradicular regions, but it is in no way a comprehensive account of all potential pathologies affecting the region.

Radiographs are one piece of the diagnostic puzzle, and accurate interpretation of radiographic findings in conjunction with a thorough review of the medical history, dental history, pulp testing, and clinical examination will yield a differential diagnosis when a nonbacterial etiology is suspected. It is not necessary, nor is it possible, to make a definitive diagnosis of many radiographic abnormalities based exclusively on imaging. However, making the determination that a radiographic lesion is not pulpal in origin should be possible in most cases and further evaluation, in conjunction with referral to the appropriate medical or dental provider, can result in a timely and accurate

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diagnosis, significantly influencing disease progression and prognosis for many patients.

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References

1. AAE and AAOMR joint position statement. *Use of cone beam computed tomography in endodontics 2015 update*, J Endod 2015;41: 1393.
2. Abella F, Patel S, Duran-Sindreu F, et al: *Evaluating the periapical status of teeth with irreversible pulpitis by using conebeam computed tomography scanning and periapical radiographs*, J Endod 2012;38: 1588.
3. Aerden T, Grisar K, Nys M, et al: *Secondary hyperparathyroidism causing increased jaw bone density and mandibular pain: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol 2018;125: e37.
4. Aggarwal V, Logani A, Shah N. *The evaluation of computed tomography scans and ultrasounds in the differential diagnosis of periapical lesions*, J Endod 2008;34: 1312.
5. Alghaithy RA, Qualtrough AJE. *Pulp sensibility and vitality tests for diagnosing pulpal health in permanent teeth: a critical review*, Int Endod J 2017;50: 135.
6. Ali M, Baughman RA. *Maxillary odontogenic keratocyst a common and serious clinical misdiagnosis*, JADA 2003;134: 877.
7. Allen CE, Li L, Peters TL, et al: *Cell-specific gene expression in Langerhans cell histiocytosis lesions reveals a distinct profile compared with epidermal Langerhans cells*, J Immunol 2010;184: 4557.
8. Alsufyani NA, Lam EWN. *Osseous (cement-osseous) dysplasia of jaws: clinical and radiographic analysis*, J Can Dent Assoc 2011;77: 1.
9. American Association of Endodontists: *Endodontic*

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www.konkur.in

implications of medication-related osteonecrosis of the jaw, 2018. https://www.aae.org/specialty/wp-content/uploads/sites/2/2018/07/AAE_MedRelated_ONJ.pd

10. Aminoshariae A, Kulild JC, Syed A. *Cone-beam computed tomography compared with intraoral radiographic lesions in endodontic outcome studies: a systematic review*, J Endod 2018;44: 1626.
11. An CH, An SY, Choi BR. *Hard and soft tissue changes of osteomyelitis of the jaws on CT images*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;114: 118.
12. Ariji Y, Ariji E, Higuchi Y, et al: *Florid cemento-osseous dysplasia radiographic study with special emphasis on computed tomography*, Oral Surg Oral Med Oral Pathol 1994;78: 391.
13. Ariji Y, Izumi M, Gotoh M, et al: *MRI features of mandibular osteomyelitis: practical criteria based on an association with conventional radiography features and clinical classification*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: 503.
14. Badalian-Very G, Vergilio JA, Degar BA, et al: *Recurrent BRAF mutations in Langerhans cell histiocytosis*, Blood 2010;116: 1919.
15. Baden E, Saroff SA. *Periapical cemental dysplasia and periodontal disease. A case report with review of the literature*, J Periodontol 1986;58: 187.
16. Beacham JT, Geist JR, Yu Q, et al: *Accuracy of cone-beam computed tomographic image interpretation by endodontists and endodontic residents*, J Endod 2018;44: 571.
17. Becconsall-Ryan K, Tong D, Love RM. *Radiolucent inflammatory jaw lesions: a 20-year analysis*, Int Endod J 2010;43: 859.

Telegram: @dental_k

www.konkur.in

18. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone. Part I*, J Am Dent Assoc 1961;62: 152.
19. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone. Part II*, J Am Dent Assoc 1961;62: 708.
20. Bender IB. *Factors influencing the radiographic appearance of bony lesions*, J Endod 1982;8: 161.
21. Bender IB, Mori K. *The radiopaque lesion: a diagnostic consideration*, Endod Dent Traumatol 1985;1: 2.
22. Bender IB. *Paget's disease*, J Endod 2003;29: 720.
23. Berge TI, Helland SB, Sælen A, et al: *Pattern of recurrence of nonsyndromic keratocystic odontogenic tumors*, Oral Surg Oral Med Oral Pathol Oral Radiol 2016;122: 10.
24. Bernardi L, Visioli F, Nor C, et al: *Radicular cyst: an update of the biological factors related to lining epithelium*, J Endod 2015;41: 1951.
25. Berres ML, Merad M, Allen CE. *Progress in understanding the pathogenesis of Langerhans cell histiocytosis: back to histiocytosis X?*, Br J Haematol 2014;169: 3.
26. Beuselinck B, Wolter P, Karadimou A, et al: *Concomitant oral tyrosine kinase inhibitors and bisphosphonates in advanced renal cell carcinoma with bone metastases*, Br J Cancer 2012;107: 1665.
27. Beylouni I, Farge P, Mazoyer JF, et al: *Florid cemento-osseous dysplasia: report of a case with computed tomography and 3D imaging*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 707-711.
28. Bhaskar SN. *Oral surgery-oral pathology conference no. 17, Walter Reed Army Medical Center. Periapical lesions–*

Telegram: @dental_k

www.konkur.in

types, incidence, and clinical features, Oral Surg Oral Med Oral Pathol 1966;21: 657.
29. Bird JM, Owen RG, D'Sa S, et al: *Guidelines for the diagnosis and management of multiple myeloma*, Br J Haematol 2011;154: 32.
30. Bishay N, Petrikowski CG, Maxymiw WG, et al: *Optimum dental radiography in bone marrow transplant patients*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 375.
31. Black CC, Addante RR, Mohila CA. *Intraosseous ameloblastoma*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;110: 585.
32. Bohay RN. *The sensitivity, specificity, and reliability of radiographic periapical diagnosis of posterior teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 639.
33. Bomelli SR, Branstetter BF, Ferguson BF. *Frequency of a dental source for acute maxillary sinusitis*, Laryngoscope 2009;119: 580.
34. Bornstein MM, Wiest R, Balsiger R, et al: *Anterior Stafne's bone cavity mimicking a periapical lesion of endodontic origin: report of two cases*, J Endod 2009;35: 1598.
35. Bornstein MM, Bingisser AC, Reichart PA, et al: *Comparison between radiographic (2-dimensional and 3-dimensional) and histologic findings of periapical lesions treated with apical surgery*, J Endod 2015;41: 804.
36. Brynolf I. *Roentgenologic periapical diagnosis. II. One, two or more roentgenograms?*, Sven Tandlak Tidskr 1970;63: 345.
37. Budnick SD. *Compound and complex odontomas*, Oral Surg Oral Med Oral Pathol 1976;42: 501.

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www.konkur.in

38. Carillo C, Penarrocha M, Ortega B, et al: *Correlation of radiographic size and the presence of radiopaque lamina with histological findings in 70 periapical lesions*, J Oral Maxillofac Surg 2008;66: 1600.
39. Carrillo R, Morales A, Rodriguez-Peralto JL. *Benign fibro-osseous lesions in Paget's disease of the jaws*, Oral Surg Oral Med Oral Pathol 1991;71: 588.
40. Carter LC, Carney YL, Perez-Pudlewski D. *Lateral periodontal cyst. Multifactorial analysis of a previously unreported series*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 210.
41. Cheng YSL, Wright JM, Walstad WR, et al: *Osteosarcoma arising in Paget's disease of the mandible*, Oral Oncol 2002;38: 785.
42. Chirapathomsakul D, Sastravaha P, Jansisyanont P. *A review of odontogenic keratocysts and the behavior of recurrences*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 5.
43. Christodoulou C, Pervena A, Klouvas G, et al: *Combination of bisphosphonates and antiangiogenic factors induces osteonecrosis of the jaw more frequently than bisphosphonates alone*, Oncology 2009;76: 209.
44. Chuong R, Kaban LB, Kozakewich H, et al: *Central giant cell lesions of the jaws: a clinocopathologic study*, J Oral Maxillofac Surg 1986;44: 708.
45. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J Endod 2007;33: 1121.
46. Corral-Gudino L, Borao-Cengotita-Bengoa M, del Pino-Montes J, et al: *Epidemiology of Paget's disease of bone: a systematic review and meta-analysis of secular changes*, Bone 2013;55: 347.

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47. Cozzolino M, Galassi A, Conte F, et al: *Treatment of secondary hyperparathyroidism: the clinical utility of etelcalcetide*, Ther Clin Risk Manag 2017;13: 679.
48. Cunha EM, Fernandes AV, Versiani MA, et al: *Unicystic ameloblastoma: a possible pitfall in periapical diagnosis*, Int Endod J 2005;38: 334.
49. Cunha JF, Gomes CC, de Mesquita RA. *Clinicopathologic features associated with recurrence of the odontogenic keratocyst: a cohort retrospective analysis*, Oral Surg Oral Med Oral Pathol Oral Radiol 2016;121: 629.
50. Dagenais M, Pharoah MJ, Sikorski PA. *The radiographic characteristics of histiocy- tosis X: a study of 29 cases that involve the jaws*, Oral Surg Oral Med Oral Pathol 1992;74: 230.
51. de Carvalhosa AA, Zandonade RM, de Souza Castro PH, et al: *8-Year follow-up of central giant cell lesion mimicking apical periodontitis*, J Endod 2014;40: 1708.
52. de Courten A, Küffer R, Samson J, et al: *Anterior lingual mandibular salivary gland defect (Stafne defect) presenting as a residual cyst*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 460.
53. de Lange J, van den Akker HP. *Clinical and radiological features of central giant-cell lesions of the jaw*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 464.
54. de Lange J, van den Akker HP, van den Berg H. *Central giant cell granuloma of the jaw: a review of the literature with emphasis on therapy options*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 603.
55. Delai D, Bernardi A, Felippe GS, et al: *Florid cemento-osseous dysplasia: a case of misdiagnosis*, J Endod 2015;41: 1923.

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56. de Noronha Santos Netto J, Machado Cerri J, Miranda AM, et al: *Benign fibro-osseous lesions: clinicopathologic features from 143 cases diagnosed in an oral diagnosis setting*, Oral Surg Oral Med Oral Pathol Oral Radiol 2013;115: e56.
57. de Paula-Silva FW, Wu MK, Leonardo MR, et al: *Accuracy of periapical radiography and cone-beam computed tomography scans in diagnosing apical periodontitis using histopathological findings as a gold standard*, J Endod 2009;35: 1009.
58. Dereci O, Duran S. *Intraorally exposed anterior Stafne bone defect: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2012;113: e1.
59. Dewan K, Owens J, Silvester K. *Maintaining a high level of suspicion for recurrent malignant disease: report of a case with periapical involvement*, Int Endod J 2007;40: 900.
60. Di Nardo D, Gambarini G, Capuani S, et al: *Nuclear magnetic resonance imaging in endodontics: a review*, J Endod 2018;44: 356.
61. Dolan JM, DeGraft-Johnson A, McDonald N, et al: *Maxillary and mandibular non-hodgkin lymphoma with concurrent periapical endodontic disease: diagnosis and management*, J Endod 2017;43: 1744.
62. Drazić R, Minić AJ. *Focal cemento-osseous dysplasia in the maxilla mimicking periapical granuloma*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88: 87.
63. D'Silva NJ, Summerlin DJ, Cordell KG, et al: *Metastatic tumors in the jaws: a retrospective study of 114 cases*, J Am Dent Assoc 2006;137: 1667.
64. Dym H, Zeidan J. *Microbiology of acute and chronic osteomyelitis and antibiotic treatment*, Dent Clin North

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www.konkur.in

Am 2017;61: 271.

65. Eberhardt JA, Torabinejad M, Christiansen EL. *A computed tomographic study of the distances between the maxillary sinus floor and the apices of the maxillary posterior teeth*, Oral Surg Oral Med Oral Pathol 1992;73: 345.
66. Ebihara A, Yoshioka T, Suda H. *Garrè's osteomyelitis managed by root canal treatment of a mandibular second molar: incorporation of computed tomography with 3D reconstruction in the diagnosis and monitoring of the disease*, Int Endod J 2005;38: 255.
67. Edwards R, Altalibi M, Flores-Mir C., The frequency and nature of incidental findings in cone-beam computed tomographic scans of the head and neck region. *a systematic review*, J Am Dent Assoc 2013;144: 161.
68. Eliasson S, Halvarsson C, Ljungheimer C. *Periapical condensing osteitis and endodontic treatment*, Oral Surg 1984;57: 195.
69. El-Naggar AK, Chan JKC, Grandis JR, et al: WHO classification of head and neck tumours ed 4 2017; IARC Lyon.
70. Enciso R, Keaton J, Saleh N, et al: *Assessing the utility of serum C-telopeptide cross-link of type 1 collagen as a predictor of bisphosphonate-related osteonecrosis of the jaw - a systematic review and meta-analysis*, JADA 2016;147: 551.
71. Epstein JB, Voss NJ, Stevenson-Moore P. *Maxillofacial manifestations of multiple myeloma. An unusual case and review of the literature*, Oral Surg Oral Med Oral Pathol 1984;57: 267.
72. Epstein JB, Epstein JD, Le ND, et al: *Characteristics of oral and paraoral malignant lymphoma: a population-*

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www.konkur.in

based review of 361 cases, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 519.

73. Erian D, Quek SYP, Subramanian G. *The importance of the history and clinical examination*, JADA 2018;149: 807.
74. Estilo CL, Fornier M, Farooki A, et al: *Osteonecrosis of the jaw related to bevacizumab*, J Clin Oncol 2008;26: 4037.
75. Estrela C, Bueno MR, Leles CR, et al: *Accuracy of cone beam computed tomography and panoramic and periapical radiography for detection of apical periodontitis*, J Endod 2008;34: 273.
76. Eversole LR, Stone CE, Strub D. *Focal sclerosing osteomyelitis/focal periapical osteopetrosis: radiographic patterns*, Oral Surg 1984;58: 456.
77. Faitaroni LA, Bueno MR, De Carvalhosa AA. *Ameloblastoma suggesting large apical periodontitis*, J Endod 2008;34: 216.
78. Faitaroni LA, Bueno MR, Carvalhosa AA, et al: *Differential diagnosis of apical periodontitis and nasopalatine duct cyst*, J Endod 2011;37: 403.
79. Fantasia JE. *Lateral periodontal cyst. An analysis of forty-six cases*, Oral Surg Oral Med Oral Pathol 1979;48: 237.
80. Ficarra G, Beninati F, Rubino I, et al: *Osteonecrosis of the jaws in periodontal patients with a history of bisphosphonates treatment*, J Clin Periodontol 2005;32: 1123.
81. Finkelstein MW, Hellstein JW, Lake KS. *Keratocystic odontogenic tumor: a retrospective analysis of genetic, immunohistochemical and therapeutic features. Proposal of a multicenter clinical survey tool*, Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116: 75.

Telegram: @dental_k

www.konkur.in

82. Fischer DJ, Klasser GD, Kaufmann R. *Intraoral swelling and periapical radiolucency*, JADA 2012;143: 985.
83. Fitzpatrick SG, Dashti H, Cohen DM, et al: *Alveolar ridge mass with multifocal intraosseous radiolucent lesions*, JADA 2011;142: 411.
84. Fizazi K, Carducci M, Smith M, et al: *Denosumab versus zoledronic acid for treatment of bone metastases in men with castration-resistant prostate cancer: a randomised, double-blind study*, Lancet 2011;377: 813.
85. Fleissig Y, Regev E, Lehman H. *Sunitinib related osteonecrosis of jaw: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113: e1.
86. Frazier KB, Baker PS, Abdelsayed R, et al: *A case report of subpontic osseous hyperplasia in the maxillary arch*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 73.
87. Fujihara H, Chikazu D, Saijo H, et al: *Metastasis of hepatocellular carcinoma into the mandible with radiographic findings mimicking a radicular cyst: a case report*, J Endod 2010;36: 1593.
88. Gallego Romero D, Torres Lagares D, Garcia Calderon M, et al: *Differential diagnosis and therapeutic approach to periapical cysts in daily dental practice*, Med Oral 2002;7: 59.
89. Gander T, Obwegeser JA, Zemann W, et al: *Malignancy mimicking bisphosphonate-associated osteonecrosis of the jaw: a case series and literature review*, Oral Surg Oral Med Oral Pathol Oral Radiol 2014;117: 32.
90. Gardner D. *Pseudocysts and retention cysts of the maxillary sinus*, Oral Surg Oral Med Oral Pathol 1984;58: 561.
91. Garlock JA, Pringle GA, Hicks ML. *The odontogenic*

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www.konkur.in

keratocyst, a potential endodontic misdiagnosis, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 452.

92. Gedmintas L, Solomon DH, Kim SC. *Bisphosphonates and risk of subtrochanteric, femoral shaft, and atypical femur fracture: a systematic review and meta-analysis*, J Bone Miner Res 2013;28: 1729.
93. Geist JR, Katz JO. *The frequency and distribution of idiopathic osteosclerosis*, Oral Surg Oral Med Oral Pathol 1990;69: 388.
94. Ghandi D, Ayoub AF, Pogrel MA, et al: *Ameloblastoma: a surgeon's dilemma*, J Oral Maxillofac Surg 2006;64: 1010.
95. Goldman M, Pearson A, Darzenta N. *Reliability of radiographic interpretations*, Oral Surg 1974;38: 340.
96. Goltzman D. *Osteolysis and cancer*, J Clin Invest 2001;107: 1219.
97. Gondak RO, Rocha AC, Neves Campos JG, et al: *Unicystic ameloblastoma mimicking apical periodontitis: a case series*, J Endod 2013;39: 145.
98. Gopikrishna V, Pradeep G, Venkateshbabu N. *Assessment of pulp vitality: a review*, Int J Paediatr Dent 2009;19: 3.
99. Green TL, Walton RE, Clark JM, et al: *Histologic examination of condensing osteitis in cadaver specimens*, J Endod 2013;39: 977.
100. Guarneri V, Miles D, Robert N, et al: *Bevacizumab and osteonecrosis of the jaw: incidence and association with bisphosphonate therapy in three large prospective trials in advanced breast cancer*, Breast Cancer Res Treat 2010;122: 181.
101. Guo J, Simon JH, Sedghizadeh P, et al: *Evaluation of the reliability and accuracy of using cone-beam computed tomography for diagnosing periapical cysts from*

Telegram: @dental_k

www.konkur.in

granulomas, J Endod 2013;39: 1485.
102. Guo Y, Wang D, Wang Y, et al: *Imaging features of medicine-related osteonecrosis of the jaws: comparison between panoramic radiography and computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol 2016;122: e69.
103. Gurol M, Burkes EJ, Jacoway J. *Botryoid odontogenic cyst: analysis of 33 cases*, J Periodontol 1995;66: 1069.
104. Halse A, Molven O. *Idiopathic osteosclerosis of the jaws followed through a period of 20–27 years*, Int Endod J 2002;35: 747.
105. Hariya Y, Yuasa K, Nakayama E, et al: *Value of computed tomography findings in differentiating between intraosseous malignant tumors and osteomyelitis of the mandible affecting the masticator space*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 503.
106. Hatzenbuehler J, Pulling TJ. *Diagnosis and management of osteomyelitis*, Am Fam Physician 2011;84: 1027.
107. Hegde RJ, Shetty R. *Nasopalatine duct cyst*, J Indian Soc Pedod Prev Dent 2006;24: 31.
108. Hegtvedt AK, Terry BC, Burkes EJ, et al: *Skin graft vestibuloplasty exostosis: a report of two cases*, Oral Surg Oral Med Oral Pathol 1990;69: 149.
109. Heikinheimo K, Kurppa KJ, Elenius K. *Novel targets for the treatment of ameloblastoma*, J Dent Res 2015;94: 237.
110. Hellstein JW, Adler RA, Edwards B, et al: *Managing the care of patients receiving antiresorptive therapy for prevention and treatment of osteoporosis: executive summary of recommendations from the American Dental Association Council on scientific affairs*, J Am Dent Assoc 2011;142: 1243.
111. Henry DH, Costa L, Goldwasser F, et al: *Randomized,*

Telegram: @dental_k

www.konkur.in

double-blind study of denosumab versus zoledronic acid in the treatment of bone metastases in patients with advanced cancer (excluding breast and prostate cancer) or multiple myeloma, J Clin Oncol 2011;29: 1125.

112. Hicks J, Flaitz CM. *Langerhans cell histiocytosis: current insights in a molecular age with emphasis on clinical oral and maxillofacial pathology practice*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 42.
113. Hirshberg A, Leibovich P, Buchner A. *Metastatic tumors of the jawbones: analysis of 390 cases*, J Oral Pathol Med 1994;23: 337.
114. Hirshberg A, Berger R, Allon I, et al: *Metastatic tumors to the jaws and mouth*, Head and Neck Pathol 2014;8: 463.
115. Hopp RN, Pucci J, Santos-Silva AR, et al: *Osteonecrosis after administration of intravitreous bevacizumab*, J Oral Maxillofac Surg 2012;70: 632.
116. Ida M, Tetsumura T, Sasaki T. *Periosteal reaction new bone formation in the jaw. A computed tomographic study*, Dentomaxillofac Radiol 1997;26: 169.
117. Imaizumi A, Kuribayashi A, Watanabe H, et al: *Non-Hodgkin lymphoma involving the mandible: imaging findings*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113: e33.
118. Jafarzadeh H, Abbott PV. *Review of pulp sensibility tests. Part I: general information and thermal tests*, Int Endod J 2010;43: 738.
119. Jafarzadeh H, Abbott PV. *Review of pulp sensibility tests. Part II: electric pulp tests and test cavities*, Int Endod J 2010;43: 945.
120. Jalali P, Kim SG. *Multiple periradicular radiolucencies mimicking endodontic lesions in renal osteodystrophy of the mandible: a case report*, Int Endod J 2016;49: 706.

Telegram: @dental_k

www.konkur.in

121. Johnson NR, Batstone MD, Savage NW. *Management and recurrence of keratocystic odontogenic tumor: a systematic review*, Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116: e271.
122. Johnson J, Jundt J, Hanna I, et al: *Resection of an ameloblastoma in a pediatric patient and immediate reconstruction using a combination of tissue engineering and costochondral rib graft*, JADA 2017;148: 40.
123. Kakehashi S, Stanley H, Fitzgerald R. *The effect of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral Pathol 1965;20: 340.
124. Kaneda T, Minami M, Ozawa K, et al: *Magnetic resonance imaging of osteomyelitis in the mandible. Comparative study with other radiologic modalities*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 634.
125. Kang SH, Kim BS, Kim Y. *Proximity of posterior teeth to the maxillary sinus and buccal bone thickness: a biometric assessment using cone-beam computed tomography*, J Endod 2015;41: 1839.
126. Katz J, Chaushu G, Rotstein I. *Stafne's bone cavity in the anterior mandible: a possible diagnostic challenge*, J Endod 2001;27: 304.
127. Kaugars GE. *Botryoid odontogenic cyst*, Oral Surg Oral Med Oral Pathol 1986;62: 555.
128. Kawai T, Murakami S, Sakuda M. *Radiographic investigation of mandibular periostitis ossificans in 55 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 704.
129. Kawai T, Murakami S, Kishino M, et al: *Gigantic dense bone island of the jaw*, Oral Surg Oral Med Oral Pathol

Telegram: @dental_k

www.konkur.in

Oral Radiol Endod 1996;82: 108.
130. Kawai T, Hiranuma H, Kishino M, et al: *Cemento-osseous dysplasia of the jaws in 54 patients: a radiographic study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 107.
131. Kawasaki G, Nakai M, Mizuno A, et al: *Malignant lymphoma of the mandible report of a case*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 345.
132. Kaya M, Şimşek-Kaya G, Gürsan N, et al: *Local treatment of chronic osteomyelitis with surgical debridement and tigecycline-impregnated calcium hydroxyapatite: an experimental study*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113: 340.
133. Keller EE, Gunderson LL. *Bone disease metastatic to the jaws*, JADA 1987;115: 697.
134. Kemp S, Gallagher G, Kabani S, et al: *Oral non-Hodgkin's lymphoma: review of the literature and World Health Organization classification with reference to 40 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: 194.
135. Khalili M, Mahboobi N, Shams J. *Metastatic breast carcinoma initially diagnosed as pulpal/ periapical disease: a case report*, J Endod 2010;36: 922.
136. Koch FP, Walter C, Hansen T, et al: *Osteonecrosis of the jaw related to sunitinib*, Oral Maxillofac Surg 2011;15: 63.
137. Koivisto T, Bowles WR, Rohrer M. *Frequency and distribution of radiolucent jaw lesions: a retrospective analysis of 9,723 cases*, J Endod 2012;38: 729.
138. Koivisto T, Bowles WR, Magajna WA, et al: *Malignant lymphoma in maxilla with cystic involvement: a case report*, J Endod 2013;39: 935.
139. Kontogiannis TG, Tosios KI, Kerezoudis NP, et al:

Telegram: @dental_k

www.konkur.in

Periapical lesions are not always a sequelae of pulpal necrosis: a retrospective study of 1521 biopsies, Int Endod J 2015;48: 68.

140. Koorbusch GF, Fotos P, Goll KT. *Retrospective assessment of osteomyelitis. Etiology, demographics, risk factors, and management in 35 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1992;74: 149.
141. Kuc I, Peters E, Pan J. *Comparison of clinical and histologic diagnoses in periapical lesions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 333.
142. Kruse C, Spin-Neto R, Wenzel A, et al: *Cone beam computed tomography and periapical lesions: a systematic review analysing studies on diagnostic efficacy by a hierarchical model*, Int Endod J 2015;48: 815.
143. Kruse-Lösler B, Diallo R, Gaertner C, et al: *Central giant cell granuloma of the jaws: a clinical, radiologic, and histopathologic study of 26 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 346.
144. Kumar SK, Rajkumar SV, Dispenzieri A, et al: *Improved survival in multiple myeloma and the impact of novel therapies*, Blood 2008;111: 2516.
145. Kyrgidis A, Vahtsevanos K, Koloutsos G, et al: *Bisphosphonate-related osteonecrosis of the jaws: a case-control study of risk factors in breast cancer patients*, J Clin Oncol 2008;26: 4634.
146. Lalonde ER, Luebke RG. *The frequency and distribution of periapical cysts and granulomas. An evaluation of 800 specimens*, Oral Surg Oral Med Oral Pathol 1965;25: 861.
147. Landgren O, Weiss BM. *Patterns of monoclonal gammopathy of undetermined significance and multiple myeloma in various ethnic/racial groups: support for genetic factors in pathogenesis*, Leukemia 2009;23: 1691.

Telegram: @dental_k

www.konkur.in

148. Lee JI, Kang SJ, Jeon SP, et al: *Stafne bone cavity of the mandible*, Arch Craniofac Surg 2016;17: 162.
149. Lee SH, Huang JJ, Pan WL, et al: *Gingival mass as the primary manifestation of multiple myeloma: report of two cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 75.
150. Leonardi Dutra K, Haas L, Porporatti AL, et al: *Diagnostic accuracy of cone-beam computed tomography and conventional radiography on apical periodontitis: a systematic review and meta-analysis*, J Endod 2016;42: 356.
151. Lim LZ, Padilla RJ, Reside GJ, et al: *Comparing panoramic radiographs and cone beam computed tomography: impact on radiographic features and differential diagnoses*, Oral Surg Oral Med Oral Pathol Oral Radiol 2018;126: 63.
152. Lin LM, Huang GT, Rosenberg PA. *Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after periapical wound healing*, J Endod 2007;33: 908.
153. Lizio G, Salizzoni E, Coe M, et al: *Differential diagnosis between a granuloma and radicular cyst: effectiveness of magnetic resonance imaging*, Int Endod J 2018;51: 1077.
154. Lojo Oliveira L, Torrijos Eslava A. *Treatment of Paget's disease of bone*, Reumatol Clin 2012;8: 220.
155. Loushine RJ, Weller RN, Kimbrough WF, et al: *Secondary hyperparathyroidism: a case report*, J Endod 2003;29: 272.
156. Love RM, Firth N. *Histopathological profile of surgically removed persistent periap- ical radiolucent lesions of endodontic origin*, Int Endod J 2009;42: 198.
157. Lyons SF, Liebowitz DN. *The roles of human viruses in*

Telegram: @dental_k

www.konkur.in

the pathogenesis of lymphoma, Semin Oncol 1998;25: 461.
158. MacDonald-Jankowski DS. *Florid cemento-osseous dysplasia: a systematic review*, Dentomaxillofac Radiol 2003;32: 141.
159. MacDonald-Jankowski DS. *Focal cemento-osseous dysplasia: a systematic review*, Dento Maxillo Facial Radiology 2008;37: 350.
160. Maillet M, Bowles WR, McClanahan SL. *Cone-Beam computed tomography evaluation of maxillary sinusitis*, J Endod 2011;37: 753.
161. Marotti J, Heger S, Tinschert J, et al: *Recent advances of ultrasound imaging in dentistry—a review of the literature*, Oral Surg Oral Med Oral Pathol Oral Radiol 2013;115: 819.
162. Marx RE. *Pamidronate (Aredia) and zoledronate (Zometa) induced avascular necrosis of the jaws: a growing epidemic*, J Oral Maxillofac Surg 2003;61: 1115.
163. Marx RE, Cillo JE, Ulloa JJ. *Oral bisphosphonate-induced osteonecrosis: risk factors, prediction of risk using serum CTX testing, prevention, and treatment*, J Oral Maxillofac Surg 2007;65: 2397.
164. Matsuzaki H, Hara M, Yanagi Y, et al: *Magnetic resonance imaging (MRI) and dynamic MRI evaluation of extranodal non-Hodgkin lymphoma in oral and maxillofacial regions*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113: 126.
165. Mawardi H, Cutler C, Treister N. *Medical management update: Non-Hodgkin lymphoma*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107: e19.
166. McCadden L, Leonard CG, Primrose WJ. *Bisphosphonate-induced osteonecrosis of the ear canal: our experience and a review of the literature*, J Laryngol

Telegram: @dental_k

www.konkur.in

Otol 2018;132: 372.
167. McCarthy PJ, McClanahan S, Hodges J, et al: *Frequency of localization of the painful tooth by patients presenting for an endodontic emergency*, J Endod 2010;36: 801.
168. Mejare IA, Bergenholtz G, Petersson K, et al: *Estimates of sensitivity and specificity of electric pulp testing depend on pulp disease spectrum: a modelling study*, Int Endod J 2015;48: 74.
169. Mehra P, Murad H. *Maxillary sinus disease of odontogenic origin*, Otolaryngol Clin North Am 2004;37: 347.
170. Mendonca EF, Sousa TO, Estrela C. *Non-Hodgkin lymphoma in the periapical region of an aandibular canine*, J Endod 2013;39: 839.
171. Morton TH Jr, Natkin E. *Hyperostosis and fixed partial denture pontics: report of 16 patients and review of literature*, J Prosthet Dent 1990;64: 539.
172. Motamedi MH, Eshghyar N, Jafari SM, et al: *Peripheral and central giant cell granulomas of the jaws: a demographic study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: e39.
173. Nair PN, Pajarola G, Schroeder HE. *Types and incidence of human periapical lesions obtained with extracted teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 93.
174. Nair PNR. *New perspectives on radicular cysts: do they heal?*, Int Endod J 1998;31: 155.
175. Nair PNR, Sundqvist G, Sjogren U. *Experimental evidence supports the abscess theory of development of radicular cysts*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 294.
176. Natkin E, Oswald RJ, Carnes LI. *The relationship of*

Telegram: @dental_k

www.konkur.in

lesion size to diagnosis, incidence, and treatment of periapical cysts and granulomas, Oral Surg Oral Med Oral Pathol 1984;57: 82.

177. Nelson BL, Linfesty RL. *Nasopalatine duct cyst*, Head Neck Pathol 2010;4: 121.
178. Neville BW, Albenesius RJ. *The prevalence of benign fibro-osseous lesions of periodontal ligament origin in black women: a radiographic survey*, Oral Surg Oral Med Oral Pathol 1986;62: 340.
179. Neville BW, Damm DD, Allen CM, et al: Oral and maxillofacial pathology ed 4 2016; Elsevier St Louis.
180. Nicolatou-Galitis O, Migkou M, Psyrri A, et al: *Gingival bleeding and jaw bone necrosis in patients with metastatic renal cell carcinoma receiving sunitinib: report of 2 cases with clinical implications*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113: 234.
181. Nikitakis NG, Brooks JK, Melakopoulos I, et al: *Lateral periodontal cysts arising in periapical sites: a report of two cases*, J Endod 2010;36: 1707.
182. Nohl FSA, Gulabivala K. *Odontogenic keratocyst as periradicular radiolucency in the anterior mandible*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 103.
183. Nurbaksh B, Friedman S, Kulkarni GV, et al: *Resolution of maxillary sinus mucositis after endodontic treatment of maxillary teeth with apical periodontitis: a cone-beam computed tomography pilot study*, J Endod 2011;37: 1504.
184. Oberli K, Bornstein MM, von Arx T. *Periapical surgery and the maxillary sinus: radiographic parameters for clinical outcome*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 848.
185. Oser DG, Henson BR, Shiang EY, et al: *Incidental*

Telegram: @dental_k

www.konkur.in

findings in small field of view cone-beam computed tomography scans, J Endod 2017;43: 901.

186. Otter R, Gerrits WB, vd Sandt MM, et al: *Primary extranodal and nodal non-Hodgkin's lymphoma. A survey of a population-based registry*, Eur J Cancer Clin Oncol 1989;25: 1203.
187. Pace R, Cairo F, Giuliani V, et al: *A diagnostic dilemma: endodontic lesion or odontogenic keratocyst? A case presentation*, Int Endod J 2008;41: 800.
188. Pak JG, Fayazi S, Shane N, et al: *Prevalence of periapical radiolucency and root canal treatment: a systematic review of cross-sectional studies*, J Endod 2012;38: 1170.
189. Pack ARC, Gaudie WM, Jennings AM. *Bony exostosis as a sequela to free gingival grafting: two case reports*, J Periodontol 1991;62: 269.
190. Parker JM, Mol A, Rivera EM, et al: *Cone-beam computed tomography uses in clinical endodontics: observer variability in detecting periapical lesions*, J Endod 2017;43: 184.
191. Patel NA, Ferguson BJ. *Odontogenic sinusitis: an ancient but underappreciated cause of maxillary sinusitis*, Curr Opin Otoloaryngol Head Neck Surg 2012;20: 24.
192. Patel S, Wilson R, Dawood A, et al: *The detection of periapical pathosis using periapical radiography and cone beam computed tomography – part 1: preoperative status*, Int Endod J 2012;8: 702.
193. Patel S, Durack C, Abella F, et al: *Cone beam computed tomography in endodontics: a review*, Int Endod J 2015;48: 3.
194. Pereira DL, Fernandes DT, Santos-Silva AR, et al: *Intraosseous non-Hodgkin lymphoma mimicking a*

Telegram: @dental_k

www.konkur.in

periapical lesion, J Endod 2015;41: 1738.
195. Perumal CJ. *An unusually large destructive nasopalatine duct cyst: a case report*, J Maxillofac Oral Surg 2011;12: 100.
196. Peters SM, Pastagia J, Yoon AJ, et al: *Langerhans cell histiocytosis mimicking periapical pathology in a 39-year-old man*, J Endod 2017;43: 1909.
197. Petersson K, Soderstrom C, Kiani-Anaraki M, et al: *Evaluation of the ability of thermal and electric tests to register pulp vitality*, Endod Dent Traumatol 1999;15: 127.
198. Petrikowski CG, Peters E. *Longitudinal radiographic assessment of dense bone islands of the jaws*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 627.
199. Phelan JA, Kritchman D, Fusco-Ramer M, et al: *Recurrent botryoid odontogenic cyst (lateral periodontal cyst)*, Oral Surg Oral Med Oral Pathol 1988;66: 345.
200. Piattelli A, Perfetti G, Carraro A. *Complex odontoma as a periapical and interradicular radiopacity in a primary molar*, J Endod 1996;22: 561.
201. Pontes FSC, Lopes MA, de Souza LL, et al: *Oral and maxillofacial manifestations of chronic kidney disease-mineral and bone disorder: a multicenter retrospective study*, Oral Surg Oral Med Oral Pathol Oral Radiol 2018;125: 31.
202. Pope O, Sathorn C, Parashos P. *A comparative investigation of cone-beam computed tomography and periapical radiography in the diagnosis of a healthy periapex*, J Endod 2014;40: 360.
203. Pratt G. *The evolving use of serum free light chain assays in haematology*, Br J Haematol 2008;141: 413.
204. Rajkumar SV, Dimopoulos MA, Palumbo A, et al: *International myeloma working group updated criteria for*

Telegram: @dental_k

www.konkur.in

the diagnosis of multiple myeloma, Lancet Oncol 2014;15: e538.

205. Ralston SH, Layfield R. *Pathogenesis of Paget disease of bone*, Calcif Tissue Int 2012;91: 97.
206. Raubenheimer EJ, Noffke CE, Mohamed A. *Expansive jaw lesions in chronic kidney disease: review of the literature and a report of two cases*, Oral Surg Oral Med Oral Pathol Oral Radiol 2015;119: 340.
207. Reddi AH, Roodman D, Freeman C, et al: *Mechanisms of tumor metastasis to the bone: challenges and opportunities*, J Bone Miner Res 2003;18: 190.
208. Ricucci D, Mannocci F, Ford TR. *A study of periapical lesions correlating the presence of a radiopaque lamina with histological findings*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 389.
209. Ricucci D, Loghin S, Siqueira JF. *Correlation between clinical and histologic pulp diagnose*, J Endod 2014;40: 1932.
210. Ritter L, Lutz J, Neugebauer J, et al: *Prevalence of pathologic findings in the maxillary sinus in cone-beam computerized tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111: 634.
211. Rizzoli R, Akesson K, Bouxsein M, et al: *Subtrochanteric fractures after long-term treatment with bisphosphonates: a European Society on Clinical and Economic Aspects of Osteoporosis and Osteoarthritis, and International Osteoporosis Foundation working group report*, Osteoporos Int 2010;22: 373.
212. Rosenberg PA, Frisbie J, Lee J, et al: *Evaluation of pathologists (histopathology) and radiologists (cone beam computed tomography) differentiating radicular cysts from granulomas*, J Endod 2010;36: 423.

Telegram: @dental_k

www.konkur.in

213. Ruggiero SL, Mehrotra B, Rosenberg TJ, et al: *Osteonecrosis of the jaws associated with the use of bisphosphonates: a review of 63 cases*, J Oral Maxillofac Surg 2004;62: 527.
214. Ruggiero SL, Dodson TB, Fantasia J, et al: *American Association of Oral and Maxillofacial Surgeons position paper on medication-related osteonecrosis of the jaw: 2014 update*, J Oral and Maxillofac Surg 2014;72: 1938.
215. Sankar D, Muthusubramanian V, Nathan JA, et al: *Aggressive nasopalatine duct cyst with complete destruction of palatine bone*, J Pharm Bioallied Sci 2016;8: S185.
216. Santos JN, Carneiro Junior B, Alves Malaquias PDTI, et al: *Keratocystic odontogenic tumour arising as a periapical lesion*, Int Endod J 2014;47: 802.
217. Saad F, Brown JE, Van Poznak C, et al: *Incidence, risk factors, and outcomes of osteonecrosis of the jaw: integrated analysis from three blinded active-controlled phase III trials in cancer patients with bone metastases*, Ann Oncol 2012;23: 1341.
218. Sapp JP, Eversole LR, Wysocki GP. Comtemporary oral and maxillofacial pathology ed 2 2002; Mosby St Louis.
219. Sauk JJ, Nikitakis NG, Scheper MA. *Are we on the brink of nonsurgical treatment for ameloblastoma?*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;110: 68.
220. Saund D, Kotecha S, Rout J, et al: *Non-resolving periapical inflammation: a malignant deception*, Int Endod J 2010;43: 84.
221. Schneider LC, Mesa ML, Brickman LH. *Complications of endodontic therapy in florid osseous dysplasia*, Oral Surg Oral Med Oral Pathol 1987;64: 114.
222. Schneider LC, Mesa ML. *Differences between florid*

Telegram: @dental_k

www.konkur.in

osseous dysplasia and chronic diffuse sclerosing osteomyelitis, Oral Surg Oral Med Oral Pathol 1990;70: 308.

223. Scolozzi P, Martinez A, Richter M, et al: *A nasopalatine duct cyst in a 7-year-old child*, Pediatr Dent 2008;30: 530.
224. Seitz S, Priemel M, Zustin J, et al: *Paget's disease of bone: histologic analysis of 754 patients*, J Bone Miner Res 2009;24: 62.
225. Selden HS, Manhoff DT, Hatges NA, et al: *Metastatic carcinoma to the mandible that mimicked pulpal/periodontal disease*, J Endod 1998;24: 267.
226. Selden HS. *Central giant cell granuloma: a troublesome lesion*, J Endod 2000;26: 371.
227. Senia ES, Sarao MS. *Periapical cemento-osseous dysplasia: a case report with twelve-year follow-up and review of literature*, Int Endod J 2015;48: 1086.
228. Seltzer S, Bender IB, Ziontz M. *The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Part I*, Oral Surg Oral Med Oral Pathol 1963;16: 846.
229. Seltzer S, Bender IB, Ziontz M. *The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Part II*, Oral Surg Oral Med Oral Pathol 1963;16: 969.
230. Shoha RR, Dowson J, Richards AG. *Radiographic interpretation of experimentally produced bony lesions*, Oral Surg Oral Med Oral Pathol 1974;38: 294.
231. Simon JH. *Incidence of periapical cysts in relation to the root canal*, J Endod 1980;6: 845.
232. Simon JH, Enciso R, Malfaz JM, et al: *Differential diagnosis of large periapical lesions using cone-beam computed tomography measurements and biopsy*, J Endod

Telegram: @dental_k

www.konkur.in

2006;32: 833.
233. Singer SR, Mupparapu M, Rinaggio J. *Florid cemento-osseous dysplasia and chronic diffuse osteomyelitis report of a simultaneous presentation and review of the literature*, JADA 2005;136: 927.
234. Siponen M, Neville BW, Damm DD, et al: *Multifocal lateral periodontal cysts: a report of 4 cases and review of the literature*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111: 225.
235. Sirotheau Corrêa Pontes F, Paiva Fonseca F, Souza de Jesus A, et al: *Nonendodontic lesions misdiagnosed as apical periodontitis lesions: series of case reports and review of literature*, J Endod 2014;40: 16.
236. Spatafore CM, Keyes G, Skidmore AE. *Lymphoma: an unusual oral presentation*, J Endod 1989;15: 438.
237. Spatafore CM, Griffin JA, Keyes GG, et al: *Periapical biopsy report: an analysis of over a 10-year period*, J Endod 1990;16: 239.
238. Speight PM, Takata T. *New tumour entities in the 4th edition of the World Health Organization classification of head and neck tumours: odontogenic and maxillofacial bone tumours*, Virchows Arch 2018;472: 331.
239. Stafne EC. *Bone cavities situated near the angle of the mandible*, J Am Dent Assoc 1942;29: 1969.
240. Stoopler ET, Vogl DT, Stadtmauer EA. *Medical management update: multiple myeloma*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 599.
241. Stopeck A, Body JJ, Fujiwara Y, et al: *Denosumab versus zolendronic acid for the treatment of breast cancer patients with bone metastases: results of a randomized phase 3 study*, Eur J Cancer Supplements 2009;7: 2.
242. Suei Y, Taguchi A, Tanimoto K. *Diagnosis and*

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www.konkur.in

classification of mandibular osteomyelitis, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 207.

243. Sullivan M, Gallagher G, Noonan V. *The root of the problem: occurrence of typical and atypical periapical pathoses*, JADA 2016;147: 646.
244. Summerlin DJ, Tomich CE. *Focal cemento-osseous dysplasia: a clinicopathologic study of 221 cases*, Oral Surg Oral Med Oral Pathol 1994;78: 611.
245. Sun L, Sun Z, Ma X. *Multiple complex odontoma of the maxilla and the mandible*, Oral Surg Oral Med Oral Pathol Oral Radiol 2015;120: e11.
246. Tataryn RW. *American Association of Endodontists*. Endodontics: Colleagues for Excellence. Maxillary sinusitis of endodontic origin, 2018. https://www.aae.org/specialty/newsletter/maxillary-sinusitis-of-endodontic-origin/.
247. Ten Cate AR. *The epithelial cell rests of Malassez and the genesis of the dental cyst*, Oral Surg Oral Med Oral Pathol 1972;34: 56.
248. Tewary S, Luzzo J, Hartwell G. *Endodontic radiography: who is reading the digital radiograph?*, J Endod 2011;37: 919.
249. Tian XM, Qian L, Xin XZ, et al: *An analysis of the proximity of maxillary posterior teeth to the maxillary sinus using cone-beam computed tomography*, J Endod 2016;42: 371.
250. Titinchi F, Nortje CJ. *Keratocystic odontogenic tumor: a recurrence analysis of clinical and radiographic parameters*, Oral Surg Oral Med Oral Pathol Oral Radiol 2012;114: 136.
251. Toller PA. *The osmolality of fluids from cysts of the jaws*, Br Dent J 1970;129: 275.

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252. Tong AC, Ng IO, Yeung KM. *Osteomyelitis with proliferative periostitis: an unusual case*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: e14.
253. Torabinejad M. *The role of immunological reactions in apical cyst formation and the fate of epithelial cells after root canal therapy: a theory*, Int J Oral Surg 1983;12: 14.
254. Torabinejad M, Rice DD, Maktabi O, et al: *Prevalence and size of periapical radiolucencies using cone-beam computed tomography in teeth without apparent intraoral radiographic lesions: a new periapical index with a clinical recommendation*, J Endod 2018;44: 389.
255. Torregrossa VR, Faria KM, Bicudo MM, et al: *Metastatic cervical carcinoma of the jaw presenting as periapical disease*, Int Endod J 2016;49: 203.
256. Treister NS, Friedland B, Woo SB. *Use of cone-beam computerized tomography for evaluation of bisphosphonate-associated osteonecrosis of the jaws*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: 753.
257. Troeltzsch M, Oduncu F, Mayr D, et al: *Root resorption caused by jaw infiltration of multiple Mmyeloma: report of a case and literature review*, J Endod 2014;40: 1260.
258. Trope M, Pettigrew J, Petras J, et al: *Differentiation of radicular cyst and granulomas using computerized tomography*, Endod Dent Traumatol 1989;5: 69.
259. Tsai P, Torabinejad M, Rice D, et al: *Accuracy of cone-beam computed tomography and periapical radiography in detecting small periapical lesions*, J Endod 2012;38: 965.
260. Tsao C, Darby I, Ebeling PR, et al: *Oral health risk factors for bisphosphonate-associated jaw osteonecrosis*, J Oral Maxillofac Surg 2013;71: 1360.

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261. Vahtsevanos K, Kyrgidis A, Verrou E, et al: *Longitudinal cohort study of risk factors in cancer patients of bisphosphonate-related osteonecrosis of the jaw*, J Clin Oncol 2009;27: 5356.
262. Venkatesh E., Stafne bone cavity and cone-beam computed tomography. *a report of two cases*, J Korean Assoc Oral Maxillofac Surg 2015;41: 145.
263. Weathers DR, Waldron CA. *Unusual multilocular cysts of the jaws (botryoid odontogenic cysts)*, Oral Surg Oral Med Oral Pathol 1973;36: 235.
264. Weisleder R, Yamauchi S, Caplan DJ, et al: *The validity of pulp testing: a clinical study*, J Am Dent Assoc 2009;140: 1013.
265. Whitaker SB, Waldron CA. *Central giant cell lesions of the jaws*, Oral Surg Oral Med Oral Pathol 1993;75: 199.
266. White SC, Pharoah MJ. Oral radiology: principles and interpretation ed 6 2009; Mosby/Elsevier St Louis.
267. Whitt JC, Dunlap CL, Martin KF. *Oral Hodgkin lymphoma: a wolf in wolf's clothing*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: e45.
268. Williams JW, Simel DL, Roberts L, et al: *Clinical evaluation for sinusitis: making the diagnosis by history and physical evaluation*, Ann Intern Med 1992;117: 705.
269. Workman AD, Granquist EJ, Adappa ND. *Odontogenic sinusitis: developments in diagnosis, microbiology, and treatment*, Curr Opin Otolaryngol Head Neck Surg 2018;26: 27.
270. Yamasaki M, Kumazawa M, Kohsaka T, et al: *Pulpal and periapical tissue reactions after experimental pulpal exposure in rats*, J Endod 1994;20: 13.
271. Yamazaki T, Yamori M, Ishizaki T, et al: *Increased incidence of osteonecrosis of the jaw after tooth extraction*

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in patients treated with bisphosphonates: a cohort study, Int J Oral Maxillofac Surg 2012;41: 1397.

272. Yonetsu K, Yuasa K, Kanda S. *Idiopathic osteosclerosis of the jaws: panoramic radiographic and computed tomographic findings*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 517.
273. Yoshiura K, Hijiya T, Ariji E, et al: *Radiographic patterns of osteomyelitis in the mandible. Plain film/CT correlation*, Oral Surg Oral Med Oral Pathol 1994;78: 116.
274. Zand V, Lotfi M, Vosoughhosseini S. *Proliferative periostitis: a case report*, J Endod 2008;34: 481.

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4: Diagnosis of the nonodontogenic toothache

Alan S. Law, Donald R. Nixdorf, Donna Mattscheck

CHAPTER OUTLINE

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> *An unthinking dentist is a bad dentist. Perfect technique misapplied is at least as unconscionable as sloppy work.*
>
> **Marjorie Jeffcoat**

A nonodontogenic toothache is, of course, an oxymoron. How can one have a toothache that is not odontogenic in etiology? The answer lies in the differentiation of people's perceptions of where they sense their pain, termed the *site of the pain*, from the location of a pathophysiologic process giving rise to the pain that may or may not be in the same region, termed the *source of the pain.* This concept of the attribution of pain to an anatomic region that is different from the location of the etiologic process is generically known as the *referred pain phenomenon* and can occur in multiple areas of the body. Thus, a nonodontogenic toothache has a source of pain that is not the tooth the patient has indicated, clearly demonstrating the diagnostic challenge (Fig. 4.1).

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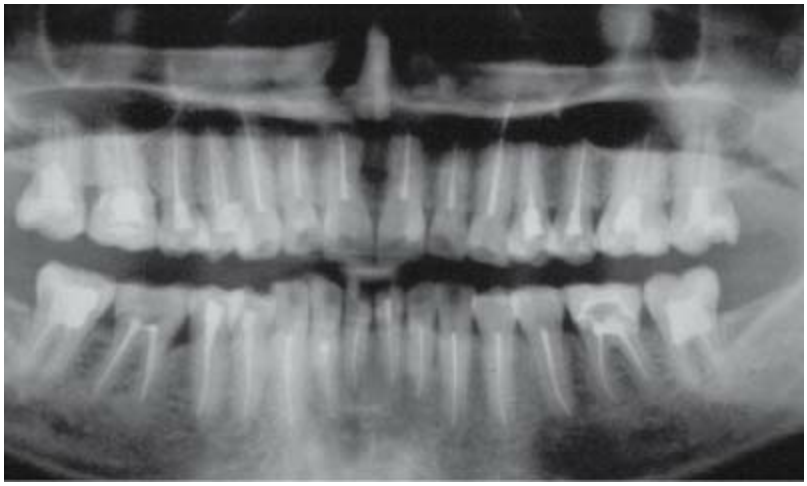

FIG. 4.1 Pantomogram of a patient who has undergone several endodontic procedures without resolution of her chief complaint.

Panoramic radiograph shows radiolucent cavity above the root apices of upper teeth. All teeth have filled root canals, but some of the teeth have radiolucent cavities in the crown. The lower teeth show meshwork of tissues with well-defined radiolucency at and below the root tips.

Source: (Courtesy Dr. Jeffrey Okeson, Lexington, Kentucky.)

Pain is common. It causes human suffering and has significant socioeconomic effects. Pain is a motivator that provokes individuals to seek care. But protracted chronic pain debilitates and can significantly impair the quality and productivity of a person's life. One survey revealed that 66% of respondents reported experiencing pain or discomfort over a 6-month period. Significantly, 40% of respondents reported that this pain affected them to a "high degree."[19] A study published in 2003 estimated the lost productive work time attributed to common pain conditions among active workers to cost $61.2 billion per year.[127] One investigator reported that over a 6-month period, 22% of Americans experienced at least one of five types of facial pain. Of these pains, the most common type (12.2%) was toothache.[81]

Although toothache is the most common pain entity occurring in the facial region,[81] many other types of pain can occur in the same general area. A primary responsibility of a dental practitioner is to diagnose pathologic entities associated with the oral cavity and masticatory apparatus. Many of these pathologic entities have pain as a primary component of their presentation. Because dental practitioners are sought out daily for the alleviation of odontogenic pain, it is imperative for them to have a basic

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working knowledge of other types of facial pain in order to make an accurate diagnosis and properly select care for patients. It is paramount to realize that not all pain entities presenting as toothache are of odontogenic origin. The presenting toothache may be a heterotopic symptom of another disorder. A heterotopic symptom is perceived to originate from a site that is different from the tissue that is actually the source of the pain. This is in contrast to primary pain, in which the perceived site of pain is the actual tissue from which the pain originates. Before discussing pain entities that mimic toothache, it is helpful to understand the neurobiologic mechanisms of orofacial pain.

Review of neuroanatomy

Somatic structures

To understand the pathways by which orofacial pain occurs, one must first gain a basic understanding of the structures involved in its transmission to higher brain centers. Structures of the orofacial region can be divided into two broad categories: somatic and neural structures. Somatic structures are those that make up the different nonneural tissues and organs. The somatic structures can be further anatomically divided into superficial and deep structures. Superficial structures include the skin, mucosa, and gingiva; pain that arises from these superficial structures is usually well localized (e.g., a sharp explorer penetrating the gingiva results in well-localized pain). Deep structures include musculoskeletal and visceral tissues. Pains from these deep structures are typically poorly localized and diffuse in nature.

Neural structures

Neural structures involved in the perception of pain include the afferent (toward the brain) and efferent (away from the brain) regulation of somatic structures. Nerve impulses are transmitted from orofacial structures to the brain via the peripheral nervous system, whereas modulation and interpretation of these impulses into what we feel as pain occurs in the central nervous system. Pain can arise solely from either central or peripheral nervous tissue but heterotopic pain, which is often involved with

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nonodontogenic toothache, likely requires central modulation to occur.

Peripheral nervous system

Pain arises as a result of tissue damage, or the potential for tissue damage, and is transmitted via terminal nerve fibers known as primary afferent nerve fibers. Two major classes of nociceptive (or pain-sensing) primary afferent nerve fibers can detect potentially damaging noxious stimuli: the A-delta and C fibers. Both fiber types have a wide distribution throughout the skin, oral mucosa, and tooth pulp. In addition, separate classes of nerve fibers exist that are involved in detecting nonnoxious stimuli such as vibration and in proprioception. These fibers can be found in the periodontal ligament (PDL), skin, and oral mucosa and include the A-beta fibers.

Primary afferent neurons.

Primarily the trigeminal, or fifth cranial, nerve detects and encodes noxious stimuli for the orofacial region. The majority of cell bodies of the trigeminal sensory fibers are in the trigeminal ganglion located on the floor of the middle cranial fossa. The peripheral axons of the trigeminal ganglion run in three divisions—the ophthalmic (V1), maxillary (V2), and mandibular (V3)—which innervate most of the oral mucosa, the temporomandibular joint (TMJ), the anterior two thirds of the tongue, the dura of the anterior and middle cranial fossae, the tooth pulp, the gingiva, and the periodontal membrane.

In the peripheral nervous system, these neurons or nerves are referred to as primary afferent (i.e., sensory) fibers. The primary afferent fibers can broadly be divided into A-beta fibers, which transmit light touch or proprioceptive information, and A-delta and C fibers, which encode pain. The tooth is densely innervated by afferent nerve fibers, which are believed to transmit pain mainly in response to thermal, mechanical, or chemical stimuli. The majority of dental nerves are C fibers that innervate the central pulp, most of which terminate beneath the odontoblasts.[23]

A-beta fibers.

The rapidly conducting myelinated neurons that respond to light touch are called A-beta fibers. Under normal conditions, activation of the A-beta fibers

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by high-intensity stimulation results in low-frequency output in the central nervous system. Activation of A-beta fibers is normally interpreted as nonpainful mechanical stimulation[133] or, under certain conditions, can be perceived as a “prepain” sensation.[23] A-beta fibers also have been shown to undergo phenotypic changes that allow them to encode painful stimuli under certain inflammatory conditions.[98]

A-delta fibers.

The A-delta fibers are lightly myelinated, have a faster conduction velocity than C fibers, and are believed to transmit a sharp or pricking sensation. A-delta fibers respond primarily to noxious mechanical stimuli rather than to chemical or thermal stimuli. Other A-delta fibers may be polymodal (responding to mechanical, chemical, and thermal stimuli)[13] or respond only to cold/mechanical[78] or hot/mechanical noxious stimuli.[39]

In the tooth pulp, A-delta fibers traverse the odontoblastic layer and terminate in the dentinal tubules.[25] Because of their location and their sensitivity to mechanical stimulation, A-delta fibers are believed to respond to stimuli that result in movement of fluid within the dentinal tubules (e.g., osmotic, mechanical probing, or thermal stimuli applied to the external surface of the tooth).[18] Consistent with the hypothesized mechanism of dentinal pain is the fact that the stimuli that cause dentinal fluid movement result in a sharp pain associated with A-delta fiber activation.[95] When intense noxious stimuli activate the A-delta fibers, the input to the central nervous system consists of high-frequency action potentials.

C fibers.

The C fibers are unmyelinated, have slower conduction velocity, and are associated with a dull, aching, or burning sensation. Most C fibers are polymodal, responding to mechanical, thermal, and chemical stimuli. Because of the difference in conduction velocities, A-delta fibers are believed to transmit early, shooting pain, whereas C fibers would transmit late, dull pain. Noxious stimuli that exceed the receptor threshold of these nociceptive primary afferent terminals result in action potentials that travel centrally, signaling tissue damage. In the pulp tissue, the more centrally located C fibers respond to thermal, mechanical, and chemical stimuli and are believed

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to be sensitized by inflammation.[39] All visceral structures are innervated primarily by afferent fibers conducting nociceptive information such as that carried by A-delta and C fibers.

Central nervous system

The primary afferent fibers are responsible for the transduction and transmission of sensory information to higher brain centers, and they do so by synapsing on neurons located within the trigeminal nucleus, which spans the midbrain and cervical spinal cord. This point marks the beginning of the central nervous system and is the point at which processing of pain information begins (Fig. 4.2).

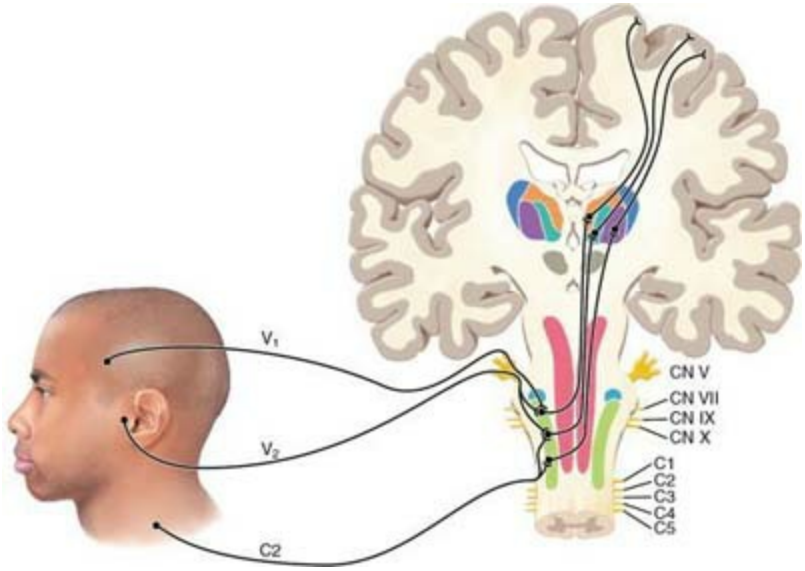

FIG. 4.2 A graphic depiction of the trigeminal nerve entering the brainstem. The primary afferent neuron synapses with a second-order neuron in the trigeminal nucleus. The second-order neuron carries pain information to the thalamus, from which it is sent to the cerebral cortex for interpretation.

A tree-like structure, the trigeminal nerve, on the right shows labels as follows: CN V, CN VII, CN IX, CN X, C1, C2, C3, C4, and C5. To the left, side view of human face is shown. V1, V2, and C2 from the trigeminal nerve lead to regions near the forehead, tragus of ear, and neck at the center, respectively.

Source: (Redrawn from Okeson JP: *Bell's orofacial pains*, ed 5, Chicago, 1995, Quintessence Publishing.)

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Just as there are different types of sensory neurons in the periphery, in the trigeminal nucleus, there are also different types of neurons that receive nociceptive input from the periphery. The ascending neurons located in the trigeminal nucleus are known collectively as *second-order* or *projection neurons* and can be subdivided into three distinct groups of neurons based on the type of information they receive: (1) low-threshold mechanoreceptors, (2) nociceptive-specific, and (3) wide dynamic range neurons.

The primary central site of termination for nociceptive fibers is the subnucleus caudalis, located in the most caudal region of the trigeminal nucleus,[39,57,144] which anatomically and functionally resembles the dorsal horn of the spinal cord and has been referred to as the medullary dorsal horn.[57] Four major components of nociceptive processing are located in the dorsal horn of the subnucleus caudalis: central terminals of afferents, local circuit neurons (interneurons), projection neurons, and descending neurons.[71] Within the subnucleus caudalis, the A-delta and C fibers terminate primarily in the outer laminae (I and IIa) and lamina V. Local circuit neurons are composed of islet cells (which are thought to be inhibitory) and stalked cells (which are believed to be excitatory).[38] Combined, the local circuit neurons may modulate nociceptive transmission from the primary afferents to the projection neurons.

The fourth component of the dorsal horn are the terminal endings of descending neurons. The descending neurons originate in the nucleus raphe magnus (NRM), the medullary reticular nuclei, and the locus ceruleus (LC). Descending brainstem neurons release serotonin (from the NRM) or norepinephrine (from the LC), which may inhibit the activity of projection neurons directly or by activating local opioid interneurons. These neurons are responsible for the endogenous abatement of pain; blockade of their activity increases pain transmission and reduces pain thresholds.

Second-order neurons.

Projection neurons have axons that cross to the contralateral medulla to ascend in the trigeminothalamic tract and project to the ventral posterior medial and intralaminar nuclei of the thalamus, where additional neurons project to the cortex. Projection neurons involved in the transmission of painful stimuli can be divided into two classes: wide dynamic range and

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nociceptive-specific neurons. Wide dynamic range neurons receive input from mechanoreceptors, thermoreceptors, and nociceptors, whereas nociceptive-specific neurons are excited solely by nociceptors. These two types of projection neurons may be responsible for signaling the severity and location of pain, respectively.[79]

Multiple primary afferent neurons may synapse on a single projection (i.e., convergence). This occurs to a much greater degree in deep tissues as opposed to cutaneous tissues. Primary afferent fibers of nontrigeminal origin, such as those derived from vagus, glossopharyngeal, facial, and cervical spinal ganglia have been shown to converge and synapse onto trigeminal projection neurons located as far caudal as spinal level C4.[74] This phenomenon of convergence may result in the clinical finding of pain that radiates beyond an area of tissue injury. Convergence may also explain why pain appears to be associated with a site other than the injured area. Interestingly, when projection neurons receive input from superficial and deep structures, the more superficial inputs usually predominate.[121] Thus, pain originating from deep structures would typically be referred to superficial areas (e.g., pain originating from the jaw muscles would typically be referred to the face rather than deeper structures).

Autonomic nervous system

The stellate ganglia supply the entire sympathetic innervation of the orofacial region, which is located bilaterally at the level of the seventh cervical vertebra. Under normal conditions, sympathetic stimulation has no influence on sensory function. However, afferent sympathetic fibers in an area of trauma may become involved in the response to pain and may also play a role in chronic pain states. Specifically, C fibers in the area of partial nerve injury may become responsive to sympathetic nerve stimulation. The modulation of nociception by the sympathetic nervous system has been shown such that release of pain neurotransmitters may be altered in the presence of sympathetic agonists and by blockade of the sympathetic nervous system, using antagonists.[70] Whether the effects of sympathetic nerve fibers on pain transmission are direct (via homeostatic regulation) or indirect remains unclear. The parasympathetic division of the autonomic nervous system has not been shown to be involved in the development or modulation of pain.

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Review of neurophysiology

Peripheral sensitization

After tissue insult there is an inflammatory reaction that often produces pain. The severity of pain that follows is related to several aspects of the injury, such as the type, extent, and location; the innervation of the tissue; and the phase of the inflammation. In the nociceptive system, tissue injury can manifest itself as increased responsiveness or reduced thresholds to a noxious stimulus, referred to as hyperalgesia. Hyperalgesia can be partially accounted for by sensitization of nociceptors (primary hyperalgesia) and by central nervous system mechanisms (secondary hyperalgesia).

In the absence of tissue damage, activation of C or A-delta fibers produces a transient pain. This pain is believed to serve as a physiologic warning. When there is tissue injury, afferent fibers may be activated by lower intensity stimuli than usual, and the quality of pain may be more persistent and intense. This phenomenon is due, in part, to the sensitization of nociceptors, including an increase in spontaneous activity.

At the site of tissue injury, there are a number of inflammatory mediators that can directly or indirectly sensitize primary afferent nociceptors (see Chapter 13 for more details). These inflammatory mediators may be released from the local tissue cells, circulating and resident immune cells, vasculature and endothelial smooth muscle cells, and peripheral nervous system cells.

Central sensitization

After peripheral tissue injury there is an afferent barrage from C fibers resulting from peripheral tissue inflammation, decreased afferent thresholds, and spontaneous firing of afferent fibers. When a second-order neuron receives a prolonged barrage of nociceptive input, the second-order neuron may also become sensitized. This results in a phenomenon referred to as central sensitization.[17] The result of central sensitization is enhanced processing (i.e., amplification) of neural impulses that are being transmitted to higher brain centers. Two effects of central sensitization are secondary hyperalgesia and referred pain.

Secondary hyperalgesia is an increased response to painful stimulation at

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the site of pain resulting from central nervous system changes. This is in contrast to primary hyperalgesia, which is a lowered pain threshold resulting from sensitization of peripheral neurons. Secondary hyperalgesia might be felt in superficial (e.g., gingiva or skin) or deep structures (e.g., muscles or teeth).

Terminology

In general, as research progresses and uncovers new ways for us to look at pain, the terminology changes. This can introduce some confusion, especially when older terms are used. Therefore, it may be helpful to present contemporary definitions of some of the basic terms and review some of the previously mentioned terms (Box 4.1).

Box 4.1

Types of Pain

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.[86]

Nociceptive pain

Pain arising from activation of nociceptors.[86]

Neuropathic pain

Pain arising as a direct consequence of a lesion or disease affecting the somatosensory system.[86,135]

Peripheral sensitization

Increased responsiveness and reduced thresholds of nociceptors to stimulation of their receptive fields.[86]

Central sensitization

Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input.[86]

Heterotopic pain

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Any pain that is felt in an area other than its true source is heterotopic pain. There are three types of heterotopic pain: referred, central, and projected.[105,106] Referred pain is felt in an area innervated by a nerve different from the one that mediates the primary pain. Referred pain cannot be provoked by stimulation of the area where the pain is felt; rather, it is brought on by manipulation of the primary source of pain (Fig. 4.3). In addition, referred pain cannot be arrested unless the primary source of pain is anesthetized. The referral of pain tends to occur in a laminated fashion (Fig. 4.4). This is because peripheral nociceptors enter the spinal trigeminal tract in a laminated fashion. As a result, there are general referral patterns in the face. In addition, the referral of pain is usually in a cephalad or upward direction. This is evidenced clinically in that pain from mandibular molars typically is referred to maxillary molars, as opposed to premolars or incisors.

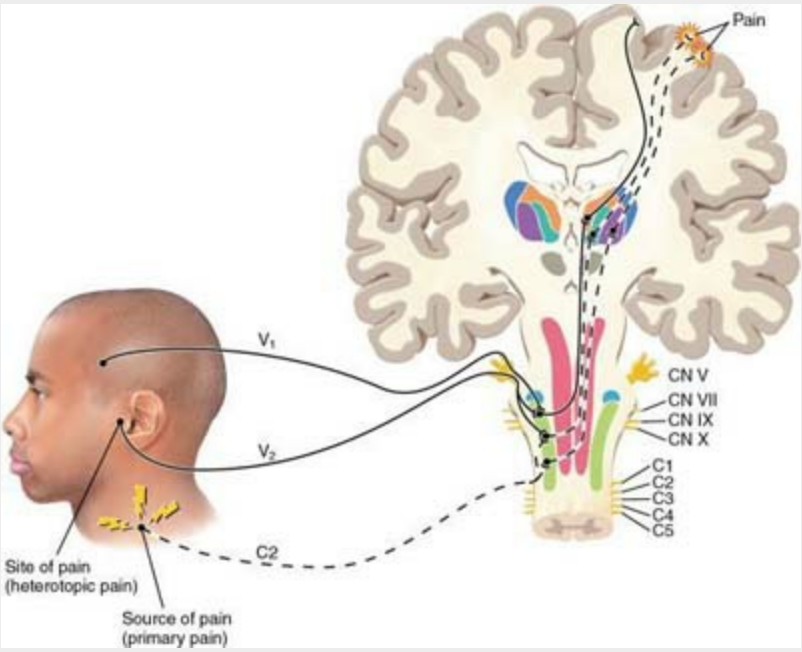

FIG. 4.3 Illustration of pain that is referred from an area innervated by one nerve *(C2)* to an area innervated by a different nerve *(V2)*. Note that this phenomenon occurs secondary to the convergence of different neurons onto the same second-order neuron in the trigeminal nucleus. The sensory cortex perceives two locations of pain. One area is the trapezius region that represents the source of pain. The second area of perceived pain is felt in the temporomandibular joint area, which is only a site of pain, not a source of pain. This pain is heterotopic (referred).

A tree-like structure, the trigeminal nerve, on the right shows labels as

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follows: CN V, CN VII, CN IX, CN X, C1, C2, C3, C4, and C5. To the left, side view of human face is shown. V1, V2, and C2 from the top right marked pain in the trigeminal nerve lead to regions near the forehead, tragus of ear marked site of pain (heterotopic pain), and neck at the center source of pain (primary pain).

Source: (Redrawn from Okeson JP: *Bell's orofacial pains*, ed 5, Chicago, 1995, Quintessence Publishing.)

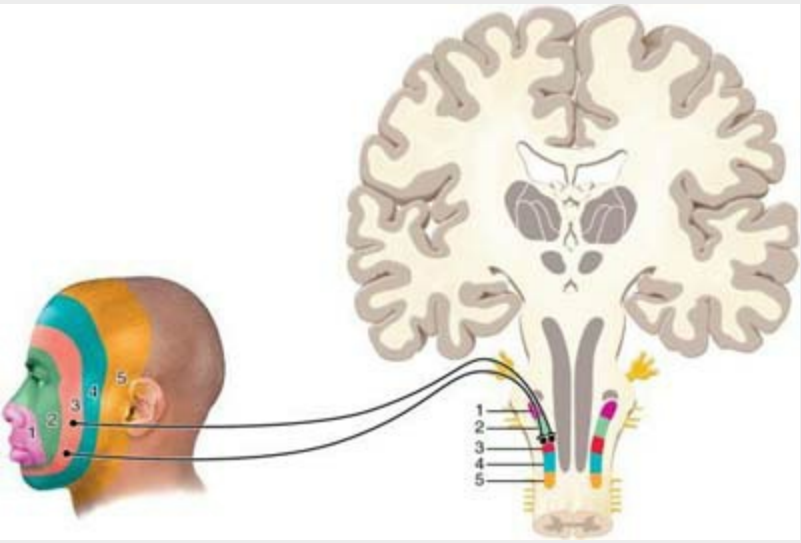

FIG. 4.4 Illustration of the laminated pattern of innervation from orofacial structures into the trigeminal nucleus. These laminated patterns commonly reflect the patterns of referred pains felt in the orofacial structures.

A tree-like structure, the trigeminal nerve, on the right shows a bar in the trunk region consisting of pink, green, red, sky blue, and yellow colors, marked 1 through 5. To the left, the side view of human face is shown, in which the regions from mouth to ear are marked from 1 thorough 5 showing same colored bands as that of the trigeminal nerve. Two branches from red bar lead to cheek.

Source: (Redrawn from Okeson JP: *Bell's orofacial pains*, ed 5, Chicago, 1995, Quintessence Publishing.)

Clinical entities that can present as toothache

Sources of odontogenic toothache

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Before considering heterotopic pains that may present as toothache, it is important to fully understand odontogenic pain as a primary source for toothache. Only two structures serve as sources for primary odontogenic pain: the pulp–dentin complex and the periradicular tissues. The innervation of the pulp is similar to that of other deep visceral tissues; thus, in various states of pathosis, it will have pain characteristics similar to deep visceral tissues. The primary nociceptors of the pulp that respond to inflammation are the slow-conducting, high-threshold C fibers. Because their threshold is high and they rarely terminate in dentinal tubules, C fibers do not respond to normal or nonpathologic dentinal stimulation. C fibers typically conduct pain that is associated with tissue damage. In addition, C fibers respond in a threshold manner that can be termed "all or nothing." For example, a slightly cold stimulus that is below the C fiber threshold will fail to produce any sensation. Only when a stimulus is intense enough to reach the threshold will the C fiber fire, resulting in the sensation of pain.

Pulpal pain is mediated by C fibers and is dull, aching, or throbbing in nature. This is in contrast to the quick, short, sharp sensation produced by A-delta fibers that mediate dentinal pain. Therefore, when pulp testing, it is meaningful to note not only whether the patient perceived the stimulus but also the nature of the stimulus perceived. A simple notation would be to use an "s" (short) to indicate a response more typical of an A-delta fiber (dentinal pain) or a "p" (prolonged) to indicate the response was more indicative of a C fiber response (pulpal pain).

Tissue inflammation can result in sensitization of nerve fibers. When peripheral nociceptors (e.g., pulpal C fibers) are sensitized, the threshold of firing in response to a given stimulus (e.g., temperature and pressure) is lowered. In states of sensitization these nociceptors can be provoked with a less intense stimulus. The threshold for excitation is still "all or nothing" but the required level of stimulation has decreased. These fibers can become so sensitized that they may fire at as low a temperature threshold as body temperature,[95] normally not sufficient to stimulate a C fiber. In fact, they can become so sensitized that they will fire in response to the normal pulse pressure of cardiac contraction, eliciting a complaint of "I can feel my heartbeat in my tooth" or "my tooth is throbbing." Sensitized C fibers can even fire without provocation, resulting in spontaneous pain.

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Similar to deep visceral tissues, the pulpal nociceptors demonstrate a high degree of convergence in the central nervous system. In a study of cat brain, 74% of the neurons tested in the subnucleus caudalis showed convergence from multiple tooth pulps.[22] In addition, the dental pulp has little to no proprioceptive neurons. The high degree of convergence from pulp tissue and the lack of proprioceptive information provided are the key factors in why purely pulpal pain can be so difficult for patients to localize. In addition to reducing localization of pain, convergence increases the referral of pain to tissues not actually affected by the inflammation. The fact that neurons from the pulps of mandibular teeth converge with those of maxillary teeth can result in pain from a mandibular pulpitis being referred to the maxillary arch. Because the patient may poorly localize pulpal pain, it is important for the clinician to localize the source of the pain. This is often accomplished through the use of tests that are employed in an attempt either to reproduce the eliciting stimulus of a patient's pain or to eliminate the pain. For example, pulpal pain should be aggravated with hot or cold stimulation and should be eliminated or significantly reduced with local anesthetic.

Unlike pulpal pain, pain of periradicular origin is easier to localize. Mechanoreceptors are numerous in the PDL and are most densely concentrated in the apical one third.[87] Once inflammation from pulpal disease extends into the PDL, patients are able to locate the source of the pain much more readily. As a musculoskeletal structure, the PDL responds to noxious stimulation in a graded fashion—that is, the degree of discomfort a patient feels in relation to periradicular pain depends on the degree of peripheral sensitization and the amount of provocation to this structure. A sensitized PDL will be uncomfortable to a patient if percussed lightly but more uncomfortable if percussed heavily. This is known as a graded response. It is, therefore, appropriate to record periradicular testing such as percussion and palpation in terms of degrees of tenderness (versus "all or nothing"). As with pulpal pain, pain of periradicular origin should also have an identifiable etiology. Periradicular pain tends to be dull, aching, or throbbing and should resolve completely with local anesthesia. If pain of suspected periradicular origin is nonresponsive to local anesthetic, it is a strong indication that the pain may be nonodontogenic in origin.

The tooth is unique in the human body in that it has a visceral-like

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component, the pulp, and a musculoskeletal component, the PDL. Therefore, odontogenic pain can have a wide variety of presentations. Tooth pain can be diffuse or well localized, mild or intense, or spontaneous or provoked with various stimuli applied at various intensities. The quality can vary between sharp and dull and aching or throbbing. This potential for extreme variability makes it possible for toothaches to mimic or be mimicked by many other types of pain that occur in the head and neck. In addition, because both the pulp tissue and PDL can be categorized as deep somatic tissue, continued nociceptive input from odontogenic pain has a great propensity to produce central excitatory effects such as secondary hyperalgesia, referred pain, secondary co-contraction of muscles, myofascial trigger points, and autonomic changes. These effects add to the complexity of diagnosing odontogenic pain and differentiating tooth pain from other sources in the region.

Sources of nonodontogenic toothache

This chapter provides information that will help the dental clinician to identify toothaches with a nonodontogenic etiology. The clinician must have a thorough knowledge of all possible causes of orofacial pain, which includes both odontogenic and nonodontogenic conditions. This knowledge prevents misdiagnosis and allows for proper treatment selection and referral if necessary. For information about treatment of these disorders, other references should be used.

Consensus on the exact taxonomy with diagnostic criteria and their interrelationships among various orofacial pain disorders has not been established. Various health care professions involved in the diagnosis and treatment of such pains have used different terms in the literature. This, of course, can and has led to confusion, especially within what we refer to as neuropathic pain. The terms used in the literature are diverse, and they overlap in meaning to an unknown degree; for example, *phantom tooth pain* and *atypical odontalgia* are used interchangeably. At other times, the literature uses the same terms to describe seemingly different disorders; for example, trigeminal neuralgia has the connotation of an idiopathic pain disorder characterized either as intense, intermittent lightning bolt–type pain within one or more distributions of the trigeminal nerve, or as continuous

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pain that is often mild to moderate in intensity that arises in association with injury to a specific branch of the trigeminal nerve. Efforts have resulted in a working diagnostic framework for neuropathic pains.[135] Our classification scheme uses this framework to enhance the clarity of communication and follows the American Academy of Orofacial Pain's guidelines for assessment, diagnosis, and management for orofacial pain,[30] even though the application of these criteria to pains that present in the orofacial region is known to be associated with misclassification.[35]

Overall, one can classify the nonodontogenic reasons for toothache into five broad groups of pain disorders:

1. Musculoskeletal and other nonprogressive pains arising from somatic structures
2. Neurovascular pain, otherwise known as headache disorders
3. Neuropathic pains
4. Pain associated with a pathologic process
5. Pain of purely psychologic origin, otherwise known as psychogenic toothache

Musculoskeletal and somatic pain

Myofascial pain.

Although any deep somatic tissue type in the head and neck has the propensity to induce central excitatory effects and therefore cause referral of pains to teeth, pains of muscular origin appear to be the most common.[46] Myofascial pain (MFP) emanates from small foci of hyperexcitable muscle tissue. Clinically these areas feel like taut bands or knots and are termed *trigger points.*[134] Typically the pain is described as a diffuse, constant, dull, aching sensation; this may lead the clinician to a misdiagnosis of pulpal pain. Another potentially misleading characteristic of masticatory muscle pain is that patients may report pain when chewing. This feature is similar to pain that is periradicular, not pulpal, in origin. On further investigation, it should become clear that the pain is triggered by contraction of masticatory muscles rather than loading of PDLs. Palpation of the muscles of mastication should reproduce the pain, whereas percussion of the teeth should not. The intensity

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of the pain will increase and can be perceived in a distant site. MFP that is perceived to emanate from a tooth is a referred type of heterotopic pain—that is, the pain is felt in an area other than the nerve branch that innervates the trigger point. Typically muscles that refer pain to teeth are the masseter, temporalis, and lateral pterygoid; muscles of the neck and nonmuscular deep structures of the face can also be a source for this type of pain.[134,142]

Although the definitive pathogenesis of MFP is unknown, authors have theorized that muscles may become disturbed through injury or sustained contraction such as clenching.[45,105] Clinically, this muscular contraction might occur as a parafunctional habit or as a protective response by localized muscle to an ongoing deep noxious input such as dental pain. Considering this theory and what is witnessed clinically, trigger points appear to be induced or aggravated by toothache. It also appears that trigger points can persist after the toothache has been resolved. This can be confusing for the clinician and frustrating for the patient. It is important to realize the relationship of these two entities. MFP can mimic toothache, and toothaches may induce the development of MFP.

Toothaches of myofascial origin may arise with or without evidence of pulpal or periapical pathosis. Definitive diagnosis is based on lack of symptoms after pulp testing and percussion or palpation sensitivity, or failure to resolve symptoms with local anesthetic blockade. In contrast, jaw function and palpation of the masticatory muscle(s) elicit toothaches of myofascial origin. Typically, local anesthetic infiltration into the trigger point(s) will resolve symptoms.

Common therapeutic modalities used to treat MFP include deep massage, relaxation techniques, “spray and stretch,” muscle relaxants, and trigger point injections. Deep massage and relaxation techniques have the advantage of being noninvasive and easily administered. Spray and stretch involves an application of a vapor coolant spray to the skin overlying the trigger point, followed by a gentle stretching of the muscle. Trigger point injections are used for both the diagnosis and treatment of MFP. Specifically, if the pain complaint is diminished on injection of the trigger point(s), then the source of the pain has been confirmed. The therapeutic efficacy of a trigger point injection varies. Some patients might experience lasting relief with one injection or several, whereas others may not. See the Additional Tests section

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for further information about trigger point injections.

Pain of sinus or nasal mucosal origin.

Sinus/nasal mucosal pain is another source of pain that can mimic toothache.[1,2,28,138] Sinus pain can exhibit symptoms of fullness or pressure below the eyes but is generally not particularly painful unless the nasal mucosa is also affected.[37] Pain from the nasal mucosa tends to be dull and aching and can also have a burning quality typical of visceral mucosal pain. In general, these pains are of viral, bacterial, or allergic etiology. Other symptoms consistent with these types of disease (e.g., congestion or nasal drainage) should be noted in the patient history.

Typical of deep visceral-like tissues, sinus/nasal mucosal pain can induce central excitatory effects such as secondary hyperalgesia, referral of pain, and autonomic changes. It is this tendency that gives sinus/nasal pain the ability to masquerade as toothache.[129a] Secondary hyperalgesia, seen clinically as a concentric spread of pain beyond the area of tissue injury, results in tenderness of the mucosa in the area of the maxillary sinuses as well as tenderness to percussion of several maxillary teeth. Teeth tender to percussion and palpation suggest periradicular inflammation. Autonomic sequelae might present as edema or erythema in the area, which could suggest a dental abscess. However, when an etiology for pulpal and therefore periradicular pathosis is absent, sinus/nasal mucosal disease should be suspected. The three cardinal symptoms of acute rhinosinusitis are (1) purulent nasal discharge, (2) nasal obstruction, and (3) facial pain-pressure-fullness.[118] Other symptoms of sinus disease include sensitivity to palpation of structures overlying sinuses (i.e., paranasal tenderness) and a throbbing or increased pain sensation when the head is placed lower than the heart. Dental local anesthetic blockade will not abate sinus/nasal mucosal pain, although topical nasal anesthetic will.

Patients with suspected sinus/nasal mucosal disease should be referred to an otolaryngologist for further diagnosis and treatment. Physical examination as well as adjunctive tests may be necessary for a definitive diagnosis. Tests may include nasal cytologic and ultrasound studies and the use of nasal endoscopes, in addition to imaging tests such as radiology and computed tomographic imaging.[36,125] Treatment of sinus/nasal mucosal pain is

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dependent on the etiology (e.g., bacterial, viral, allergic, or obstructive).

Salivary gland pain.

Pain referred from one or more of the salivary glands may be perceived as tooth pain; the authors have not encountered this response in clinical practice, but it has been reported to present as a nonodontogenic toothache.[80,115] Because the primary somatosensory innervation of the major salivary glands comes from the mandibular branch, it is conceivable that such a presentation will occur most often in mandibular teeth.

Neurovascular pain

Neurovascular pains, otherwise and interchangeably referred to as headache disorders, have qualities similar to pulpal pain. These types of pain can be intense, often pulsatile, and are known to occur only in the head. The International Headache Society (Oxford, UK) has developed a classification system that is widely accepted even though validation studies of these criteria have yet to be published. The interested reader should consult the classification system for more details on this topic.[56] Primary neurovascular pain disorders are thought to be a referred pain phenomenon, meaning that intracranial branches of the trigeminal nerve become sensitized via incompletely understood mechanisms and the associated pain and symptoms are perceived in the somatic structures of the head. Most commonly people report pain presenting in the forehead, back of the head, and temples but also in the sinuses, jaws, and teeth.

The current understanding of the pathophysiology of headaches implies that dental disease and treatments are not likely to be a cause of a person developing a headache disorder, but rather, because the same neuroanatomic circuitry is involved, these aspects of dentistry can be thought of as an inciting event, similar to the analogy that exercise producing increased demands on the cardiovascular system can be an inciting event for an acute myocardial infarction. For this reason, dental clinicians should be aware of the diagnostic status of their patients, because patients with headache disorders are likely to experience more peritreatment pain complications that are related to the innate hyperexcitability of the trigeminal nervous system in these people.

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Of most interest to the dental clinician are the primary headache disorders, which make up the bulk of the headache disorders that occur within the population and have been reported to present as nonodontogenic toothache. To simplify thinking, these primary headache disorders can be grouped into three major subdivisions: (1) migraine, (2) tension-type headache, and (3) cluster headache and other trigeminal autonomic cephalalgias (TACs).

Migraine is a common headache experienced by about 18% of females and 6% of males.[82,128] It is associated with significant amounts of disability, which is the motivating factor that brings the patient to seek care and the reason why this type of headache is the one most often seen in medical clinics.[131] Migraine has been reported to present as toothache[4,26,34,52,96,103] and is likely the most common neurovascular disorder to do so. In addition, people with migraine headaches are thought of as having increased regional pain sensitivity that has diagnostic and treatment implications for the clinician.[102]

Migraine headaches typically last between 4 and 72 hours. They tend to be unilateral in presentation and pulsatile in quality, with a moderate to severe intensity to the pain. Patients may also experience nausea or vomiting, as well as photophobia or phonophobia, which are different from toothache. The headache is usually aggravated with routine physical activity, such as walking up stairs. Caffeine/ergotamine compounds have been used widely in the past as abortive agents for migraine headaches, but in contemporary times they have been replaced with triptans, such as sumatriptan and rizatriptan.[93] Of note, migraine headaches may partially or fully abate with the use of nonsteroidal anti-inflammatory medications in a similar fashion as toothaches.

Tension-type headache is the most frequent headache disorder experienced, with a wide range of reported prevalence (41% to 96%).[117,123] The concept of tension-type headache pain presenting as toothache has not been reported in the literature to our knowledge, likely because the construct of what a tension-type headache is has not been clearly defined. Some research supports the notion that a tension-type headache has a significant musculoskeletal component to the pain,[129] whereas other research suggests otherwise. Tension-type headaches are likely a heterogeneous group of similarly presenting head pains that have overlapping pathophysiologic

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mechanisms, which has led some researchers to consider aspects of tension-type headache to be the same as musculoskeletal orofacial pain, otherwise known as temporomandibular disorders (TMDs).[55] This has further been supported by data from a TMD validation study to derive criteria for such headaches that are of TMD origin.[6,122]

Cluster headaches and other TACs are rare neurovascular disorders that are strictly unilateral pains defined by the concurrent presentation of at least one ipsilateral autonomic symptom—such as nasal congestion, rhinorrhea, lacrimation, eyelid edema, periorbital swelling, facial erythema, ptosis, or miosis—that occurs with the pain. The major distinguishing features between these headache disorders are the duration and frequency of the pain episodes, as well as the gender most often afflicted. Cluster headache is the most common of the group, occurring in men three to four times more often than in women, with pain episodes lasting between 15 minutes and 2 hours that occur at a frequency of eight episodes per day to one every other day. These headaches come in clusters, with active periods of 2 weeks to 3 months,[56] thus the name. Elimination of pain after 10 minutes with inhalation of 100% oxygen is diagnostic for cluster headache,[49] whereas sublingual ergotamine and sumatriptan are also effective acute treatments for cluster headache.[42] Paroxysmal hemicrania, which has a 3:1 female predilection, presents with characteristics similar to those of cluster headache but with a frequency of more than five per day and a duration lasting 2 to 30 minutes.[56] This headache disorder has a 100% response to indomethacin but is refractory to other treatments,[65] thus underscoring the need for obtaining an accurate diagnosis from an experienced clinician.

From a nonodontogenic perspective, cluster headache[4,14,21,51] and almost all the other TACs have been reported in the literature to present as nonodontogenic toothache.[4,11,12,31,74a,92,110,120] The concurrent autonomic features, such as discoloration or swelling in the anterior maxilla, might compound the diagnostic problem by suggesting tooth abscess. It is important to note that neurovascular headaches tend to be episodic with complete remission between episodes, whereas toothache pain usually has at least some background pain that stays between any exacerbations. Provocation of the tooth should not result in a clear increase in pain but cause a slight alteration because this tissue has become hypersensitized. Local anesthetic is

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unpredictable in these cases and can mislead the clinician. Management by the typical clinician is to determine that the pain is not of odontogenic origin and then to refer the patient to an appropriate care provider. Other neurovascular disorders not classified as primary headaches have been reported to present as nonodontogenic toothache, such as cough headache.[91] One would not expect a dental clinician who does not have a specific focus on orofacial pain to arrive at such a specific diagnosis but rather to be aware of and sensitive to the fact that more obscure headache disorders exist and should be considered in the differential diagnosis of a nonodontogenic toothache that is not easily classified.

Neuropathic pain

All previously described pain entities can be classified as somatic pain. That is, they are a result of noxious stimulation of somatic structures. These impulses are transmitted by normal neural structures, and their clinical characteristics are related to stimulation of normal neural structures. Neuropathic pain actually arises from abnormalities in the neural structures themselves, specifically the somatosensory system. The clinical examination generally reveals no somatic tissue damage, and the response to stimulation of the tissue is disproportionate to the stimulus. For this reason, neuropathic pains can be misdiagnosed as psychogenic pain simply because a local cause cannot be readily identified. There are many ways to categorize neuropathic pain in the orofacial region. For the purposes of this chapter and ease of discussion, neuropathic pain is divided into four subcategories: neuralgia, neuroma, neuritis, and neuropathy. It should be acknowledged that these subcategories are arbitrary and are not mutually exclusive.

Neuralgia.

As alluded to previously, not all uses of the term *neuralgia* refer to what is often thought of as the classic trigeminal neuralgia or tic douloureux. Sometimes the term *neuralgia* is used to describe pain felt along a specific peripheral nerve distribution, such as with postherpetic neuralgia and occipital neuralgia, as opposed to a focus of pain disorders that have similar characteristics and are thought to have common underlying pathophysiologic mechanisms. When used in the generic sense to describe pains that present

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intraorally, the term can lead to a great deal of confusion.

Although deviations are not uncommon, trigeminal neuralgia is characteristically an intense, sharp shooting pain that is most often unilateral. Ipsilateral to the perceived location of the symptoms is an area that, on stimulation such as light touch, elicits sharp shooting pain. The area that elicits the pain is referred to as a trigger zone, and it can be in the distribution of the resultant pain or in a different distribution—but is always ipsilateral. Although most patients present with a characteristic trigger zone, not all patients will present with this finding. An important characteristic of trigger zones is that the response to the stimulus is not proportional to the intensity of the stimulus—that is, slight pressure on a trigger zone results in severe pain. In addition, once triggered, pain typically subsides within a few minutes until triggered again. This is in contrast to odontogenic pain, which may come and go but does not do so in such a predictable and repeatable manner. Finally, the trigger for odontogenic pain is an area that has no sensory abnormalities (e.g., dysesthesia or paresthesia).

Trigger zones for trigeminal neuralgia tend to be related to areas of dense somatosensory innervation, such as the lips and teeth. For this reason, triggers that elicit this type of pain may include chewing and may lead both the patient and clinician to think of a diagnosis of odontogenic pain. In addition, because the trigger involves peripheral input, anesthetizing the trigger zone may diminish symptoms. This can be very misleading to the clinician if the assumption is that local anesthetic blocks only odontogenic pain.

Because symptoms can be quite severe, patients may consent to or even insist on treatment even though the clinical findings do not definitively support an odontogenic etiology. The possibly misleading symptoms, along with the willingness of the patient to consent to what may seem to be desperate measures, emphasize the importance of a thorough history and clinical evaluation. The absence of a dental etiology for the symptoms (e.g., large restorations, dental trauma, or recent dental treatment) in the presence of the characteristic sharp shooting pain should alert the clinician to consider trigeminal neuralgia in the differential diagnosis. In general, these individuals should be referred to a neurologist or orofacial pain/oral medicine clinician for a complete diagnostic workup and treatment, because case series have

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suggested 15% to 30% of patients have secondary reasons for their pain,[58,143] such as brain tumors and multiple sclerosis.

Trigeminal neuralgia typically presents in individuals older than 50 years of age. The etiology is thought to be irritation/compression of the root of the trigeminal nerve, prior to the Gasserian ganglion, possibly as a result of carotid artery pressure. Individuals with multiple sclerosis develop trigeminal neuralgia more frequently than the general population. For this reason, a person younger than 40 years of age who develops trigeminal neuralgia should also be screened for multiple sclerosis[147] or other intracranial pathosis.[58]

The two general treatment options for trigeminal neuralgia are pharmacologic and surgical procedures. Because of the possible complications associated with surgery, this form of treatment is usually considered only after attempting pharmacologic therapies. Several medications, including carbamazepine, baclofen, gabapentin, and more recently pregabalin and oxcarbazepine, have been used to treat trigeminal neuralgia. Drugs aimed at relieving nociception, such as nonsteroidal anti-inflammatory agents, have no significant benefit in these patients, nor do opioid-based analgesics. Clinical trials support carbamazepine as a first-line drug for treating trigeminal neuralgia.[8] In patients who experience pain relief from carbamazepine, the effect is usually rapid; most will report a decrease in severity of symptoms within the first couple of days.

What is thought to be a variation of trigeminal neuralgia, and may also mimic toothache, is pretrigeminal neuralgia. Pretrigeminal neuralgia, as the name suggests, has been described as symptoms that are different from those of classic trigeminal neuralgia but that respond to pharmacotherapy like classic trigeminal neuralgia and, over time (usually weeks to 3 years), take on the classic characteristics of trigeminal neuralgia. The definitive features include the presence of a dull aching or burning pain that is less paroxysmal in nature but still triggered by a light touch within the orofacial region, with variable periods of remission.[48] The subsequent onset of true neuralgic pain may be sudden or may appear several years later,[105] which emphasizes the need for long-term follow-up of these patients to obtain an accurate final diagnosis.

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Neuroma.

The term *neuroma* has been around for many years and is often overused in an attempt to describe other types of neuropathic pain. A traumatic neuroma, also known as an amputation neuroma, is a proliferative mass of disorganized neural tissue at the site of a traumatically or surgically transected nerve. A part of the diagnosis, therefore, is confirmation of a significant event that would account for the damage to the nerve. Symptoms will not develop until the neural tissue on the proximal stump has had time to proliferate, typically about 10 days after the event. Tapping over the area of a neuroma elicits volleys of sharp electrical pain (i.e., Tinel sign) similar to trigeminal neuralgia. In contrast to trigeminal neuralgia, there should be a zone of anesthesia peripheral to the area of the neuroma[111] that can be identified by checking for loss of pinprick sensibility, such as with the use of an explorer.

Treatment of a neuroma involves pharmacologic management, often via local measures, and may involve surgical coaptation of the nerve with prognosis being variable and dependent on adequate distal nerve tissue and the time interval between injury and reconstruction.[148] Therefore, early recognition and referral are of key importance to prevent significant distal nerve degeneration.[76] Although neuromas most commonly develop in the area of the mental foramen, lower lip, and tongue, there is some evidence that they can also form in extraction sites and after pulpal extirpation. Neuromas were found to form in extraction sites between 4 and 6 months after removal of the tooth in an experimental animal model.[69] Although not all neuromas that form are painful, this could be a potential explanation for ongoing pain in extraction sites after healing has appeared to occur.[111] It is interesting to ponder the possibility of neuroma formation in deafferentation injuries such as pulpectomy and the implications this might have on continued PDL sensitivity after adequate root canal treatment. For treatment of neuromas that are not amenable to surgical correction, see the Neuropathy section of this chapter.

Neuritis.

Neuritis is a condition caused by inflammation of a nerve or nerves secondary to injury or infection of viral or bacterial etiology. In general, pain from a virally induced neuritis, such as recurrent herpes simplex or herpes

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zoster, is associated with skin or mucosal lesions (Fig. 4.5). This presentation does not result in much of a diagnostic challenge, but pain can precede the vesicular outbreak by many days or even weeks.[47] Because neuritic disorders are caused by reactivation of a virus that has been dormant in the trigeminal ganglion, they are considered projected pain with distribution within the dermatomes innervated by the affected peripheral nerves. The nerves affected by the virus may solely supply deeper tissues and therefore may not produce any cutaneous lesions. In the absence of skin or mucosal lesions, a viral neuritis can be difficult to diagnose[47,60,67] and should be considered in the differential diagnosis of a patient with a history of primary herpes zoster infection. Bacterial infection of the sinuses or dental abscess can also cause neural inflammation that may result in pain. This pain occurs simultaneously with pain of the infected tissues and usually dissipates once the etiology is addressed. In susceptible individuals, virally or bacterially induced neuritis may produce a postinfection neuropathy of the infected nerve. The pain is fairly constant and can be dull, aching, and burning. Also, the pain may be accompanied by allodynia, a painful response to normally nonnoxious stimulation such as light brushing of the skin. Oral acyclovir has become the most common treatment for acute herpetic outbreaks and has been shown to be efficacious in decreasing the duration and severity of pain after herpes zoster infection. Efficacy is based only on administration in the prevesicular, not the vesicular, stage. The addition of prednisolone to acyclovir produces only slight benefits over acyclovir alone. Neither acyclovir alone nor its combination with prednisolone appears to reduce the frequency of postherpetic neuralgia.[140]

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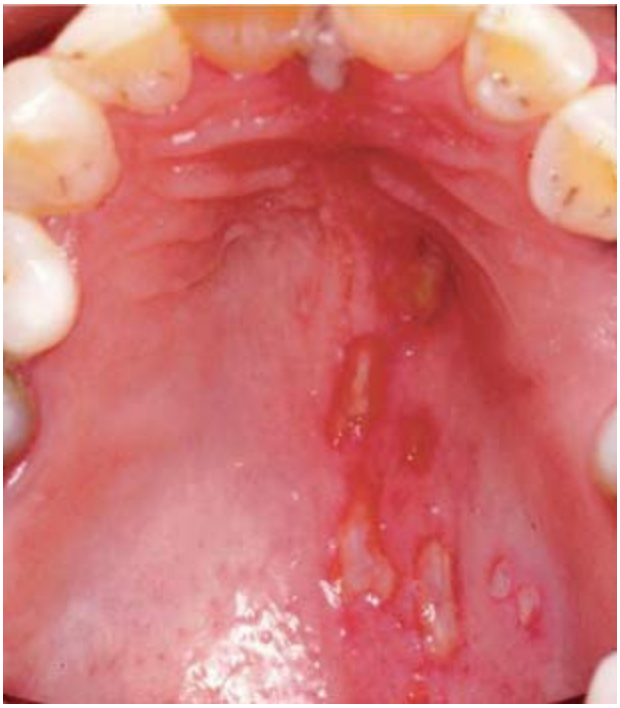

FIG. 4.5 Herpes zoster involving the maxillary division of the left trigeminal nerve of the palate of a 45-year-old male. He complained of a deep, diffuse dull ache of his maxillary left quadrant for 1 week before this vesicular outbreak.

Close-up view of upper teeth shows several mucosal lesions on the palate next to the teeth. The inner side of teeth have yellow stains.

Localized traumatic injury can also induce neuritis. This injury can be chemical, thermal, or mechanical in nature. A classic endodontic example of a chemical injury to a nerve is the overextension of a highly neurotoxic paraformaldehyde-containing paste (e.g., Sargenti paste) onto the inferior alveolar nerve canal. Chemical trauma can be due to certain toxic components of the endodontic filling materials (such as eugenol), irrigating solutions (such as sodium hypochlorite), or intracanal medicaments (such as formocresol) (Fig. 4.6).[94] Mechanical compression, in addition to thermal trauma, may be a factor when thermoplasticized material is overextended, using an injectable[50] or carrier-based technique. Mechanical nerve trauma is more commonly associated with oral surgical procedures, such as orthognathic surgery, and third molar extraction.

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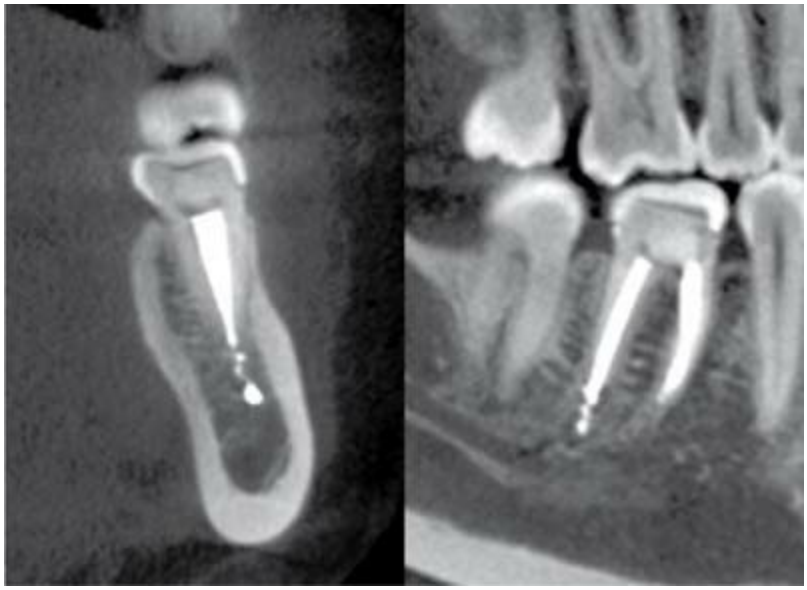

FIG. 4.6 Extrusion of the filling material from the distal canal of tooth #30 of a 36-year-old female. Her complaint was of extreme pain after completion of the root canal treatment followed by sharp, burning continuous pain that could be made worse by a light touch of the tooth.

Radiograph on the left shows a single tooth with filled root canal that is broken near the tip. The root is anchored in porous tissue. Enamel of the tooth is radiopaque. Radiograph on the right shows six teeth. The central tooth in the lower jaw has filled root canals. One of the root canals is comparatively long. Roots of all teeth are surrounded by porous tissue and well-defined radiolucency.

Neuritic complications have also been documented after mandibular implant surgery at a rate of 5% to 15%, with permanent neuropathies, which are discussed later, resulting in approximately 8% of these cases.[66] It is unfortunate that traumatic neuritis is often misdiagnosed as a posttreatment chronic infection and that the area is reentered and debrided. Additional surgical insult further traumatizes the nerve, prolonging the already present nociceptive barrage, which puts the patient at an increased risk of developing central hyperalgesia. Undiagnosed and mistreated cases of acute neuritis not only lead to unnecessary dental procedures but may also aggravate the neuritis and, therefore, the neuritic pain has a greater chance of becoming chronic, something that is often referred to as neuropathic pain.

Neuritic pain typically is a persistent, nonpulsatile burning often associated with sensory aberrations such as paresthesia, dysesthesia, or anesthesia. The pain can vary in intensity, but when stimulated, the pain provoked is

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disproportionate to the stimulus.

Treatment of acute neuritis is based on its etiology. In instances of chemical trauma (e.g., Sargenti paste) where an obvious irritant is present, surgical debridement of the nerve to remove any substance that can continue to irritate the nerve is an important aspect of treatment. With neuritis secondary to mechanical compression (e.g., implant placement) of a nerve, nerve decompression by removal of the implant fixture is indicated. Such localized, acute, traumatically induced neuritis is inflammatory in nature and, therefore, can also benefit from supportive pharmacotherapies such as steroids. For management of neuritis that is not responsive to the previously cited treatments, medications used to treat neuropathic pain may be used (see Neuropathy). For neuritis occurring secondary to an infection, such as one of odontogenic or viral etiology, treatment is directed at eliminating the offending pathogen and minimizing injury to the afferent nerves.

Neuropathy.

In this chapter we use the word *neuropathy* as the preferred term for localized, sustained nonepisodic pain secondary to an injury or change in a neural structure. Historically, other terms have been used including *atypical facial pain.* This term suggests pain that is felt in a branch of the trigeminal nerve and that does not fit any other pain category. Pain of an unknown source that is perceived in a tooth may be labeled *atypical odontalgia.* Pain that persists after the tooth has been extracted is referred to as *phantom tooth pain.* The major limitation in the use of all these terms is that they merely suggest an area where a pain of unknown etiology exists and completely lack any information regarding the pathophysiology. Although each of these terms has been extensively described in the literature,[88,89] probably none actually represents one discrete condition but rather a collection of various conditions. Based on these thoughts, a consensus process resulted in proposing new terminology, *persistent dentoalveolar pain disorder (PDAP)*, and diagnostic criteria (Fig. 4.7).[99]

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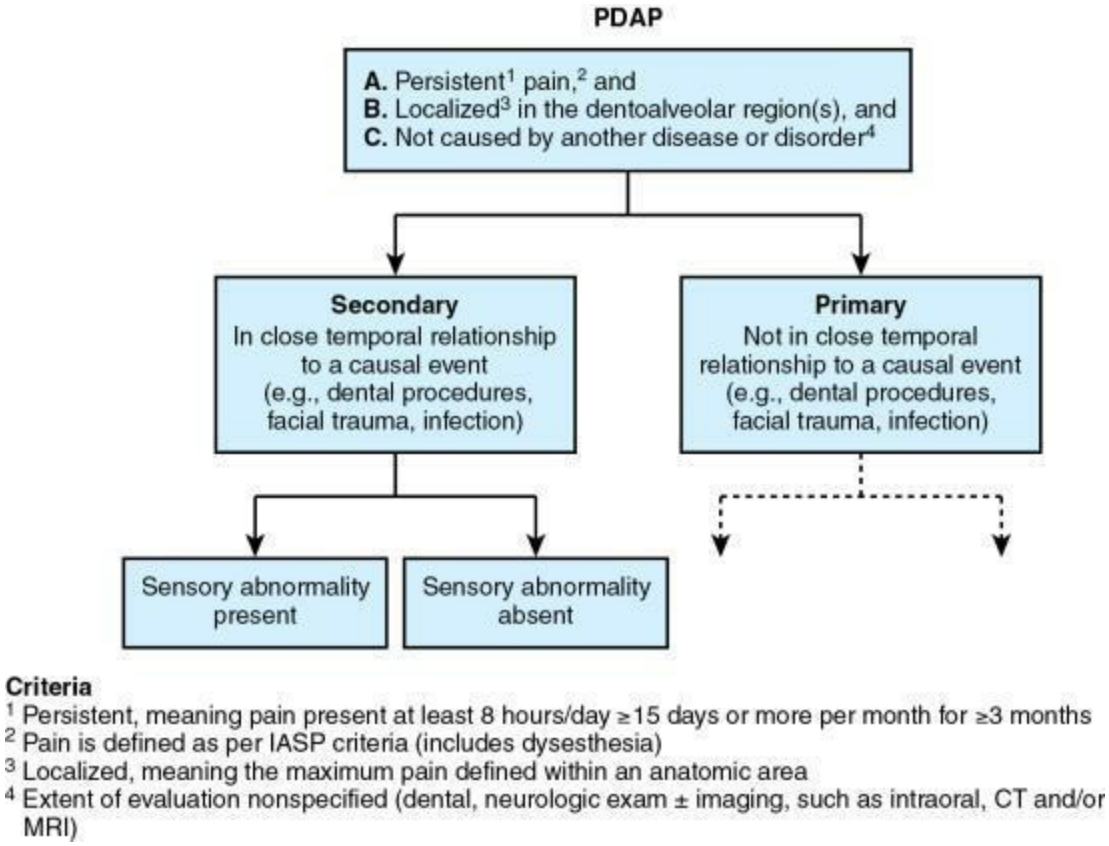

FIG. 4.7 Diagnostic criteria for persistent dentoalveolar pain disorder *(PDAP)*. *CT,* Computed tomography; *IASP,* International Association for the Study of Pain; *MRI,* magnetic resonance imaging.

Flow chart shows criteria for PDAP. A. Persistent (1) pain, (2) and; B. Localized (3) in the dentoalveolar region(s), and; C. Not caused by another disease or disorder (4). It leads to two text boxes, one is titled secondary and the other is titled primary. Text below secondary reads, In close temporal relationship to a causal event (e.g., dental procedures, facial trauma, infection). Text below primary reads, Not in close temporal relationship to a causal event (e.g., dental procedures, facial trauma, infection). The first text box titled secondary further leads to two boxes, each reads, sensory abnormality present.

Four criteria's are as follows:

1. Persistent, meaning pain present at least 8 hours/day greater than or equal to 15 days or more per month for greater than or equal to 3 months.

2. Pain is defined as per IASP criteria (includes dysesthesia).

3. Localized, meaning the maximum pain defined within an anatomic area.

4. Extent of evaluation non-specified (dental, neurologic exam ± imaging, such as intraoral, CT and/or MRI).

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Once a nerve has been sensitized via injury or disease it may remain so and present as a peripherally sensitized nerve. This peripheral sensitization and the ongoing pain (nociceptive barrage) that accompanies it can induce changes in the central nervous system. *Peripheral* sensitization and *central* sensitization can potentially impact the clinical presentation of a neuropathy. A typical clinical course of someone with an undiagnosed neuropathy might consist of treatment for a toothache. When the pain does not resolve with nonsurgical root canal treatment, it might then be followed by apical surgery and then perhaps an extraction. The extraction site might then be explored and debrided in a misguided attempt to remove any potential source of the patient's ongoing pain. After each treatment, there tends to be a reduction of the pain for a short time and then a return to its original, or even increased, level of pain intensity. It is likely that this is a result of a new neural injury consisting of reorganization and resprouting that increases the inhibition of nerve firing for a time. Surgical approaches to neuropathies are not effective because they do not desensitize the nerve. On the contrary, surgical intervention may aggravate the situation by inflicting an additional neural injury in the periphery and contributing to the already present nociceptive input. This intervention therefore puts the patient at increased risk of developing persistent pain, which is supported by a couple of long-term observational studies[3,113] and further supported by the observation that patients with pain following root canal therapy did not uniformly experience elimination of this pain with apicoectomy surgery.[108]

A diagnosis of neuropathy is based primarily on history and examination with the use of selected diagnostic tests to rule out other potential etiologies. The history should reveal some inflammation-inducing event (see the earlier sections Neuritis and Neuroma), although the nature of the initial insult is not always identified, as seemingly spontaneous development of such pains has been reported.[99] Typically, the examination is grossly unremarkable with no evidence of local tissue damage, leaving the clinician to rely mainly on the patient's report of symptoms. Although quality of pain is no longer thought to be capable of distinguishing neuropathic pains from others, patients repeatedly reported several features that may be key for identification purposes (Box 4.2).[40] Regarding examination features, the area where the pain is perceived may be hyperalgesic or allodynic—that is, noxious

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stimulation to the area will be perceived as more painful or nonnoxious stimulation will now be perceived as painful. This phenomenon is documented by reports where standardized applications of stimuli to the affected tissue are performed and demonstrate exaggerated responses.[85,90,146] Besides gain in sensory function, loss of function has also been observed,[85] which is more in line with a general definition for neuropathic pain.[135] Furthermore, maintenance of pain following local anesthesia to the affected region[83] and a lack of pain reduction with fentanyl and ketamine[7] all suggest the role of a central pain-related mechanism.

Box 4.2

Recurrent Patient-Reported Themes Regarding Their Persistent Dentoalveolar Pain Disorder

- Difficult for patients to respond to history taking because their words do not adequately describe what they feel; therefore, time may be needed to obtain the necessary information
- Well localized to a region within the dentoalveolar structures
- Pain is perceived to be deep in the tissues, rather than on the surface
- Continuous pain, one that never stops and seems to always be there
- Pain has the sensation of feeling pressure with a dull ache quality
- Complex and confounding descriptors, such as itching, tingling, or pricking, are sometimes present

Research related to the diagnostic imaging of cases that may be PDAP suggests two roles: first, to identify pathosis contributing to the pain presentation and, second, as a means of obtaining a positive finding for this enigmatic chronic pain disorder. As for the first role, diagnostic imaging is recommended to assess for odontogenic-related pathosis and other regionally presenting disease, as most pain in the dentoalveolar region is tooth related. For patients suspected of having PDAP, the diagnostic yield of cone-beam computed tomography (CT) was reported to be superior to periapical radiograph, but the findings were of questionable significance.[112] For patients without local pathosis and PDAP, magnetic resonance imaging (MRI) of the

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brain revealed several cases of intracranial findings thought to be related to the pain presentation (e.g., cysts, tumors, infarcts).[104] This is consistent with the clinical experience of one of the authors, which has led to brain imaging being routine prior to a diagnosis of PDAP being rendered. As for the second role of diagnostic imaging, conventional dental radiographic techniques have consistently been found to be unable to identify patients with PDAP, thus prompting pilot investigation into other imaging techniques. Results suggest high levels of sensitivity and specificity can be reached using thermography[64] revealing a "cold" image profile.[63] On the contrary, results suggest that a technetium-99 bone scan had low sensitivity and specificity for detecting dentoalveolar regions with chronic pain,[32] and MRI techniques do not seem to have been studied.

Neuropathic disorders have a predilection for women but can affect both genders. These patients are usually older than 30 years of age and may have a history of migraine.[126] In the orofacial region, neuropathies are most commonly seen in the maxillary premolar area and molar region.[61,108]

Neuropathies can be classified on the basis of their clinical presentation and response to therapies. *Peripheral neuropathy* may develop after sensitization of a peripheral nerve and presents clinically as described previously. Diagnosis of peripheral neuropathy is based on its favorable response to peripheral neural blockade. Treatment is directed at decreasing the sensitization of peripheral nerves and reducing ectopic neuronal firing. Topical as well as systemic medications can be used to treat cutaneous peripheral neuropathies. Topical medications include topical anesthetics, capsaicin-containing compounds, and anticonvulsants, as well as nonsteroidal antiinflammatory drugs (NSAIDs), sympathomimetic agents, and *N*-methyl-d-aspartate (NMDA) receptor-blocking agents[109] with encouraging results.[72]

The clinical presentation of a *central neuropathy* is similar to that of a peripheral neuropathy. After sensitization of peripheral nerves and the accompanying nociceptive barrage, the pain is nonremitting and lacks evidence of tissue insult. Unlike its peripheral counterpart, allodynia and secondary hyperalgesia are clearly present—that is, the area of pain is significantly larger than the initial site of injury. The most telling sign that a neuropathy has taken on a more central component is that local anesthetics are no longer effective. Therefore, the treatment must be directed toward the

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central processing of pain. This is done with medications such as NMDA receptor agonists (ketamine), gabapentin, tricyclic antidepressants, and opioids. The prognosis for a central neuropathy is not as good as for a peripheral neuropathy, as central neuropathic pain tends to become more refractory with time. Treatment is often based on the management of pain, rather than its cure, and sometimes is best performed in a multidisciplinary chronic pain clinic facility.

The last variation of neuropathic pain is sympathetically enhanced or maintained pain. In cases of sympathetically maintained pain (SMP), peripheral nerve fibers upregulate the expression of adrenergic receptors, making them responsive and sensitive to sympathetic input. SMP may also have a central component, whereby the constant sympathetic drive alters neuronal excitability. Neuronal injury may induce sprouting of sympathetic axons into the trigeminal spinal nucleus because basket-like formations of sympathetic fibers have been reported around the cell bodies of sensory neurons in the dorsal root ganglia.[141] Increases in sympathetic drive, such as with stress and fever, may aggravate SMP. Diagnosis of SMP is based on blocking sympathetic outflow to the affected region via sympathetic nerve blocks. In the orofacial region this would require a stellate ganglion block. The block is considered diagnostic for SMP if it effectively decreases the patient's pain. Multiple blocks can also be used as a form of therapy. Other therapies include drugs that target peripheral α_2-adrenoceptors (agonists) or α_1-adrenoceptors (antagonists), such as guanethidine, phentolamine, and clonidine. SMP presenting in the orofacial region is extremely rare and therefore makes clinicians prone to deriving a falsely positive diagnosis of this condition.[54,101] Furthermore, researchers have failed to produce SMP-type pain in animals,[10] something that is presumed to be due to the fact that the efferent nerve fibers in the head and neck region run with the blood vessels as opposed to the afferent nerves, as they do elsewhere in the human body. For these reasons, the likelihood of this type of pain presenting as "toothache" is extremely low and thus does not require much discussion here.

Toothache referred from a distant organic source

A variety of pathologies that seem to be unrelated have been reported to present as nonodontogenic toothache.[107,115] The only common link that can

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be identified is that branches of cranial nerves innervate the involved tissues and hence the trigeminal nucleus processes nociceptive input. Therefore, conceivably, any somatic structure with cranial nerve innervation has the potential to cause pain that the patient perceives as a toothache. For this reason, once dentoalveolar etiologies for such pain have been ruled out, all possible sources of nonodontogenic pain, including distant pathology, should be considered in the differential diagnosis. Several of these types of organic pathologies that have been reported to present as toothache are described in the following sections.

Cardiac and thoracic structures.

Cardiac pain has been cited as the cause of nonodontogenic toothache in a number of case reports.[9,41,62,77,97,136] Classically, cardiac pain presents as a crushing substernal pain that most commonly radiates to the left arm, shoulder, neck, and face. Although not as common, anginal pain may present solely as dental pain, generally felt in the lower left jaw.[16] Similar to pain of pulpal origin, cardiac pain can be spontaneous and diffuse, with a cyclic pattern that fluctuates in intensity from mild to severe. The pain can also be intermittent and the patient may be completely asymptomatic at times. The quality of cardiac pain when referred to the mandible is chiefly aching and sometimes pulsatile. Cardiac pain may be spontaneous or increased with physical exertion, emotional upset, or even the ingestion of food.[9] Cardiac pain cannot be aggravated by local provocation of teeth. Anesthetizing the lower jaw or providing dental treatment will not reduce the pain. It can be decreased with rest or a dose of sublingual nitroglycerin. Diagnosis of cardiac pain, along with immediate referral, is mandatory to avoid impending myocardial infarction.

Besides pain of cardiac origin, other chest structures have been reported to produce nonodontogenic toothache pain. Various cancerous lesions of the lungs have been described to present a mandibular pain, with the pain being both ipsilateral and contralateral to the side where the tumor is present.[24,59] Furthermore, diaphragmatic pain is mediated via the phrenic nerve and may present as nonodontogenic tooth pain.[15]

Intracranial structures.

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Space-occupying lesions within and around the brain are known to impinge on structures innervated with somatosensory fibers, such as the dural and perivascular tissues, causing pain. These pains are highly variable, with a common complaint being headache or head pain. Just as intracranially derived pain may be referred to the face and jaws in neurovascular disorders, it may also present as a toothache.[137] To outline the vast differences in clinical features of such pain, intracranial lesions have also been reported to cause trigeminal neuralgic pain in response to treatment of what was first thought to be toothache.[29] This extreme variability has been observed by one of the authors, which leads to the recommendation that if local etiologic factors are not readily identified in a patient with toothache symptoms, magnetic resonance brain imaging should be considered.

Throat and neck structures.

Nonodontogenic toothache has been reported to arise from various structures of the neck, but these reports are sparse, and hence, it is not possible to draw conclusions regarding how patients with these pain-provoking disorders may present. Squamous cell carcinoma of the lateral pharyngeal surface presenting as ipsilateral mandibular molar pain has been observed by one of the authors. This finding is consistent with previous reports of nonodontogenic pain being associated with smooth muscle tumors of a similar location.[139] Vascular structures of the neck have also been implicated in the production of toothache symptoms, with a report of a patient initially presenting for dental care when pain was from the result of a life-threatening carotid artery dissection.[119]

Craniofacial structures.

Clinically, pain from other craniofacial structures has been observed as being the most common reason for organic pathologies presenting as nonodontogenic toothache, likely because these structures are innervated by branches of the trigeminal nerve. Tumors in the maxillary sinus[27,43,145] and jaw,[132] as well as metastatic disease, particularly within the mandible,[33,53,114,124] have been reported. The clinical presentation of symptoms is highly variable, but a common feature is sensory loss along the distribution of the nerve, the result of pain arising from nerve impingement.

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This underscores the need for regional imaging techniques, such as pantomography or CT (as opposed to just periapical radiographs). This is especially true in patients who have a history of cancer. One must also not forget that nerve impingement anywhere along the distribution of the trigeminal nerve, even within the cranial vault itself,[20] can elicit nonodontogenic tooth pain.

Vascular structures within the craniofacial region have also been reported to present as nonodontogenic toothache, with arteritis being the pain-provoking pathology.[68,73] These pains have been described as a continuous dull pain that can sometimes be made worse with jaw function. The stereotypical presentation includes a history of eyesight changes, such as blurred vision, and the examination feature of pulseless, indurated temporal arteries that are painful to palpation. A laboratory finding of an elevated erythrocyte sedimentation rate (ESR) is suggestive of the disorder, and diagnosis is confirmed by temporal artery biopsy. Treatment includes administration of corticosteroids; therefore, because permanent blindness is a possible sequela if cranial arteries are left unmanaged, immediate referral to the appropriate medical colleague is indicated.

Psychogenic toothache

A group of mental disorders known as somatoform disorders, which is when a patient has somatic complaints yet lacks a physical cause, is the only type of psychological disorder that has the potential of presenting as tooth pain. Patients with somatoform disorders are not fabricating the suffering or consciously seeking benefit with their toothache complaint; rather it is a reflection of their distorted perception and presentation based on their psychological disorder. There are four specific diagnoses worth covering within this group, to provide a basis of the different presentations, and they are[111a]:

1. A conversion disorder, which is when a neurological symptom that is inconsistent with a neurologic disease but is nevertheless genuine and distressing yet attributable to a psychological trigger, such as in an instance when a child witnesses the tragic deaths of both parents and becomes mute.[5] It is possible a conversion disorder could manifest as

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a psychogenic toothache, but to our knowledge it has not been reported in the literature and has not be observed in our clinical practices. Rather, when the psychologic disorder manifests as pain, the complaint tends to be more regional in nature and not as specific as toothache.

2. A somatization disorder, and its variants, present with unexplained physical symptoms beginning at a young age of an adult (i.e., <30 years old), persisting for years in duration, and multiple complaints of gastrointestinal, pain, neurological, and sexual symptoms.[111a] Somatization disorder is up to 10 times more common in females, much more common related individuals, but no definitive cause has been identified. There is usually a pattern of high levels of health care utilization, with such individuals being very familiar in chronic pain clinics because of the persistence of a pain complaint being part of the presenting symptomology. While somatization disorder is usually only discussed in general as it may present a tooth-related complaint and the frequency of such presentations is unknown, it is well known to present with TMDs, and screening for somatoform symptoms is included in the Diagnostic Criteria for TMD.[90,110,126]
3. Hypochondriasis is a misinterpretation of a physical symptom and fixation on the fear of it being a life-threating condition, presenting in equal proportions in men and women.[111a] Therefore, strictly speaking, a hypochondriacal patient would present with odontogenic tooth pain that is real, so it would not be a nonodontogenic reason for toothache, and the psychological disorder would manifest as exaggerated fear in relationship to the meaning this pain has to the patient.
4. Body dysmorphic disorder is a debilitating preoccupation with a physical defect, real or imagined[111a]. Sensory perceptions are not part of this disorder's presentation, but related complaints such as an "uneven bite" is what has been reported in the literature in relation to this disorder.[81]

It is important to make a distinction between somatoform disorders and factitious pain or malingering behavior.[5] In factitious pain there are physical

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or psychological symptoms that are produced by the individual and are under voluntary control. Malingering is similar to factitious pain with the added characteristic that the symptoms are presented for obvious and recognizable benefit.[54] This category poses a significant diagnostic challenge because evidence of local tissue damage typical of heterotopic pain entities previously discussed in this chapter is lacking. It is important to emphasize that psychogenic pain is rare. When arriving at this diagnosis it is critical that all other potential diagnoses have been ruled out.

The diagnosis of psychogenic toothache is one of exclusion and is based on the clinician's awareness of other heterotopic pain characteristics and behavior. Of particular note are centrally emanating pains, cardiac pain, neurovascular pain, and neuropathic pain. Adding to the diagnostic difficulty is that comorbid psychologic disorders are commonly present with chronic intraoral pain disorders, including those erroneously presenting as toothache.[84,130] This has led to the current thinking that psychologic disorders (i.e., depression, anxiety, somatization) may not be related to the initiation or perpetuation of chronic pain disorders but rather are a consequence of living with chronic pain.

Psychogenic pain is known to be precipitated by severe psychologic stress. These pains present a general departure from the characteristics of any other pain condition—that is, they may not fit normal anatomic distributions or physiologically plausible patterns. The pain may be felt in multiple teeth and may jump around from one tooth to another. The intensity of pain reported tends to be more severe than is reflected by the patient's level of concern about the condition. Patients' responses to therapy are variable and include a lack of response or an unusual or unexpected response. Early identification of psychogenic pain and referral to a psychologist or psychiatrist is necessary to avoid irreversible and unnecessary dental treatment.

Frequency of nonodontogenic toothache

The prevalence of nonodontogenic toothache in the population is unknown, as is the prevalence of such a presentation for dental care. Specific to endodontics, pain is thought to be present in 5.3% of patients 6 or more months following treatment[100] with about half of those individuals being estimated to have nonodontogenic etiologies accounting for their complaint

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of that pain by literature review.[100] Clinical evaluation of patients that received root canal treatment 3 to 5 years prior revealed 5% experiencing persistent pain and about two thirds of those patients having nonodontogenic pain.[141a] A questionnaire-based assessment of root canal–treated patients 6 months following care found 10% reporting persistent pain, and a clinical evaluation of a subset of these patients revealed about half having nonodontogenic pain.[102a] The most common diagnosis of nonodontogenic pain in this small number of patients, comprising about three fourths of the cases, was MFP referring pain from muscles of mastication, with the remaining one fourth being neuropathic pain.[102a] These diagnoses and distribution of patients with nonodontogenic pain is similar to what was reported as being referred for evaluation by endodontists.[82a]

Taking a patient's history

Pain diagnosis is largely based on the patient's subjective history; however, patients rarely give all pertinent diagnostic information about their pain of their own accord. Often it is necessary to carefully extract the details of the patient's pain complaint through systematic and thorough questioning. This is known as "taking a history," and it involves both careful listening and astute questioning. Fig. 4.8 is an example of a basic diagnostic workup for odontogenic pain. It can be easily used to obtain histories of typical odontogenic pain by circling all descriptors that apply and then filling in the remaining blanks. As the details of a patient's pain complaint are gathered, the clinician should be mentally progressing through an algorithm of possible diagnoses, as each detail should lend itself to one type of pain over another. After completing a thorough and accurate history of the complaint(s) (Fig. 4.9), often the diagnosis has already been narrowed down to one particular pain entity. This is particularly true with odontogenic pain. The only question that will remain is "which tooth is it?" It is critical to keep in mind that whereas patients will provide information about the perceived site of pain, it is the clinician's examination that will reveal the true source of their pain. With more complicated pain complaints, the clinician may have a list of possible diagnoses. This is known as a differential diagnosis. This differential will guide the examination and testing in an effort to confirm one diagnosis

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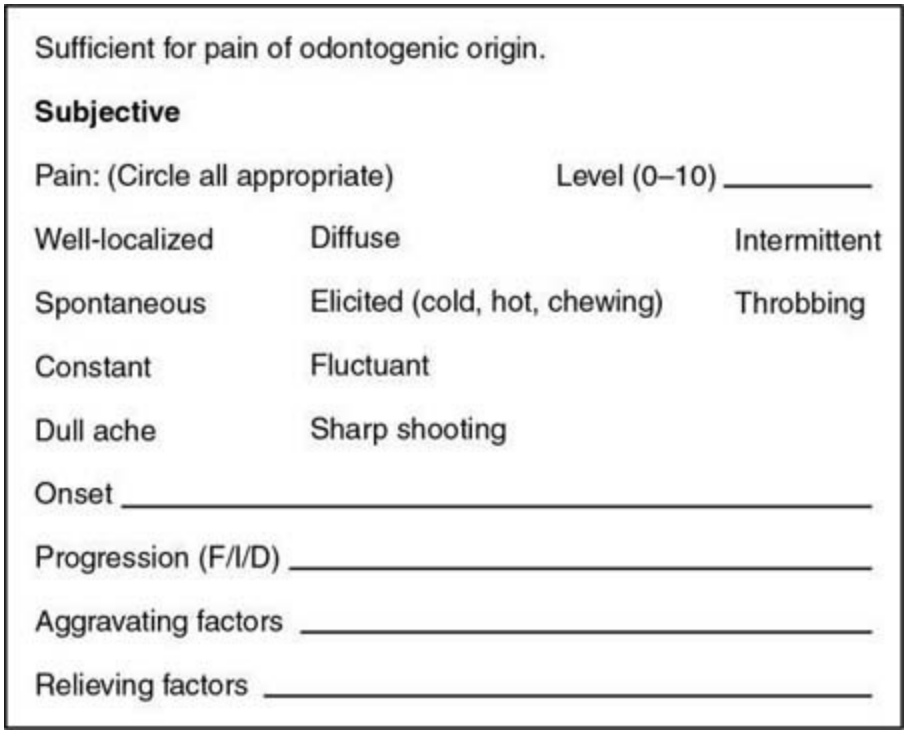
while ruling out all others. If after completing the subjective examination all items on the differential are outside the clinician's scope of practice, then the clinician should continue the examination until he or she has a firm idea of the possible diagnosis so that a proper referral can be made. In addition, it is paramount that all odontogenic sources have been ruled out and that this information is communicated to the health care provider to whom the patient is referred. If no differential can be formulated after the history has been taken, then the history should be redirected to the patient to confirm that the information is complete and accurate. If the patient is unable to provide sufficient information regarding the pain complaint, then it may be helpful to have the patient keep a pain history, detailing the aspects of the pain on a daily basis. Of utmost importance is to avoid treatment when the diagnosis is uncertain. Diagnostic therapy (i.e., "let's do a root canal treatment and see if it helps") may result in costly treatment that does not improve the patient's condition and could be a factor in aggravating and perpetuating a patient's pain. Treatment should always specifically address a diagnosis.



Sufficient for pain of odontogenic origin.

Subjective

Pain: (Circle all appropriate) Level (0–10) _____

Well-localized	Diffuse	Intermittent
Spontaneous	Elicited (cold, hot, chewing)	Throbbing
Constant	Fluctuant	
Dull ache	Sharp shooting	

Onset _____

Progression (F/I/D) _____

Aggravating factors _____

Relieving factors _____

FIG. 4.8 Example of a form to evaluate odontogenic pain.

Template is titled "sufficient for pain of odontogenic origin."

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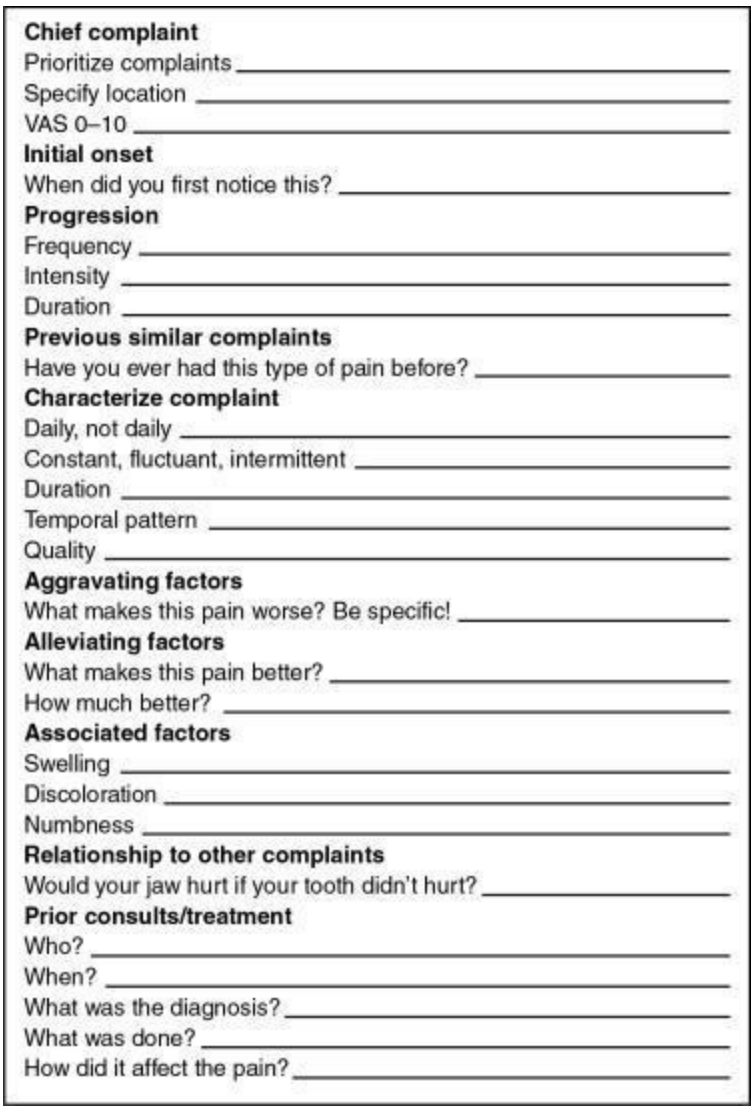
Text reads, subjective. It is followed by pain: (Circle all appropriate) and level (0 to 10) blank space.

The options are well-localized, diffuse, intermittent, spontaneous, elicited (cold, hot, chewing), throbbing, constant, fluctuant. dull ache, and sharp shooting. This followed by blank space for onset, progression (F/I/D), aggravating factors, and relieving factors.



Chief complaint
Prioritize complaints _____
Specify location _____
VAS 0–10 _____
Initial onset
When did you first notice this? _____
Progression
Frequency _____
Intensity _____
Duration _____
Previous similar complaints
Have you ever had this type of pain before? _____
Characterize complaint
Daily, not daily _____
Constant, fluctuant, intermittent _____
Duration _____
Temporal pattern _____
Quality _____
Aggravating factors
What makes this pain worse? Be specific! _____
Alleviating factors
What makes this pain better? _____
How much better? _____
Associated factors
Swelling _____
Discoloration _____
Numbness _____
Relationship to other complaints
Would your jaw hurt if your tooth didn't hurt? _____
Prior consults/treatment
Who? _____
When? _____
What was the diagnosis? _____
What was done? _____
How did it affect the pain? _____

FIG. 4.9 Example of a form to evaluate pain history. *VAS,* Verbal analog scale.

Template to evaluate pain history is as follows:

Chief complaint: Prioritize complaints, specify location, and VAS 0–10. Initial

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onset: When did you first notice this? Progression: Frequency, intensity, duration. Previous similar complaints: Have you ever had this type of pain before? Characterize complaint: Daily, not daily, constant, fluctuant, intermittent, duration, temporal pattern, quality. Aggravating factors: What makes this pain worse? Be specific! Alleviating factors: What makes this pain better? How much better? Associated factors: Swelling, discoloration, numbness. Relationship to other complaints: Would your jaw hurt if your tooth didn't hurt? Prior consults/treatment: Who? When? What was the diagnosis? What was done? How did it affect the pain? Blank spaces are given for filling information.

A complete medical history along with current medications and drug allergies should always be ascertained. It is also important to make note of demographic information, as patients of certain genders and ages are more at risk for some disorders compared with others.

Recording a patient's *chief complaint* in the patient's own words is a medical legal necessity but falls short of constituting a thorough pain history. A complete history begins with a patient's general pain complaint—for example, "My tooth hurts." Patients may have more than one pain complaint—for example, "My tooth hurts and it is starting to make my jaw hurt." All pain complaints should be noted and investigated separately. Understanding the specific components of the complaints makes it possible to discern the relationship between them—that is, either the complaints are wholly separate and two types of pathosis are present, or one source of pain is merely creating a heterotopic pain that is wholly secondary to the first.

Begin with determining the *location* at which the patient perceives the pain. Aspects of the location involve localization and migration. Pain should be definable as either well localized or diffuse and either superficial or deep. Easily localized superficial pain tends to be cutaneous or neurogenic. Musculoskeletal pain is felt deeply and is more localizable once it is provoked. Deep, diffuse pain is suggestive of deep somatic pain, be it visceral or musculoskeletal. Both tissue types are involved in a high degree of nociceptor convergence in the trigeminal nucleus and, therefore, much more likely to be involved in creating heterotopic pain. Typical referral patterns of deep somatic pain tend to follow peripheral dermatomes that reflect the laminations in the trigeminal nucleus. Referred pain also tends to occur in a cephalad direction. Therefore, referred pain from a deep somatic tissue such as tooth pulp, cardiac tissue, or skeletal muscle will respect this pattern. Pain

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that spreads distally along a nerve branch is much more indicative of a projected type of heterotopic pain. Projected pains imply a neurogenic source and possibly one that is secondary to impingement from intracranial pathosis. Recall that superficial sources of pain are not likely to be involved in referral, so if a patient is indicating that the pain is superficial and spreading, this is highly suggestive of a neurogenic rather than a cutaneous source.

Assessment of the *intensity* of pain is easily accomplished using a verbal analog scale (VAS). This question is best phrased, “On a scale of one to ten, zero being no pain and ten being the worst pain you can imagine, how bad is your pain?” Not only can intensity provide insight about pain type; it can also help guide posttreatment pain management as well as provide a baseline for response to therapies.

Identifying the *onset* of pain may provide information regarding etiology. Question if the onset followed a particular event such as a dental appointment or a traumatic injury. Beware of these relationships, as they can be misleading. Having a temporal correlation does not necessarily ensure a cause-and-effect relationship. The onset of pain may be either gradual or sudden. Severe pain of sudden onset can signal a more serious problem. Pain that has been present over a protracted period of time, particularly if the pain has been unchanging, is highly suggestive of a nonodontogenic pain source.

Other *temporal* aspects of pain include frequency and duration. The clinician should ask the patient, “How often does the pain occur and how long does it last?” These temporal aspects may establish patterns that point more clearly to one condition over another.

Progression of the patient’s pain over time should be noted. Whether pain is better, worse, or unchanging since its onset should be broken down into three factors: frequency, intensity, and duration. Static pain that does not change over time is typically not odontogenic in origin.

The *quality* of pain—that is, “what it feels like”—is a critical aspect of a pain history. Knowledge of pain characteristics as they relate to tissue types is essential. Pain quality can be difficult for patients to describe, and it is often necessary to provide them with a list of descriptors from which to choose. In instances of odontogenic pain, the list is fairly short. The deep visceral and musculoskeletal components of a tooth limit true odontogenic pain to having qualities that are dull, aching, or throbbing. If there is an aspect of sharpness to the pain, it is helpful to understand whether the

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sharpness is stabbing in nature, which would indicate A-delta fiber–mediated dentinal pain, or whether it is electrical in nature, which would indicate neuralgia. Common examples of pain descriptors and their respective pain types are listed in Table 4.1.

Table 4.1

Examples of Pain Descriptors

Origin	Quality of Pain
Muscular	Dull, aching
Neurogenic	Shocking, burning
Vascular	Throbbing, pulsatile

Factors that precipitate or aggravate the patient's pain complaint are of key importance in diagnosis. Not only do aggravating factors suggest the tissue types that may be involved, but they also aid in directing the objective tests. When gathering information, it is important to be specific. If a patient reports pain while eating, keep in mind that many structures are stimulated during mastication, such as muscles, TMJs, mucosa, PDLs, and, potentially, pulps. Be specific as to the aggravating factor. The lack of any aggravating factors indicates that the pain is not of odontogenic origin.

Alleviating factors can provide insight as to the nature of the pain. If a medication relieves the pain, it is critical to know the specific medication, its dosage, and the degree to which the pain was attenuated. It is equally important to know what has no effect on the intensity of the pain. For example, if a pain of midlevel intensity is completely unresponsive to anti-inflammatory drugs, then it is probably not inflammatory in origin.

Associated factors such as swelling, discoloration, and numbness must be ascertained, as well as their correlation with symptoms. Swelling of acute onset suggests an infection, and its concurrent pain would be of inflammatory origin. Swelling that comes and goes with the intensity of pain suggests an autonomic component. The same can be said for discoloration, such as redness. Numbness or any other type of sensory aberrations should be recorded. If the altered sensation is a major component of the pain complaint, then it should be investigated separately and its relationship to the pain

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determined. Pains that occur with sensory aberrations tend to have a strong neurogenic component.

If the patient complains of more than one pain, an effort should be made during the subjective history to determine the *relationship of the complaints*. One pain might serve as an aggravating factor to the other. There may be a correlation as to the onset, intensity, or progression of the complaints. Also, keep in mind that patients may actually have more than one type of pathosis occurring concurrently and there may be no relationship whatsoever. Ask whether there have been any *previous similar complaints* and, if so, what happened. Recurrence of similar pains might reveal a pattern that lends itself to a particular pain diagnosis.

It is critical to gain knowledge of any prior consultations that have occurred. Details regarding the type of clinician, actual workup performed, and diagnosis rendered will help to narrow down a differential. Any treatment that was performed should be ascertained along with its effect on the chief complaint.

Patient examination

As stated previously, the purpose of the history is to gather information about a patient's pain complaint in order to formulate a list of possible diagnoses based on specific pain characteristics. Poor or improper symptom analysis will lead to a false differential, and any testing will therefore have limited meaning. Performing a general examination, including an extraoral, intraoral, and hard- and soft-tissue assessment, is requisite to confirm the health of various structures and to identify possible pain-producing etiologies. When a patient presents with a toothache, the pain is usually of odontogenic origin. Diagnostic procedures are often limited to confirming a suspect tooth rather than identifying a nonodontogenic source of pain. Standard pulpal and periradicular tests serve to aid in both ruling in odontogenic pain and therefore ruling out nonodontogenic pain as a diagnosis. Remember that the site of pain is determined by patient history, but the true source of the pain should be revealed with testing. If the chief complaint cannot be reproduced with standard tests, then additional tests may be necessary to narrow down a differential diagnosis. For details on general examinations and standard tests, please refer to Chapter 1.

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Additional tests

Further tests should be chosen with forethought in an effort to develop a workable differential that can guide the clinician toward a meaningful consultation or an appropriate referral for the patient. These tests may consist of palpation or provocation of various structures, sensory testing, or diagnostic blocks. The application of these tests is not covered in detail in this chapter. For more information about the application and interpretation of these tests, please consult other sources.

Palpation and percussion are common tests to differentiate odontogenic pain from pain of sinus origin. Palpation of the sinuses consists of firm pressure placed over the involved sinus (usually maxillary). In addition, pain of sinus origin may be provoked with a lowering of the patient's head.

If pain of muscular origin is suspected, then an attempt to reproduce this pain can be done by palpation of the muscles of mastication or provocation via functional manipulation. The temporalis, deep and superficial masseter, medial pterygoid, and digastric muscles should be palpated in an effort to discover tenderness or trigger points that reproduce the pain complaint. The medial pterygoid is only partially accessible to palpation and may need to be functionally tested by stretching the muscle (opening wide) or contracting the muscle (biting firmly). The lateral pterygoid may be difficult if not impossible to palpate intraorally and therefore is more appropriately assessed by functional manipulation. Pain emanating from this muscle may be increased by protruding the jaw against resistance. Exacerbating the chief complaint by muscular function provides a strong indication of a myofascial source of pain.

Because of the complexity of innervation and the occurrence of heterotopic pain in the orofacial region, it may be difficult to definitively determine the origin of pain by testing alone. It cannot be stressed enough that primary pain should not only be provoked by local manipulation but also be relieved by anesthetic blocking. In diagnostic anesthesia the relief of pain has a typical onset and duration, depending on the particular anesthetic used. In addition, the pain should be completely diminished or else suspicion of a central component or a coexisting disorder should arise. The use of diagnostic anesthesia may be necessary and useful in augmenting the diagnostic workup (Fig. 4.10). Topical anesthetic can be helpful in the investigation of

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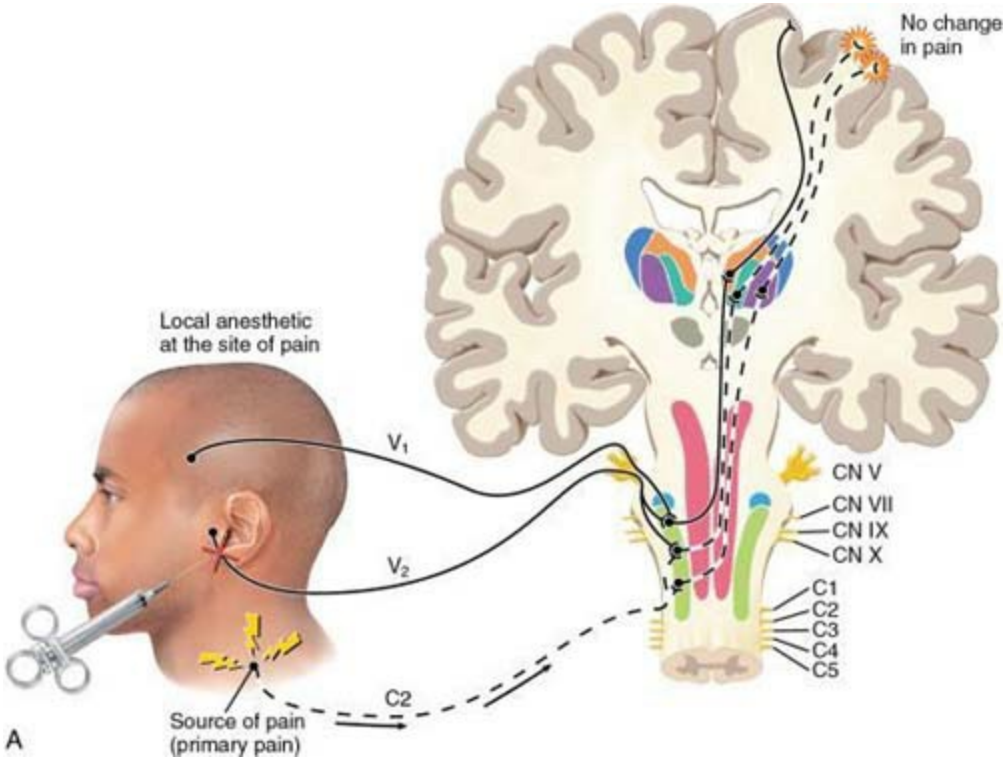
cutaneous pain and peripheral neuropathies. Anesthetic injection, including peripheral nerve blocks, can be used to determine whether the etiology of the pathosis is peripheral to the area of the block. Pain that persists after the onset of usual signs of anesthesia suggests a central component. The patient's history and general examination are of key importance in differentiating between the pain of a central neuropathy and the central pain emanating from an intracranial mass.



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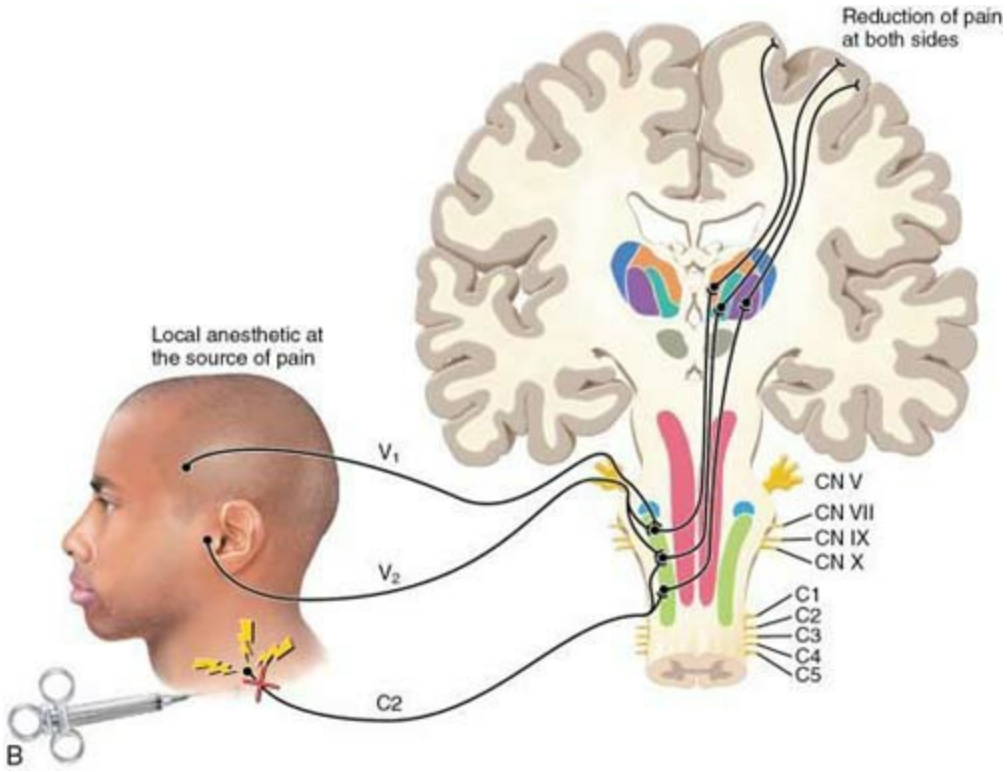

FIG. 4.10 **A**, Local anesthetic at the site of pain fails to reduce the pain. **B**, Local anesthetic at the source of pain reduces the pain at the source as well as at the site.

A) A tree-like structure, the trigeminal nerve, on the right shows labels as follows: CN V, CN VII, CN IX, CN X, C1, C2, C3, C4, and C5. To the left, side view of human face is shown. V1, V2, and C2 from the trigeminal nerve lead to regions near the forehead, tragus of ear, and neck at the center. Source of pain (primary pain) is marked in the neck. Local anesthetic at the site of pain is injected near the ear that doesn't cause any change in pain.

B) Local anesthetic at the source of pain is injected, which results in reduction of pain at both sides.

Source: (Redrawn from Okeson JP: *Bell's orofacial pains*, ed 5, Chicago, 1995, Quintessence Publishing.)

Pain that is primarily muscular, as suggested by trigger points discovered on examination, can be further investigated by local anesthetic injection into the trigger point. Trigger point injections are typically performed with either a 27- or a 25-gauge needle and a minimally myotoxic anesthetic such as 2% lidocaine or 3% mepivacaine without a vasoconstrictor. Myofascial trigger point injections may temporarily relieve pain at the trigger point as well as at the site of referral.

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Sympathetic efferent activity can play a role in the enhancement or maintenance of chronic pain. In the head and neck, sympathetic activity flows through the stellate ganglion located bilaterally near the first rib. When there is suspicion of a sympathetic component to a patient's pain, a stellate ganglion block can be used to provide diagnostic insight. A trained anesthesiologist usually performs this procedure. An effective block delivered to the stellate ganglion will interrupt sympathetic outflow to the ipsilateral side of the face, resulting in a partial Horner syndrome. This is evidenced by flushing, congestion, lacrimation, miosis of the pupil, ptosis, and anhidrosis.[75] A sympathetic blockade that diminishes or eliminates a pain state may guide future treatment such as repeated blocks or systemic treatment with sympathetically active drugs (e.g., clonidine and prazosin).[116]

Neurologic conditions, both peripheral and central, can present as pain in the orofacial region. A role of the clinician is to help rule out gross neurologic conditions secondary to intracranial pathosis. Systemic complaints such as nausea, dizziness, or changes in one of the special senses should raise suspicion of intracranial pathosis. A neurologic screening examination, including a gross sensory and motor evaluation of cranial nerves II through XII, should be performed. For details on cranial nerve examination, please refer to other sources.[44] Investigation of sharp/dull differentiation as well as light touch discrimination between the different branches of the trigeminal nerve can provide insight as to the location and etiology of the pathosis.

Case studies

Case 1

A 56-year-old male presents with a chief complaint of "This tooth still hurts and it's getting worse. It even hurts when I smile." He has a history of angina secondary to a 70% occlusion of his right coronary artery. He also reports a history of hypercholesterolemia. He has no history of myocardial infarction and denies any other significant medical history. The patient is taking lovastatin (Mevacor, 400 mg/day), nifedipine (Procardia, 60 mg once per day), and atenolol (50 mg once per day). He has no known drug allergy.

The patient was referred by a periodontist for evaluation of continued pain associated with tooth #3. He had been receiving periodontal maintenance

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therapy for generalized moderate adult periodontitis for more than 5 years. He had root canal treatment and a mesial buccal root amputation of tooth #3 as treatment for a localized area of advanced periodontitis 6 months ago.

Subjective history.

After careful questioning, it becomes apparent that the patient is experiencing pain of two different qualities: an intermittent ache associated with tooth #3 and an intermittent sharp shooting pain also associated with tooth #3. The intermittent dull ache was of gradual onset 9 months ago. This pain was unaffected by the nonsurgical root canal treatment and root amputation. This pain has increased in frequency, intensity, and duration over the past 3 months. There is no temporal component. The dull ache is aggravated by biting and by the occurrence of the sharp shooting pain. The sharp shooting pain had a sudden onset 6 months ago. It has also increased in frequency, intensity, and duration without a temporal component. It can occur spontaneously or when the patient is “smiling big.” The patient reports that the sharp shooting pain can also be aggravated by pressing lightly on his face in the area overlying #3, but not by pressing intraorally on #3.

Examination.

The coronal portion of the amputated mesiobuccal root had been restored with IRM (Intermediate Restorative Material; DENTSPLY Caulk, Milford, DE). No cracks, fractures, sinus tracts, or swelling is detected. There are generalized probing depths of 4 mm throughout the upper right sextant. Tooth #3 has an 8-mm-broad probing defect mesially with bleeding on probing. For the results of clinical testing, see Table 4.2.

Table 4.2

Case 1: Clinical Results of Testing

	TOOTH		
Test	**#2**	**#3**	**#4**
Endo Ice*	+ (s)†	–	+ (s)
Percussion	–	+	–
Palpation	–	–	–

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*Endo Ice (Coltène/Whaledent, Cuyahoga Falls, OH) is used to detect pulp vitality.

†*s*, Pain of short duration.

A periapical radiograph (Fig. 4.11) shows evidence of prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3. Mild to moderate horizontal bone loss is evident in the quadrant. No radiographic evidence of caries or apical radiolucencies is noted.

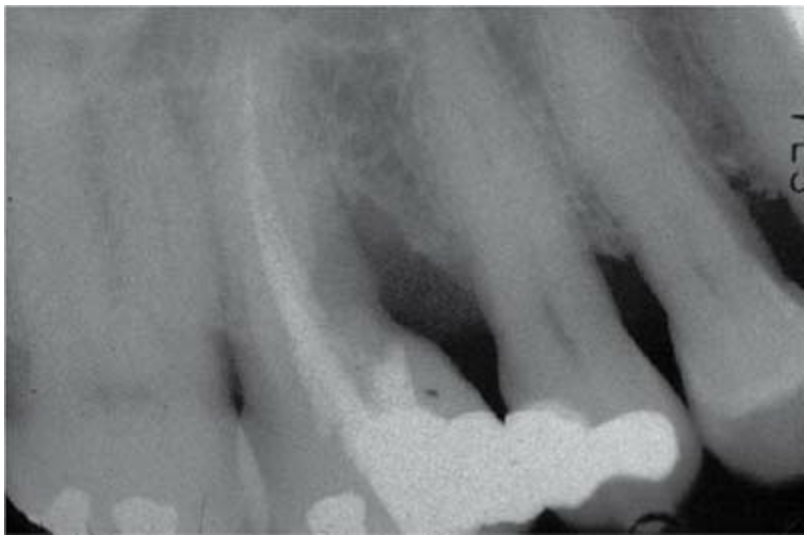

FIG. 4.11 Periapical radiograph showing prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3.

Radiograph shows four teeth. Crowns of two teeth have a large diffused radiopaque patch. Root canal of one of the two teeth is filled with a significant radiolucency near it. The gum line shows low-density tissue in-between two teeth.

Additional tests.

In the absence of a clear etiology, a more extensive extraoral examination is performed. Cranial nerves II through XII are intact. The sharp shooting pain is predictably produced with light brushing of the skin over the area of tooth #3. This examination increases the patient's subjective complaint of a dull ache associated with tooth #3. With the likelihood of two possible sources of pain existing, a diagnostic anesthetic block of tooth #3 is performed: buccal infiltration of tooth #3 with 27 mg of 3% mepivacaine without epinephrine. After 3 minutes the patient no longer reports a dull ache at tooth #3 and he is

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nontender to percussion. His sharp shooting pain can still be initiated with light brushing of the skin over the area of tooth #3 and continues to cause a dull ache in the area of tooth #3. Diagnoses of trigeminal neuralgia and advanced localized adult periodontitis of tooth #3 are made. The patient is referred to a neurologist for evaluation and treatment. The diagnosis of trigeminal neuralgia is confirmed and he is placed on carbamazepine at 100 mg/day.

Case 2

A 28-year-old male presents with a chief complaint of “My teeth on the right side hurt.” His medical history is not significant. He denies any systemic disease and has no known drug allergies. He is currently taking 600 mg of ibuprofen as needed for pain. He is taking no other medications. The patient was referred by his general clinician for evaluation of pain associated with his teeth on the right side.

Subjective history.

After careful questioning, it is determined that the patient is experiencing pain of two different types. The most distressing pain to the patient is a diffuse, right-sided, constant low-grade dull ache (3/10 on a VAS). The onset was gradual, beginning 2 years ago. The pain has recently increased in intensity and duration. This pain is aggravated by opening wide and increases in intensity after the occurrence of a sharp pain that is induced by biting down. There is no notable temporal component, and the patient has made no attempts to obtund the pain.

His other pain type had a sudden onset approximately 4 months ago. This pain is localized to the area of the right first molars. It is an intermittent sharp shooting pain (8/10 on VAS) that occurs when biting.

Examination.

Tooth #3 has an occlusal amalgam with cracks evident on the mesial marginal ridge and the buccal groove. Tooth #30 has an occlusal amalgam, and cracks are noted on the mesial and distal marginal ridges of the tooth. There are no swellings or sinus tracts and no probing greater than 4 mm on the right side. A periapical radiograph demonstrates no evidence of caries or

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apical radiolucencies. The patient's sharp pain is reproduced with a bite test applied to the mesial lingual cusp of tooth #30. After the bite test, the patient reports that his dull ache has intensified. For the results of clinical testing, see Table 4.3. Thirty seconds after pulp testing has been completed, the patient again reports that his dull ache has intensified.

Table 4.3

Case 2: Clinical Results of Testing

	TOOTH					
Test	**#2**	**#3**	**#4**	**#31**	**#30**	**#29**
Endo Ice	+ (s)	+ (s)	+ (s)	+ (s)	+ (s)	+ (s)
Percussion	–	–	–	–	–	–
Palpation	–	–	–	–	–	–

Additional tests.

In consideration of an uncertain diagnosis, a more extensive examination is performed. Palpation and provocation tests of the muscles of mastication reveal a trigger point in the patient's right deep masseter. Palpation of this trigger point immediately intensifies his "toothache." A trigger point injection of 3% mepivacaine without epinephrine is done in an effort to clarify a diagnosis. After a trigger point injection, all tests are repeated. Palpation of the trigger point no longer produces pain. The bite test and cold test still produce a short sharp pain but are no longer followed by a dull ache.

Diagnoses of reversible pulpitis secondary to a cracked tooth #31 and MFP of the right masseter are made. The patient is given home care instructions for treatment of his MFP, and he is referred to his general clinician for cuspal coverage of both teeth #3 and #30.

Summary

As clinicians who are frequently called on to diagnose and treat complaints of orofacial pain, it is important to have a thorough knowledge of the odontogenic and nonodontogenic causes. The basis for this knowledge begins

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with an understanding of the anatomy and physiology of the pain system, and how alterations in this system can result in pain that is poorly localized and therefore misdiagnosed. A realization that pain does not always originate in the structures in which they are felt, along with an understanding of the neurobiologic basis of heterotopic pain, is necessary to ensure accurate diagnosis of orofacial pain.

There are several indicators that a toothache may be nonodontogenic in origin. Red flags for nonodontogenic pain include toothaches that have no apparent etiology for pulpal or periradicular pathosis; pain that is spontaneous, poorly localized, or migratory; and pain that is constant and nonvariable. In addition, pain that is described as burning, pricking, or "shock-like" is less likely to be pulpal or periradicular in origin.

A thorough pain history and an examination of dental and nondental structures are essential to differentiate between odontogenic and nonodontogenic sources of pain. Examples of key components of the pain history and examination are included in this chapter for reference. In addition, the chapter has focused on the more common nonodontogenic sources of orofacial pain. As stated previously, the role of the dental clinician is to diagnose and treat disorders of the oral cavity and masticatory structures. In the event that a nondental pathosis is suspected, a differential diagnosis of probable disorders is essential as part of a referral to a more appropriate health care provider. In addition, an understanding of any potential role or interaction of dental structures in the patient's pain complaint should be communicated as part of the referral.

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References

1. Abu-Bakra M, Jones NS. *Prevalence of nasal mucosal contact points in patients with facial pain compared with patients without facial pain*, J Laryngol Otol 2001;115: 629.
2. Albin R, Wiener J, Gordon R, et al: *Diagnosis and treatment of pansinusitis: report of case*, J Oral Surg 1979;37: 604.
3. Allerbring M, Haegerstam G. *Chronic idiopathic orofacial pain: a long-term follow-up study*, Acta Odontol Scand 2004;62: 66.
4. Alonso AA, Nixdorf DR. *Case series of four different headache types presenting as tooth pain*, J Endod 2006;32: 1110.
5. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-IV) 1994; American Psychiatric Association Washington, DC 447.
6. Anderson GC, John MT, Ohrbach R, et al: *Influence of headache frequency on clinical signs and symptoms of TMD in subjects with temple headache and TMD pain*, Pain 2011;152: 765.
7. Baad-Hansen L, Juhl GI, Jensen TS, et al: *Differential effect of intravenous S-ketamine and fentanyl on atypical odontalgia and capsaicin-evoked pain*, Pain 2007;129: 46.
8. Backonja M. *Use of anticonvulsants for treatment of neuropathic pain*, Neurology 5 suppl 2, 2002;59: S14.
9. Batchelder B, Krutchkoff DJ, Amara J. *Mandibular pain as the initial and sole clinical manifestation of coronary insufficiency*, J Am Dent Assoc 1987;115: 710.
10. Benoliel R, Eliav E, Tal M. *No sympathetic nerve*

Telegram: @dental_k

www.konkur.in

sprouting in rat trigeminal ganglion following painful and non-painful infraorbital nerve neuropathy, Neurosci Lett 2001;297: 151.

11. Benoliel R, Sharav Y. *Paroxysmal hemicrania: case studies and review of the literature*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 285.
12. Benoliel R, Sharav Y. *SUNCT syndrome: case report and literature review*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 158.
13. Besson J, Chaouch A. *Peripheral and spinal mechanisms of nociception*, Physiol Rev 1987;67: 67.
14. Bittar G, Graff-Radford SB. *A retrospective study of patients with cluster headaches*, Oral Surg Oral Med Oral Pathol 1992;73: 519.
15. Blows WT. *Diaphragmatic cramp as a possible cause of noncardiac chest pain and referred mandibular pain*, J Neurosci Nurs 1999;31: 187.
16. Bonica J. The management of pain with special emphasis on the use of analgesic block in diagnosis, prognosis and therapy 1953; Lea & Febiger Philadelphia.
17. Bonica J. The management of pain 1990; Lea & Febiger Philadelphia.
18. Brannstrom M, Johnson G, Nordenvall KJ. *Transmission and control of dentinal pain: resin impregnation for the desensitization of pain*, J Am Dent Assoc 1979;99: 612.
19. Brattberg J, Thorslund M, Wickman A. *The prevalence of pain in a general population. The results of a postal study in a county in Sweden*, Pain 1989;37: 215.
20. Brazis PW, Wharen RE, Czervionke LF, et al: *Hemangioma of the mandibular branch of the trigeminal nerve in the Meckel cave presenting with facial pain and sixth nerve palsy*, J Neurophthalmol 2000;20: 14.

Telegram: @dental_k

www.konkur.in

21. Brooke RI. *Periodic migrainous neuralgia: a cause of dental pain*, Oral Surg Oral Med Oral Pathol 1978;46: 511.
22. Broton J, Hu JW, Sessle BJ. *Effects of temporomandibular joint stimulation on nociceptive and nonnociceptive neurons of the cat's trigeminal subnucleus caudalis (medulla dorsal horn)*, J Neurophysiol 1988;59: 1575.
23. Brown A, Beeler WJ, Kloka AC, et al: *Spatial summation of pre-pain and pain in human teeth*, Pain 1985;21: 1.
24. Buddery DJ. *Mandible pain*, Br Dent J 2003;194: 121.
25. Byers M. *Dental sensory receptors*, Int Rev Neurobiol 1984;25: 39.
26. Campbell JK. *Facial pain due to migraine and cluster headache*, Semin Neurol 1988;8: 324.
27. Chan YW, Guo YC, Tsai TL, et al: *Malignant fibrous histiocytoma of the maxillary sinus presenting as toothache*, J Chin Med Assoc 2004;67: 104.
28. Chen YH, Tseng CC, Chao WY, et al: *Toothache with a multifactorial etiology: a case report*, Endod Dent Traumatol 1997;13: 245.
29. Cirak B, Kiymaz N, Arslanoglu A. *Trigeminal neuralgia caused by intracranial epidermoid tumor: report of a case and review of the different therapeutic modalities*, Pain Physician 2004;7: 129.
30. de Leeuw R. *Differential diagnosis of orofacial pain*, de Leeuw R Orofacial pain: guidelines for assessment, diagnosis, and management 2008; Quintessence Publishing Hanover Park, IL 49.
31. Delcanho RE, Graff-Radford SB. *Chronic paroxysmal hemicrania presenting as toothache*, J Orofac Pain 1993;7: 300.
32. DeNucci DJ, Chen CC, Sobiski C, et al: *The use of*

Telegram: @dental_k

www.konkur.in

SPECT bone scans to evaluate patients with idiopathic jaw pain, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 750.

33. Dewan K, Owens J, Silvester K. *Maintaining a high level of suspicion for recurrent malignant disease: report of a case with periapical involvement*, Int Endod J 2007;40: 900.
34. Dodick DW. *Migraine with isolated face pain: a diagnostic challenge*, Cephalalgia 2007;27: 1199.
35. Drangsholt MT, Svennson P, Nguyen KL: Which orofacial pain conditions are neuropathic? A systematic review using NEUPSIG '08 criteria, World Congress on Pain, International Association for the Study of Pain, August 2008.
36. Druce H. *Diagnosis of sinusitis in adults: History, physical examination, nasal cytology, echo, and rhinoscope*, J Allergy Clin Immunol 1992;90: 436.
37. Druce H, Slavin RG. *Sinusitis: critical need for further study*, J Allergy Clin Immunol 1991;88: 675.
38. Dubner R, Bennet G. *Spinal and trigeminal mechanisms of nociception*, Ann Rev Neurosci 1983;6: 381.
39. Dubner R, Hayes RL, Hoffman DS. *Neural and behavioral correlates of pain in the trigeminal system*, Res Publ Assoc Res Nerv Ment Dis 1980;58: 63.
40. Durham J, Exley C, John MT, et al: *Persistent dentoalveolar pain: the patient's experience*, J Orofac Pain 2013;27: 6.
41. Durso BC, Israel MS, Janini ME, et al: *Orofacial pain of cardiac origin: a case report*, Cranio 2003;21: 152.
42. Ekborn K. *Treatment of cluster headache: clinical trials, design and results*, Cephalalgia 1995;15: 33.
43. Ellinger RF, Kelly WH. *Maxillary sinus lymphoma: a*

Telegram: @dental_k

www.konkur.in

consideration in the diagnosis of odontogenic pain, J Endod 1989;15: 90.

44. Evans RW. Saunders manual of neurologic practice 2003; Elsevier Science Philadelphia.
45. Fricton J. *Masticatory myofascial pain: an explanatory model integrating clinical, epidemiological and basic science research*, Bull Group Int Research Sci Stomatol Odontol 1999;41: 14.
46. Fricton JR. *Critical commentary: A unified concept of idiopathic orofacial pain: clinical features*, J Orofac Pain 1999;13: 185.
47. Fristad I, Bardsen A, Knudsen GC, et al: *Prodromal herpes zoster: a diagnostic challenge in endodontics*, Int Endod J 2002;35: 1012.
48. Fromm GH, Graff-Radford SB, Terrence CF, et al: *Pre-trigeminal neuralgia*, Neurology 1990;40: 1493.
49. Gallagher R, Mueller L, Ciervo CA. *Increased prevalence of sensing types in men with cluster headaches*, Psychol Rev 2000;87: 555.
50. Gatot A, Peist M, Mozes M. *Endodontic overextension produced by injected thermopolasticized gutta-percha*, J Endod 1989;15: 273.
51. Gaul C, Gantenbein AR, Buettner UW, et al: *Orofacial cluster headache*, Cephalalgia 2008;28: 903.
52. Gaul C, Sandor PS, Galli U, et al: *Orofacial migraine*, Cephalalgia 2007;27: 950.
53. Gaver A, Polliack G, Pilo R, et al: *Orofacial pain and numb chin syndrome as the presenting symptoms of a metastatic prostate cancer*, J Postgrad Med 2002;48: 283.
54. Gayathri H, Madhan B. *Case report: malingering and factitious disorders*, B Dent J 2015;218: 610.
55. Giri S, Nixdorf D. *Sympathetically maintained pain*

Telegram: @dental_k

www.konkur.in

presenting first as temporomandibular disorder, then as parotid dysfunction, Tex Dent J 2007;124: 748.

56. Glaros A, Urban D, Locke J. *Headache and temporomandibular disorders: evidence for diagnostic and behavioural overlap*, Cephalalgia 2007;27: 542.
57. Goadsby PJ. *The international classification of headache disorders, ed 2*, Cephalalgia suppl 1, 2004;24: 1-106.
58. Gobel S, Falls W, Hockfield S: The division of the dorsal and ventral horns of the mammalian caudal medulla into eight layers using anatomic criteria. In Anderson D, Matthews B, editors: *Pain in the trigeminal region, proceedings of a symposium held in the Department of Physiology, University of Bristol, England, on July 25–27, 1977*, Amsterdam, 1977, Elsevier Press, p 443.
59. Goh B, Poon CY, Peck RH. *The importance of routine magnetic resonance imaging in trigeminal neuralgia diagnosis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 424.
60. Goldberg HL. *Chest cancer refers pain to face and jaw: a case review*, Cranio 1997;15: 167.
61. Goon WW, Jacobsen PL. *Prodromal odontalgia and multiple devitalized teeth caused by a herpes zoster infection of the trigeminal nerve: report of case*, J Am Dent Assoc 1988;116: 500.
62. Graff-Radford SB, Solberg WK. *Atypical odontalgia*, J Craniomandib Disord 1992;6: 260.
63. Graham LL, Schinbeckler GA. *Orofacial pain of cardiac origin*, J Am Dent Assoc 1982;104: 47.
64. Gratt BM, Graff-Radford SB, Shetty V, et al: *A 6-year clinical assessment of electronic facial thermography*, Dentomaxillofac Radiol 1996;25: 247.
65. Gratt BM, Pullinger A, Sickles EA, et al: *Electronic*

Telegram: @dental_k

www.konkur.in

thermography of normal facial structures: a pilot study, Oral Surg Oral Med Oral Pathol 1989;68: 346.

66. Greenberg DA, Aminoff MJ, Simon RP. Clinical neurology 2002; McGraw-Hill, p 390 New York.
67. Gregg J. *Neuropathic complications of mandibular implant surgery: review and case presentations*, Ann R Australas Coll Dent Surg 2000;15: 176.
68. Gregory WB Jr, Brooks LE, Penick EC. *Herpes zoster associated with pulpless teeth*, J Endod 1975;1: 32.
69. Guttenberg SA, Emery RW, Milobsky SA, et al: *Cranial arteritis mimicking odontogenic pain: report of case*, J Am Dent Assoc 1989;119: 621.
70. Hansen H. *Neuro-histological reactions following tooth extractions*, Int J Oral Surg 1980;9: 411.
71. Hargreaves K, Bowles WR, Jackson DL. *Intrinsic regulation of CGRP release by dental pulp sympathetic fibers*, J Dent Res 2003;82: 398.
72. Hargreaves K, Dubner R. Mechanisms of pain and analgesia 1991; Elsevier Press Amsterdam.
73. Heir G, Karolchek S, Kalladka M, et al: *Use of topical medication in orofacial neuropathic pain: a retrospective study*, Oral Surg Oral Med Oral Path Oral Radiol Endod 2008;105: 466.
74. Hellmann DB. *Temporal arteritis: a cough, toothache and tongue infarction*, JAMA 2002;287: 2996.

74a Hryvenko I, Cervantes AR, Law AS, et al: *Hemicrania continua: case series presenting in an orofacial pain clinic*, Cephalgia 2018;38: 1950.

75. Jacquin MF, Renehan WE, Mooney RD, et al: *Structure-function relationships in rat medullary and cervical dorsal horns. I. Trigeminal primary afferents*, J Neurophysiol 1986;55: 1153.

Telegram: @dental_k

www.konkur.in

76. Kisch B. *Horner's syndrome, an American discovery*, Bull Hist Med 1951;25: 284.
77. Kraut R, Chahal O. *Management of patients with trigeminal nerve injuries after mandibular implant placement*, J Am Dent Assoc 2002;133: 1351.
78. Kreiner M, Okeson JP, Michelis V, et al: *Craniofacial pain as the sole symptom of cardiac ischemia: a prospective multicenter study*, J Am Dent Assoc 2007;138: 74.
79. Lamotte R, Campbell JN. *Comparison of responses of warm and nociceptive C-fiber afferents in monkey with human judgements of thermal pain*, J Neurophysiol 1978;41: 509.
80. LeBars D, Dickenson AH, Besson JM, et al: *Aspects of sensory processing through convergent neurons*, Yaksh TL Spinal afferent processing 1986; Plenum Press New York.
81. Leon-Salazar V, Morrow L, Schiffman E. *Pain and persistent occlusal awareness: What should dentists do?*, J Am Dent Assoc 2012;143: 989.
82. Li Y, Li HJ, Huang J, et al: *Central malignant salivary gland tumors of the jaw: retrospective clinical analysis of 22 cases*, J Oral Maxillofac Surg 2008;66: 2247.
82a Linn J, Trantor I, Teo N, et al: *The differential diagnosis of toothache from other orofacial pains in clinical practice*, Aust Dent J 2007;52: S100.
83. Lipton J, Ship JA, Larach-Robinson D. *Estimated prevalence and distribution of reported orofacial pain in the United States*, J Am Dent Assoc 1993;124: 115.
84. Lipton RB, Stewart WF, Diamond S, et al: *Prevalence and burden of migraine in the United States: data from the American Migraine Study II*, Headache 2001;41: 646.

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www.konkur.in

85. List T, Leijon G, Helkimo M, et al: *Effect of local anesthesia on atypical odontalgia—a randomized controlled trial*, Pain 2006;122: 306.
86. List T, Leijon G, Helkimo M, et al: *Clinical findings and psychosocial factors in patients with atypical odontalgia: a case-control study*, J Orofac Pain 2007;21: 89.
87. List T, Leijon G, Svensson P. *Somatosensory abnormalities in atypical odontalgia: a case-control study*, Pain 2008;139: 333.
88. Loeser JD, Treede RD. *The Kyoto protocol of IASP basic pain terminology*, Pain 2008;137: 473.
89. Long A, Loeschr AR, Robinson PP. *A quantitative study on the myelinated fiber innervation of the periodontal ligament of cat canine teeth*, J Dent Res 1995;74: 1310.
90. Manfredini D, Winocur E, Ahlberg J, et al: *Psychosocial impairment in temporomandibular disorders patients. RDC/TMD axis II findings from a multicenter study*, J Dent 2010;38: 765.
91. Marbach J, Raphael KG. *Phantom tooth pain: a new look at an old dilemma*, Pain Med 2000;1: 68.
92. Melis M, Lobo SL, Ceneviz C, et al: *Atypical odontalgia: a review of the literature*, Headache 2003;43: 1060.
93. Moana-Filho EJ, Nixdorf DR, Bereiter DA, et al: *Evaluation of a magnetic resonance-compatible dentoalveolar tactile stimulus device*, BMC Neurosci 2010;11: 142.
94. Moncada E, Graff-Radford SB. *Cough headache presenting as a toothache: a case report*, Headache 1993;33: 240.
95. Moncada E, Graff-Radford SB. *Benign indomethacin-responsive headaches presenting in the orofacial region: eight case reports*, J Orofac Pain 1995;9: 276.

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96. Mondell B. *A review of the effects of almotriptan and other triptans on clinical trial outcomes that are meaningful to patients with migraine*, Clin Ther 2003;25: 331.
97. Morse D. *Infection-related mental and inferior alveolar nerve paresthesia: literature review and presentation of two cases*, J Endod 1997;23: 457.
98. Nahri M. *The neurophysiology of the teeth*, Dent Clin North Am 1990;34: 439.
99. Namazi MR. *Presentation of migraine as odontalgia*, Headache 2001;41: 420.
100. Natkin E, Harrington GW, Mandel MA. *Anginal pain referred to the teeth: report of a case*, Oral Surg Oral Med Oral Pathol 1975;40: 678.
101. Neumann S, Doubell TP, Leslie T, et al: *Inflammatory pain hypersensitivity mediated by phenotypic switch in myelinated primary sensory neurons*, Nature 1996;384: 360.
102. Nixdorf DR, Drangsholt MT, Ettlin DA, et al: *International RDC-TMD Consortium: Classifying orofacial pains: a new proposal of taxonomy based on ontology*, J Oral Rehabil 2012;39: 161.
102a Nixdorf DR, Law AS, Lindquist K, et al: *Frequency, impact, and predictors of persistent pain after root canal treatment: a national dental PBRN study*, Pain 2016;157: 159.
103. Nixdorf DR, Moana-Filho EJ, Law AS, et al: *Frequency of persistent tooth pain after root canal therapy: a systematic review and meta-analysis*, J Endod 2010;36: 224.
104. Nixdorf DR, Sobieh R, Gierthmuhlen J. *Using an n-of-1 trial to assist in clinical decision making for patients with*

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orofacial pain, J Am Dent Assoc 2012;143: 259.
105. Nixdorf DR, Velly AM, Alonso AA. *Neurovascular pains: implications of migraine for the oral and maxillofacial surgeon*, Oral Maxillofac Surg Clin North Am 2008;20: 221, vi.
106. Obermann M, Mueller D, Yoon MS, et al: *Migraine with isolated facial pain: a diagnostic challenge*, Cephalalgia 2007;27: 1278.
107. Ogutcen-Toller M, Uzun E, Incesu L. *Clinical and magnetic resonance imaging evaluation of facial pain*, Oral Surg Oral Med Oral Path Oral Radiol Endod 2004;97: 652.
108. Okeson J. Orofacial pain: guidelines for assessment, diagnosis and management 1996; Quintessence Publishing Chicago.
109. Okeson JP. Bell's orofacial pains: the clinical management of orofacial pain ed 6 2005; Quintessence Publishing Chicago.
110. Okeson JP, Falace DA. *Nonodontogenic toothache*, Dent Clin North Am 1997;41: 367.
111. Oshima K, Ishii T, Ogura Y, et al: *Clinical investigation of patients who develop neuropathic tooth pain after endodontic procedures*, J Endod 2009;35: 958.
111a Oyama O, Paltoo C, Greengold J. *Somatoform disorders*, Am Fam Physician 2007;76: 1333.
112. Padilla M, Clark GT, Merrill RL. *Topical medications for orofacial neuropathic pain: a review*, J Am Dent Assoc 2000;131: 184.
113. Pareja JA, Antonaci F, Vincent M. *The hemicrania continua diagnosis*, Cephalalgia 2001;21: 940.
114. Peszkowski M, Larsson AJ. *Extraosseous and intraosseous oral traumatic neuromas and their*

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association with tooth extraction, J Oral Maxillofac Surg 1990;48: 963.

115. Pigg M, List T, Petersson K, et al: *Diagnostic yield of conventional radiographic and cone-beam computed tomographic images in patients with atypical odontalgia*, Int Endod J 2011;44: 1092.
116. Pigg M, Svensson P, Drangsholt M, et al: *7-year follow-up of patients diagnosed with atypical odontalgia: a prospective study*, J Orofac Pain 2013;27: 151.
117. Pruckmayer M, Glaser C, Marosi C, et al: *Mandibular pain as the leading clinical symptom for metastatic disease: nine cases and review of the literature*, Ann Oncol 1998;9: 559.
118. Quail G. *Atypical facial pain: a diagnostic challenge*, Aust Fam Physician 2005;34: 641.
119. Raja S, Davis KD, Campbell JN. *The adrenergic pharmacology of sympathetically-maintained pain*, J Reconstr Microsurg 1992;8: 63.
120. Rasmussen BK, Jensen R, Schroll M, et al: *Epidemiology of headache in a general population–a prevalence study*, J Clin Epidemiol 1991;44: 1147.
121. Rosenfeld RM, Andes D, Bhattacharyya N, et al: *Clinical practice guideline: adult sinusitis*, Otolaryngol Head Neck Surg 2007;137(suppl): S1.
122. Roz TM, Schiffman LE, Schlossberg S. *Spontaneous dissection of the internal carotid artery manifesting as pain in an endodontically treated molar*, J Am Dent Assoc 2005;136: 1556.
123. Sarlani E, Schwartz AH, Greenspan JD, et al: *Chronic paroxysmal hemicrania: a case report and review of the literature*, J Orofac Pain 2003;17: 74.
124. Schaible HG. *Basic mechanisms of deep somatic tissue*,

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McMahon SB Koltzenburg M Textbook of pain 2006; Elsevier Philadelphia 621.

125. Schiffman E, Ohrbach R, List T, et al: *Diagnostic criteria for headache attributed to temporomandibular disorders*, Cephalalgia 2012;32(9): 683.

126. Schiffman E, Ohrbach R, Truelove E, et al: *Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: Recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group*, J Oral Facial Pain Headache 2014;28: 6.

127. Schwartz BS, Stewart WF, Simon D, et al: *Epidemiology of tension-type headache*, JAMA 1998;279: 381.

128. Selden HS, Manhoff DT, Hatges NA, et al: *Metastatic carcinoma to the mandible that mimicked pulpal/periodontal disease*, J Endod 1998;24: 267.

129. Setzen G, Ferguson BJ, Han JK, et al: *Clinical consensus statement: appropriate use of computed tomography for paranasal sinus disease*, Otolaryngol Head Neck Surg 2012;147(5): 808.

129a Shueb SS, Boyer HC, Nixdorf DR. *Nonodontogenic "tooth pain" of nose and sinus origin*, J Am Dent Assoc 2016;147: 457.

130. Sicuteri F, Nicolodi M, Fusco BM, et al: *Idiopathic pain as a possible risk factor for phantom tooth pain*, Headache 1991;31: 577.

131. Stewart W, Ricci JA, Chee E, et al: *Lost productive time and cost due to common pain conditions in the US workforce*, JAMA 2003;290: 2443.

132. Stewart WF, Lipton RB, Celentano DD, et al: *Prevalence of migraine headache in the United States: relation to age, income, race, and other sociodemographic factors*, JAMA

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1992;267: 64.
133. Svennson P. *Muscle pain in the head: overlap between temporomandibular disorders and tension-type headaches*, Curr Opin Neurol 2007;20: 320.
134. Takenoshita M, Sato T, Kato Y, et al: *Psychiatric diagnoses in patients with burning mouth syndrome and atypical odontalgia referred from psychiatric to dental facilities*, Neuropsychiatr Dis Treat 2010;6: 699.
135. Tepper SJ, Dahlof CG, Dowson A, et al: *Prevalence and diagnosis of migraine in patients consulting their physician with a complaint of headache: data from the landmark study*, Headache 2004;44: 856.
136. Thomas G, Pandey M, Mathew A, et al: *Primary intraosseous carcinoma of the jaw: pooled analysis of world literature and report of two new cases*, Int J Oral Maxillofac Surg 2001;30: 349.
137. Torebjork H, Lundberg LE, LaMotte RH. *Pain, hyperalgesia and activity in nociceptive C units in humans after intradermal injection of capsaicin*, J Physiol 1992;448: 749.
138. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual 1983; Williams & Wilkins, p 1 Baltimore.
139. Treede RD, Jensen TS, Campbell JN, et al: *Neuropathic pain: redefinition and a grading system for clinical and research purposes*, Neurology 2008;70: 1630.
140. Tzukert A, Hasin Y, Sharav Y. *Orofacial pain of cardiac origin*, Oral Surg Oral Med Oral Pathol 1981;51: 484.
141. Uehara M, Tobita T, Inokuchi T. *A case report: toothache caused by epidermoid cyst manifested in cerebellopontine angle*, J Oral Maxillofac Surg 2007;65: 560.

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141a Vena DA, Collie D, Wu H, et al: *Prevalence of persistent pain 3 to 5 years post primary root canal therapy and its impact on oral health–related quality of life: PEARL Network findings*, J Endod 2014;40: 1917.

142. Webb DJ, Colman MF, Thompson K, et al: *Acute, life-threatening disease first appearing as odontogenic pain*, J Am Dent Assoc 1984;109: 936.

143. Wertheimer-Hatch L, Hatch GF 3rd, Hatch KF, et al: *Tumors of the oral cavity and pharynx*, World J Surg 2000;24: 395.

144. Wood M, Johnson RW, McKendrick MW, et al: *A randomized trial of acyclovir for 7 days or 21 days with and without prednisolone for treatment of acute herpes*, N Engl J Med 1994;330: 896.

145. Woolf C. *Phenotype modification of primary sensory neurons: the role of nerve growth factor in the production of persistent pain*, Philos Trans R Soc Lond B Biol Sci 1996;351: 441.

146. Wright EF. *Referred craniofacial pain patterns in patients with temporomandibular disorder*, J Am Dent Assoc 2000;131: 1307.

147. Yang J, Simonson TM, Ruprecht A, et al: *Magnetic resonance imaging used to assess patients with trigeminal neuralgia*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 343.

148. Yokota T. Neural mechanisms of trigeminal pain 1985; McGraw-Hill New York.

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5: Case assessment and treatment planning

Paul A. Rosenberg, Matthew Malek

CHAPTER OUTLINE

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The process of case selection and treatment planning begins after a clinician has diagnosed an endodontic problem. The clinician must determine whether the patient's oral health needs are best met by providing endodontic treatment and maintaining the tooth or by advising extraction. The use of cone beam computed technology (CBCT), rotary instruments, ultrasonics, and microscopy, as well as new materials, has made it possible to retain teeth that previously would have been extracted. In addition, even teeth that have failed initial endodontic treatment can often be successfully retreated using nonsurgical or surgical procedures.

Increased knowledge of factors involved in intraoperative and postoperative pain have led to new pain preventive strategies. For example, recognition of the importance of anxiety control, premedication with a nonsteroidal antiinflammatory drug (NSAID) or acetaminophen, profound local anesthesia, and appropriate occlusal adjustment enables clinicians to complete endodontic procedures without intraoperative or posttreatment pain.

The scope of endodontics has changed; the clinician now has more viable options than ever before. In recent years, there is increasing evidence to support the implementation of regenerative procedures for some immature teeth, vital pulp therapy, and replantation of teeth with failed non-surgical or surgical endodontic procedures. Treatment planning must now include those options as well as nonsurgical or surgical endodontics.

Questions concerning tooth retention and possible referral can only be answered after a complete patient evaluation. The evaluation must include assessment of medical, psychosocial, and dental factors as well as

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consideration of the relative complexity of the endodontic procedure. Although most medical conditions do not contraindicate endodontic treatment, some can influence the course of treatment and require specific modifications. Comprehensive texts are available that review the subject of dental care for the medically compromised patient.[15,51,118] The American Academy of Oral Medicine (Edmonds, WA) has an excellent website (www.aaom.com) that can be used to elicit information about medically compromised patients.

The American Dental Society of Anesthesiology (ADSA) has produced a valuable source of information, “Ten Minutes Saves a Life” application, which is available for most common smartphone platforms. Its goal is to optimize patient safety and outcomes during in-office medical emergencies (see https://www.adsa-arf.org/tenminutes).

Perhaps the most important advice for a clinician who plans to treat a medically compromised patient is to be prepared to communicate with the patient’s physician. The proposed treatment can be reviewed, and medical recommendations should be documented. Fig. 5.1 depicts a sample medical consultation letter that can be modified as necessary.

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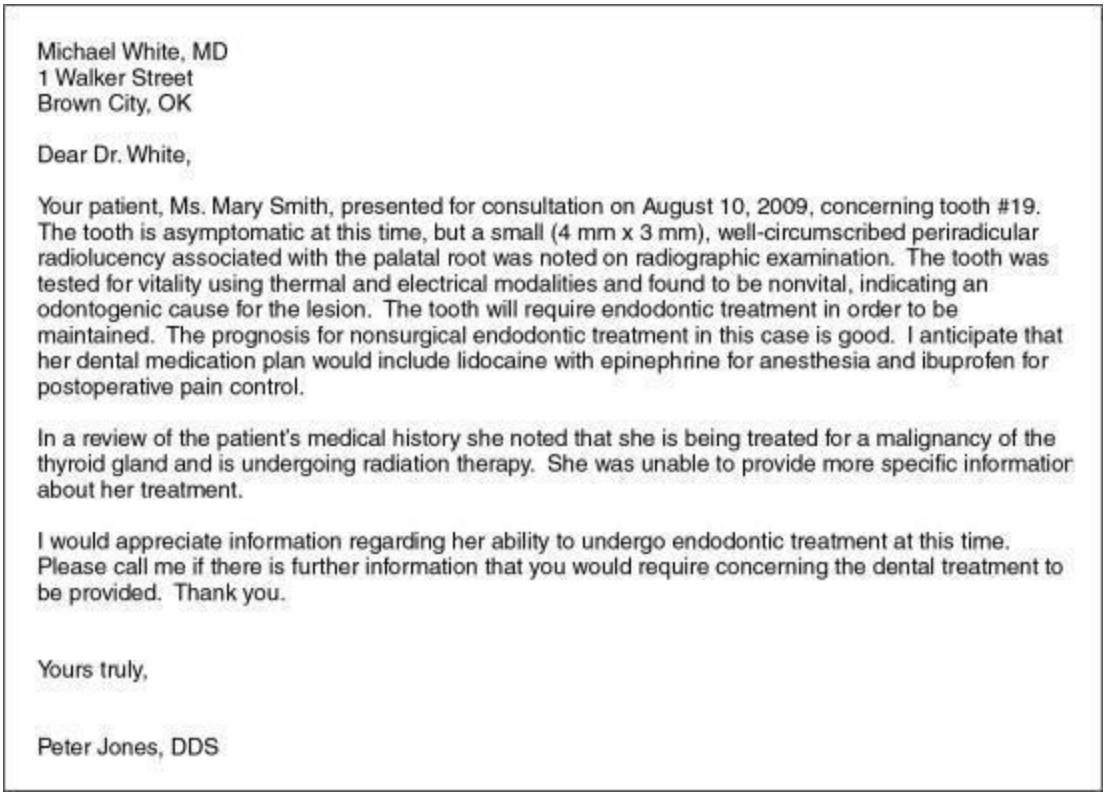

Michael White, MD
1 Walker Street
Brown City, OK

Dear Dr. White,

Your patient, Ms. Mary Smith, presented for consultation on August 10, 2009, concerning tooth #19. The tooth is asymptomatic at this time, but a small (4 mm x 3 mm), well-circumscribed periradicular radiolucency associated with the palatal root was noted on radiographic examination. The tooth was tested for vitality using thermal and electrical modalities and found to be nonvital, indicating an odontogenic cause for the lesion. The tooth will require endodontic treatment in order to be maintained. The prognosis for nonsurgical endodontic treatment in this case is good. I anticipate that her dental medication plan would include lidocaine with epinephrine for anesthesia and ibuprofen for postoperative pain control.

In a review of the patient's medical history she noted that she is being treated for a malignancy of the thyroid gland and is undergoing radiation therapy. She was unable to provide more specific informatior about her treatment.

I would appreciate information regarding her ability to undergo endodontic treatment at this time. Please call me if there is further information that you would require concerning the dental treatment to be provided. Thank you.

Yours truly,

Peter Jones, DDS

FIG. 5.1 Sample medical consultation letter.

Sample of medical consultation letter is as follows:

Michael White, MD

1 Walker Street

Brown City, OK

Dear Dr. White,

Your patient, Ms. Mary Smith, presented for consultation on August 10, 2009, concerning tooth #19. The tooth is asymptomatic at this time, but a small (4 mm x 3 mm), well-circumscribed periradicular radiolucency associated with the palatal root was noted on radiographic examination. The tooth was tested for vitality using thermal and electrical modalities and found to be nonvital, indicating an odontogenic cause for the lesion. The tooth will require endodontic treatment in order to be maintained. The prognosis for nonsurgical endodontic treatment in this case is good. I anticipate that her dental medication plan would include lidocaine with epinephrine for anesthesia and ibuprofen for postoperative pain control.

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I would appreciate information regarding her ability to undergo endodontic treatment at this time. Please call me if there is further information that you would require concerning the dental treatment to be provided. Thank you.

Yours truly,

Peter Jones, DDS

The American Society of Anesthesiologists (ASA; Park Ridge, IL) Physical Status Classification system is commonly used to express medical risk (Box 5.1). The ASA classification system remains the most widely used assessment method for preanesthetic patients, despite some inherent limitations to its use as a peritreatment risk predictor. This classification system is a generally accepted useful guide for pretreatment assessment of relative risk but does not advise appropriate treatment modifications. The clinician should go beyond the classification system and gather more information from the patient and physician, including the patient's compliance with suggested medication, frequency of physician visits, and most recent visit.

Box 5.1

American Society of Anesthesiologists Physical Status Classification System

ASA I: A normal healthy patient healthy, non-smoking, no or minimal alcohol use.

ASA II: A patient with mild systemic disease, mild diseases only without substantive functional limitations. Examples include (but not limited to): current smoker, social alcohol drinker, pregnancy, obesity (30 < BMI < 40), well-controlled DM/HTN, mild lung disease.

ASA III: A patient with severe systemic disease, substantive functional limitations; one or more moderate to severe diseases. Examples include (but not limited to): poorly controlled DM or HTN, COPD, morbid obesity (BMI ≥40), etc.

ASA IV: A patient with severe systemic disease that is a constant threat to life. Examples include (but not limited to): recent (<3 months) MI, CVA, TIA, or CAD/stents, etc.

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ASA V: A moribund patient who is not expected to survive without the operation.
ASA VI: A declared brain-dead patient whose organs are being removed for donor purposes.

ASA, American Society of Anesthesiologists; *BMI*, body mass index; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *CVA*, cerebrovascular accident; *DM*, diabetes mellitus; *HTN*, hypertension; *MI*, myocardial infarction; *TIA*, transient ischemic attack.
Excerpted from ASA Physical Status Classification System 2019 of the American Society of Anesthesiologists. A copy of the full text can be obtained from ASA, 1061 American Lane, Schaumburg, IL, 60173-4973 or online at www.asahq.org.

Typical questions include the following: *Do you take medication as prescribed by your physician?* Or, *when was the last time you were examined by your physician?* Other systems have been proposed that would better reflect the increasing number of medically complex patients treated by clinicians as Americans live longer.[35] Regardless of the classification system used, these generalized guidelines need to be individualized for the patient under care.

An alternative means of considering risk assessment is to review the following issues:

- History of allergies
- History of drug interactions, adverse effects
- Presence of prosthetic valves, joints, stents, pacemakers, and so on
- Antibiotics required (prophylactic or therapeutic)
- Hemostasis (normal expected, modification to treatment)
- Patient position in chair
- Infiltration or block anesthesia with or without vasoconstrictor
- Significant equipment concerns (radiographs, ultrasonics, electrosurgery)
- Emergencies (potential for occurrence, preparedness)
- Anxiety (past experiences and management strategy)

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A review of these areas provides the clinician with essential background data before initiating treatment.

Common medical findings that may influence endodontic treatment planning

Cardiovascular disease

Patients with some forms of cardiovascular disease are vulnerable to physical or emotional stress that may be encountered during dental treatment, including endodontics. Patients may be confused or not understand the specifics of their particular cardiovascular problem.

In those situations, consultation with the patient's physician is mandatory before initiation of endodontic treatment. "For patients with symptoms of unstable angina or those who have had an MI (myocardial infarction) within the past 30 days (major risk category), elective treatment should be postponed."[51] However, one study found "no significant increase in the risk of experiencing a second vascular event after dental visits, including those that involved invasive procedures, in periods up to 180 days after a first recorded ischemic stroke, transient ischemic attack (TIA) or acute MI."[101]

The use of vasoconstrictors in local anesthetics poses potential problems for patients with ischemic heart disease. In these patients, local anesthetics without vasoconstrictors may be used as needed. If a vasoconstrictor is necessary, patients with intermediate clinical risk factors (i.e., a past history of MI without ischemic symptoms) and those taking nonselective beta-blockers can safely be given up to 0.036 mg epinephrine (two cartridges containing 1:100,000 epinephrine) at a single appointment. For patients at higher risk (i.e., those who have had an MI within the past 7 to 30 days and unstable angina), the use of vasoconstrictors should be discussed with the physician.[51]

Vasoconstrictors may interact with some antihypertensive medications and should be used only after consultation with the at-risk patient's physician. Local anesthetic agents with minimal or no vasoconstrictors are usually adequate for nonsurgical endodontic procedures (see also Chapter 6 on pain control).[51] A systematic review of the cardiovascular effects of epinephrine

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concluded that the increased risk for adverse events among uncontrolled hypertensive patients was low, and the reported adverse events associated with epinephrine use in local anesthetics was minimal.[6] Another review highlighted the advantages of including a vasoconstrictor in the local anesthesia and stated that "pain control was significantly impaired in those patients receiving the local anesthetic without the vasoconstrictor as compared to those patients receiving the local anesthetic with vasoconstrictor."[14]

Some heart conditions make a patient more susceptible to an infection of the heart valves, induced by a bacteremia. That infection, called *infective* or *bacterial endocarditis,* is potentially fatal. In 2008, the American College of Cardiology and American Heart Association (AHA) Task Force on Practice Guidelines published an update on their previous guidelines, which focused on infectious endocarditis. This guideline stated that prophylaxis against infective endocarditis is reasonable for the following patients at highest risk for adverse outcomes from infective endocarditis who undergo dental procedures that involve manipulation of either gingival tissue or the periapical region of teeth or perforation of the oral mucosa: patients with prosthetic cardiac valves or prosthetic material used for cardiac valve repair, patients with previous infective endocarditis, and patients with select congenital heart diseases.[73]

The specific recommendations are summarized in an American Association of Endodontists (AAE) (Chicago, IL) reference guide found online at https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/aae_antibiotic-prophylaxis-2017update.pdf. Because the AHA periodically revises its recommended antibiotic prophylactic regimen for dental procedures, it is essential that the clinician stay current concerning this important issue.

There is a low compliance rate among at-risk patients regarding their use of the suggested antibiotic coverage before dental procedures. Therefore, the clinician must question patients concerning their compliance with the prescribed prophylactic antibiotic coverage before endodontic therapy. If a patient has not taken the antibiotic as recommended, it may be administered up to 2 hours after the procedure.[51]

Patients with artificial heart valves are considered susceptible to bacterial

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endocarditis. Consulting the patient's physician in such cases regarding antibiotic premedication is essential. Some physicians elect to administer parenteral antibiotics in addition to or in place of the oral regimen.

A dentist may be the first to detect elevated blood pressure if he or she routinely evaluates blood pressure before treatment. Furthermore, patients receiving treatment for hypertension may not be controlled adequately because of poor compliance or inappropriate drug therapy. Abnormal blood pressure readings may be the basis for physician referral.

There is a widespread belief among dentists and physicians that oral anticoagulation therapy with drugs such as warfarin (Coumadin, Bristol-Myers Squibb, New York, NY, USA) must be discontinued before dental treatment to prevent serious hemorrhagic complications, especially during and after surgical procedures. Aspirin is a drug commonly used as an anticoagulant on a daily basis without the supervision of a physician. However, clinical studies do not support the routine withdrawal of anticoagulant therapy before dental treatment for patients who are taking such medications.[12,41,51]

When patients report they are receiving an anticoagulant medication, they can benefit from the clinician using the following guidelines:

- Identify the reason why the patient is receiving anticoagulant therapy.
- Know the drug classification, mechanism of action, and its half-life. For instance, 1 to 2 hours for standard heparin, 2 to 4 hours for heparin with low-molecular-weight preparations, and 4 to 5 days for warfarin.[51]
- Assess the potential risk versus benefit of altering the drug regimen.
- Know the laboratory tests used to assess anticoagulation levels. For example, the international normalized ratio (INR) value should be 3.5 or less for patients who are taking warfarin to safely undergo dental or surgical endodontic procedures. However, newer-generation direct thrombin inhibitors, such as dabigatran (Pradaxa) (Pradaxa Boehringer Ingelheim Pharmaceuticals, Ingelheim, Germany), do not require INR monitoring.[51]
- Be familiar with methods used to obtain hemostasis both

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intraoperatively and postoperatively.
- Be familiar with the potential complications associated with prolonged or uncontrolled bleeding.
- Consult the patient's physician to discuss the proposed dental treatment and to determine the need to alter the anticoagulant regimen.

Diabetes

The Centers for Disease Control and Prevention (CDC, Atlanta, GA) in 2017 reported that an estimated 30.3 million people of all ages, or 9.4% of the US population, had diabetes in 2016. (See National Diabetes Statistics Report available at https://www.cdc.gov/diabetes/pdfs/data/statistics/national-diabetes-statistics-report.pdf.) Diabetes was the seventh leading cause of death in the United States in 2016 (heart disease being the first).[119]

Diabetes mellitus appears to have multiple causes and several mechanisms of pathophysiology.[51] It can be thought of as a combination of diseases that share the key clinical feature of glucose intolerance. Patients with diabetes, even those who are well controlled, require special consideration during endodontic treatment. The patient with well-controlled diabetes who is free of serious complications such as renal disease, hypertension, or coronary atherosclerotic disease is a candidate for endodontic treatment. However, special considerations exist in the presence of acute infections. The non–insulin-controlled patient may require insulin, or the insulin dose of some insulin-dependent patients may have to be increased.[83] When surgery is required, consultation with the patient's physician is advisable in order to consider adjustment of the patient's insulin dosage, antibiotic prophylaxis, and dietary needs during the posttreatment period.

The clinician should ask patients with diabetes, who self-monitor their glucose levels, to bring a glucometer to each visit. If pretreatment glucose levels are below normal fasting range (80 to 120 mg/dL), it may be appropriate to take in a carbohydrate source.[5] A source of glucose (e.g., glucose tablets, orange juice, or soda) should be available if signs of insulin shock (hypoglycemic reaction caused by over-control of glucose levels) occur.[51] Signs and symptoms of hypoglycemia include confusion, tremors, agitation, diaphoresis, and tachycardia.[5] The clinician can avoid a

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hypoglycemic emergency by taking a complete, accurate history of the time and amount of the patient's insulin and meals. When questions arise concerning the appropriate course to follow, the patient's physician should be contacted or treatment deferred.

Appointments should be scheduled with consideration given to the patient's normal meal and insulin schedule.[83] Usually, a patient with diabetes who is well managed medically and is under good glycemic control without serious complications such as renal disease, hypertension, or coronary atherosclerotic heart disease can receive any indicated dental treatment.[60] However, patients with diabetes who have serious medical complications may need a modified dental treatment plan. For instance, although prophylactic antibiotics are generally not required, it "may be prescribed for a patient with brittle (very difficult to control) diabetes, for whom an invasive procedure is planned but whose oral health is poor and the fasting plasma glucose exceeds 200 mg/dL."[51] Local anesthesia would not be an issue in the presence of well-controlled diabetes, "but for patients with concurrent hypertension or history of recent myocardial infarction, or with a cardiac arrhythmia, the dose of epinephrine should be limited to no more than two cartridges containing 1:100,000 epinephrine."[51]

There is some evidence in support of a relationship between glycemic control and periapical inflammation in diabetic patients.[8] A literature review of human studies also indicated a positive association between diabetes and larger number of periapical lesions.[16] One study determined that although apical periodontitis may be significantly more prevalent in untreated teeth in patients with type 2 diabetes, the disease does not seem to influence the response to root canal treatment.[55] However, other studies suggest that diabetes is associated with a decrease in the success of endodontic treatment in cases with pretreatment periradicular lesions.[30,105] In a prospective study on the impact of systemic diseases on the risk of tooth extraction, it has been also shown that an increased risk of tooth extraction after nonsurgical root canal treatment was significantly associated with diabetes mellitus, hypertension, and coronary heart disease.[113] The nature of such an association has not been fully explained, and may not be a causal relationship.

It is suggested that clinicians discuss the possible negative effect of

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diabetes on the outcome of endodontic treatment with appropriate patients. Patients with diabetes and other systemic diseases may be best served by referral to an endodontist for treatment planning.

Pregnancy

Although pregnancy is not a contraindication to endodontics, it may require modification of treatment planning. Protection of the fetus is a primary concern when administration of ionizing radiation or drugs is considered. The most important of safety devices associated with dental radiography is the protective lead apron with thyroid collar. Also important are the use of high-speed film, digital imaging, filtration, and collimation.[93] The American College of Obstetricians and Gynecologists' "Committee Opinion" published in 2013 and reaffirmed in 2017 recommends that clinicians reassure patients that "prevention, diagnosis, and treatment of oral conditions, including dental x-rays (with shielding of the abdomen and thyroid) and local anesthesia (lidocaine with or without epinephrine), are safe during pregnancy" (this document can be accessed at: https://www.acog.org/-/media/project/acog/acogorg/clinical/files/committee-opinion/articles/2013/08/oral-health-care-during-pregnancy-and-through-the-lifespan.pdf). The US Food and Drug Administration also states that "the risk of not having a needed x-ray could be much greater than the risk from the radiation" (refer to https://www.fda.gov/Radiation-Emitting Products/RadiationEmittingProductsandProcedures/MedicalImaging/MedicalX Rays/ucm142632.htm).

Over the last three decades, the use of prescription medication in the first trimester has increased by over 60%, and use of four or more medications more than tripled. By the year 2008, approximately 50% of women reported taking at least 1 medication during pregnancy.[61] These data suggest the increasing need for both the patient and the clinician to be informed about the indications, contraindications, and safety of consuming drugs during pregnancy. Box 5.2 presents commonly used dental drugs usually compatible with both pregnancy and breast-feeding. It should be noted that most clinicians are used to the old five-alphabet pregnancy categorization of the drugs (A, B, C, D, and X). In 2014, the US Food and Drug Administration changed its Pregnancy and Lactation Labeling. The new labels include "a

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summary of the risks of using the drug during pregnancy and lactation, a discussion of the data supporting that summary, and relevant information to help health care providers make prescription decisions and counsel women about the use of drugs during pregnancy and lactation" (see https://www.fda.gov/media/100406/download).

Box 5.2

Partial List of Drugs Usually Compatible With Both Pregnancy and Breast-Feeding

- Local anesthetics including lidocaine, etidocaine, and prilocaine
- Many antibiotics including penicillins, clindamycin, and azithromycin
- Acetaminophen
- Acyclovir
- Prednisone
- Antifungals including fluconazole and nystatin

One of the areas of concern in treating pregnant patients is to select the safest type of analgesics.

The analgesic of choice during pregnancy had been acetaminophen.[82] However, a link between acetaminophen and childhood asthma has been suggested. Research has found that "the use of acetaminophen in middle to late but not early pregnancy may be related to respiratory symptoms in the first year of life."[78] This finding, although not completely validated, and questioned due to possible confounding factors,[4] should be discussed with pregnant patients when an analgesic is being considered. In a 2016 advisory, the National Health Service of Great Britain advised pregnant patients not to take ibuprofen from the 30th week of pregnancy onwards. They stated that at that stage of pregnancy there is an association with increased risks of complications including heart problems. They went on to advise that before 30 weeks of pregnancy, patients should avoid taking ibuprofen, as it may lead to increased risks of complications including miscarriage (see https://www.nhs.uk/common-health-questions/pregnancy/can-i-take-ibuprofen-when-i-am-pregnant/). Aspirin and NSAIDs also convey risks for

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constriction of the ductus arteriosus, as well as for postpartum hemorrhage and delayed labor.[82] In a recent large epidemiology study done on 29,078 case and 10,962 control mothers from 1997 to 2011, the authors found that "compared to periconceptional use of acetaminophen, selected birth defects occurred more frequently among infants of women using NSAIDs and/or opioids." However, the authors were not able to definitely determine whether these risks were related to the drugs or to indications for treatment.[37] Due to ongoing research in this area, the clinician is strongly advised to routinely review current information concerning the use of medications before prescribing them for pregnant patients. Other than the CDC, one of the resources for both the clinician and the patient is the following website, a service of the nonprofit Organization of Teratology Information Specialists (OTIS), which is an excellent resource for evidence-based information on the safety of medications and other exposures during pregnancy and while breast-feeding: https://mothertobaby.org.

Based on US Food and Drug Administration pregnancy risk factor definitions,[35] local anesthetics administered with epinephrine generally are considered safe for use during pregnancy. Lidocaine is one of the safest local anesthetics, therefore delaying dental care during pregnancy is not advisable; however, the dose and type of the local anesthetic must be carefully determined for women who have contracted medical conditions that can induce serious pregnancy-related complications.[43] Few anxiolytics are considered safe to use during pregnancy. However, a single, short-term exposure to nitrous oxide–oxygen (N_2O-O_2) for less than 35 minutes is not thought to be associated with any human fetal anomalies, including low birth weight.[51] If a need exists for antibiotic therapy, penicillins, cephalosporins, and macrolides are considered first-line agents.

A major concern is that a drug may cross the placenta and be toxic or teratogenic to the fetus. In addition, any drug that is a respiratory depressant can cause maternal hypoxia, resulting in fetal hypoxia, injury, or death. Ideally, no drug should be administered during pregnancy, especially during the first trimester. If a specific situation makes adherence to this rule difficult, then the clinician should review the appropriate current literature and discuss the case with the physician and patient.[13,58,74]

Further considerations exist during the postpartum period if the mother

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breast-feeds her infant. A clinician should consult the responsible physician before using any medications for the nursing mother. Alternative considerations include using minimal dosages of drugs, having the mother bank her milk before treatment, having her feed the child before treatment, or suggesting the use of a formula for the infant until the drug regimen is completed. Limited data are available on drug dosages and the effects on breast milk.[51]

In terms of treatment planning, elective dental care is best avoided during the first trimester because of the potential vulnerability of the fetus. The second trimester is the safest period in which to provide routine dental care. Complex surgical procedures are best postponed until after delivery.

Malignancy

Some malignancies may metastasize to the jaws and mimic endodontic pathosis, whereas others can be primary lesions (Fig. 5.2). The most common malignancies metastasize to the jaws are breast, lung, thyroid, and prostate.[56] A panoramic radiograph and a cone-beam computed tomographic image are useful in providing an overall view of all dental structures. When a clinician begins an endodontic procedure on a tooth with a well-defined apical radiolucency, it might be assumed to result from a nonvital pulp. However, pulp sensibility testing is essential to confirm a lack of pulp vitality. A vital response in such cases indicates a nonodontogenic lesion.

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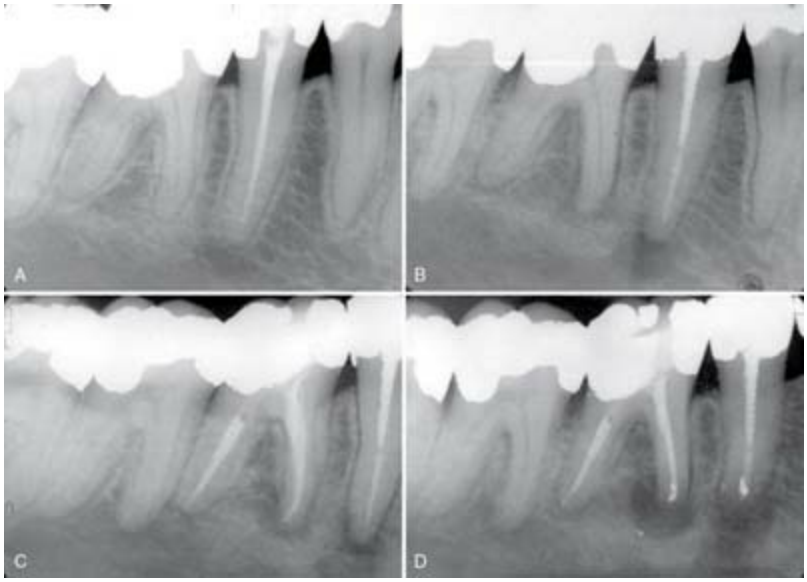

FIG. 5.2 **A,** Periapical view of tooth #29 after endodontic treatment by a general dental clinician. The diagnosis was irreversible pulpitis. **B,** Patient was referred to an endodontist 4 months later to evaluate radiolucencies of teeth #29 and #30. Symptoms indicated irreversible pulpitis of tooth #30, with concurrent lower right lip and chin paresthesia. Past medical history revealed breast cancer in remission. **C,** Nonsurgical endodontics was performed on tooth #30. Immediate referral was made to an oncologist/oral surgeon for biopsy to rule out nonodontogenic origin of symptoms. **D,** Surgical posttreatment radiograph of teeth #29 and #30. The biopsy report confirmed metastatic breast cancer.

A) Radiograph shows low-density tissue between teeth. 29th tooth has filled root canal with a porous tissue next to the root tip.

B) Radiograph shows a cleavage at the mid-way of root canal in 29th tooth. The region near root tip shows a dark circular patch.

C) Radiograph shows molar tooth with filled root canals. Root is surrounded by trabecular tissues.

D) Radiograph shows a well-defined circular patch at root apex of the molar.

Source: (Courtesy Dr. R. Sadowsky, Dr. L. Adamo, and Dr. J. Burkes.)

Careful examination of pretreatment radiographs from different angulations is important because lesions of endodontic origin would not be shifted away from the radiographic apex in the various images. Alternative methods, such as CBCT, may provide important diagnostic information (see

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Chapter 2 on radiographic interpretation).

A definitive diagnosis of periradicular lesions can be made only after biopsy. When a discrepancy exists between the initial diagnosis and clinical findings, consultation with an endodontist is advisable.

Patients undergoing chemotherapy or radiation to the head and neck may have impaired healing responses.[51] Treatment should be initiated only after the patient's physician has been consulted. A dialogue among the dentist, physician, and patient is required prior to determining whether a tooth or teeth should be extracted or endodontically treated prior to radiation.

The effect of the external beam of radiation therapy on normal bone is to decrease the number of osteocytes, osteoblasts, and endothelial cells, thus decreasing blood flow. Pulps may become necrotic from this impaired condition.[51] Toxic reactions during and after radiation and chemotherapy are directly proportional to the amount of radiation or dosage of cytotoxic drug to which the tissues are exposed. Delayed toxicities can occur several months to years after radiation therapy.

Oral infections and any potential problems should be addressed before initiating radiation. It is advised that symptomatic nonvital teeth be endodontically treated at least 1 week before initiating radiation or chemotherapy, whereas treatment of asymptomatic nonvital teeth may be delayed.[51] The outcome of endodontic treatment should be evaluated within the framework of the toxic results of radiation and drug therapy. The white blood cell (WBC) count and platelet status of a patient undergoing chemotherapy should also be reviewed before endodontic treatment. In general, routine dental procedures can be performed if the granulocyte count is greater than 2000/mm^3 and the platelet count is greater than 50,000/mm^3. If urgent care is needed and the platelet count is below 50,000/mm^3, consultation with the patient's physician is required.[51]

Medication-related osteonecrosis of the jaws

Bisphosphonates offer great benefits to patients at risk of bone metastases and in the prevention and treatment of osteoporosis, although this and other drugs (e.g., denosumab) are associated with a rare occurrence of osteonecrosis.[90]

To distinguish medication-related osteonecrosis of the jaws (MRONJ)

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from other delayed healing conditions, the following working definition of MRONJ has been adopted by the American Association of Oral and Maxillofacial Surgeons (AAOMS): Patients may be considered to have MRONJ if all of the following three characteristics are present[90]:

1. Current or previous treatment with an antiresorptive drug such as a bisphosphonate or an antiangiogenic drug such as sunitinib (Sutent) (Pfizer, New York, NY, USA), sorafenib (Nexavar) (Bayer, Leverkusen, Germany), or sirolimus (Rapamune) (Pfizer, New York, NY, USA).
2. Exposed, necrotic bone, or bone that can be probed through an intraoral or extraoral fistula in the maxillofacial region that has persisted for more than 8 weeks.
3. No history of radiation therapy to the jaws.

A patient's risk of developing osteonecrosis of the jaw while receiving oral bisphosphonates appears to be low, but there are factors known to increase the risk for MRONJ (Box 5.3). According to aforementioned position statements of the AAOMS, such risks include a history of taking intravenous (IV) bisphosphonates, as opposed to intraoral (IO), taking bisphosphonates for treatment of cancer (high risk), corticosteroid therapy, history of tobacco use, duration of bisphosphonate therapy (>2 to 3 years), a history of an oral operative procedure (i.e., dentoalveolar surgery and extraction), anatomical factors (mandible higher risk than maxilla), concomitant oral diseases (i.e., periodontal disease or periapical pathology), gender (female), age, and genetic predispositions.

Box 5.3

Risk Factors for Development of Bisphosphonate-Associated Osteonecrosis

- History of taking bisphosphonates for more than 2–3 years, especially with intravenous therapy
- History of cancer, osteoporosis, or Paget disease
- History of traumatic dental procedure

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- Patient more than 65 years of age
- History of periodontitis
- History of chronic corticosteroid use
- History of smoking
- History of diabetes

Treatment outcomes of MRONJ are unpredictable, and prevention strategies are extremely important. Management of high-risk patients might include nonsurgical endodontic treatment of teeth that otherwise would be extracted. The combination of orthodontic extrusion and bloodless extraction —exfoliation of the extruded roots after their movement—has also been suggested with the aim of minimizing trauma and enhancing the health of the surrounding tissues in patients at risk of developing MRONJ or when a patient refuses to undergo conventional tooth extraction.[102]

For patients at higher risk of MRONJ, surgical procedures such as extractions, endodontic surgery, or placement of dental implants should be avoided. (See https://www.aaoms.org/docs/govt_affairs/advocacy_white_papers/mronj_posi Sound oral hygiene and regular dental care may be the best approach to lowering the risk of MRONJ. Patients taking bisphosphonates and undergoing endodontic therapy should sign an informed consent form, inclusive of the risks, benefits, and alternative treatment plans. The following recommendations have been suggested to reduce the risk of MRONJ associated with endodontic treatment[62]:

- Apply a 1-minute mouth rinse with chlorhexidine prior to the start of the treatment with the aim of lowering the bacterial load of the oral cavity.
- Avoid the use of anesthetic agents with vasoconstrictors in order to prevent impairment of tissue vascularization.
- Work under aseptic conditions, including removing of all caries and placement of rubber dam prior to intracanal procedures.
- Avoid damage to the gingival tissues during the placement of rubber dam.
- Avoid maintaining patency of the apical foramen to prevent

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bacteremia.

- Use techniques that reduce the risk of overfilling and overextension.

Aggressive use of systemic antibiotics is indicated in the presence of an infection in a patient taking bisphosphonates.[51] Discontinuing bisphosphonate therapy may not eliminate any risk of developing MRONJ.[54,57,59] Some clinicians have proposed use of the CTX (C-terminal telopeptide of type I collagen α_1 chain) test (Quest Diagnostics, Madison, NJ) for assessing the risk of developing bone osteonecrosis (BON). For patients who have developed MRONJ, close coordination with an oral maxillofacial surgeon or oncologist is highly recommended.

The AAE updated guideline published on 2018 titled: Endodontic Implications of MRONJ, states: “absent data, the committee considers the risk for ONJ after dental implant placement and endodontic or periodontic procedures that require exposure and manipulation of the bone as comparable to the risk associated with tooth extraction.” (See https://www.aae.org/specialty/wp-content/uploads/sites/2/2018/07/AAE_MedRelated_ONJ.pdf.) An astute awareness of the potential risk of MRONJ in patients receiving bisphosphonate therapy is critical. Increased attentiveness to the prevention, recognition, and management of MRONJ will allow the clinician to make the best treatment decisions. Our knowledge of MRONJ is developing rapidly, and it is essential that the clinician monitor the literature for changes in treatment protocols.[54,57,59]

Human immunodeficiency virus and acquired immunodeficiency syndrome

From 1987 through 1994, human immunodeficiency virus (HIV) disease mortality increased and reached a plateau in 1995. Subsequently, the mortality rate for this disease decreased an average of 33% per year from 1995 through 1998, and 6.2% per year from 1999 through 2016.[119] This dramatic improvement seems to be due to the use of a combination of highly active antiretroviral therapy (HAART) and improved preventive strategies.[51]

It is important, when treating patients with acquired immunodeficiency syndrome (AIDS), that the clinician understand the patient’s level of

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immunosuppression, drug therapies, and the potential for opportunistic infections. Although the effect of HIV infection on long-term prognosis of endodontic therapy is unknown, it has been demonstrated that clinicians may not have to alter their short-term expectations for periapical healing in patients infected with HIV.[80] The clinical team must also minimize the possibility of transmission of HIV from an infected patient, and this is accomplished by adherence to universal precautions. (See Universal Precautions for Prevention of Transmission of HIV and Other Bloodborne Infections, available at https://www.cdc.gov/niosh/topics/bbp/universal.html.)

Although saliva is not the main route for transmission of HIV, the virus has been found in saliva and its transmission through saliva has been reported.[32] Infected blood can transmit HIV, and during some procedures it may become mixed with saliva. Latex gloves and eye protection are essential for the clinician and staff. HIV can be transmitted by needlestick or via an instrument wound, but the frequency of such transmission is low, especially with small-gauge needles. Nevertheless, "Patients at high risk for AIDS and those in whom AIDS or HIV has been diagnosed should be treated in a manner identical to that for any other patient—that is, with standard precautions."[51]

A vital aspect of treatment planning for the patient with HIV/AIDS is to determine the current $CD4^+$ lymphocyte count and level of immunosuppression. In general, patients having a $CD4^+$ cell count exceeding 350 cells/mm^3 may receive all indicated dental treatments. Patients with a $CD4^+$ cell count of less than 200 cells/mm^3 or severe neutropenia (neutrophil count lower than 500/μL) will have increased susceptibility to opportunistic infections and may be effectively medicated with prophylactic drugs. WBC and differential counts, as well as a platelet count, should be ordered before any surgical procedure is undertaken. Patients with severe thrombocytopenia may require special measures (platelet replacement) before surgical procedures. Care in prescribing medications must also be exercised with any medications after which the patient may experience adverse drug effects, including allergic reactions, toxic drug reactions, hepatotoxicity, immunosuppression, anemia, serious drug interactions, and other potential problems. The practitioner should also be aware of oral manifestations of the disease as far as it concerns diagnosis and treatment planning. For instance,

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candidiasis of the oral mucosa, Kaposi sarcoma, hairy leukoplakia of the lateral borders of the tongue, herpes simplex virus (HSV), herpes zoster, recurrent aphthous ulcerations, linear gingival erythema, necrotizing ulcerative periodontitis, necrotizing stomatitis, oral warts, facial palsy, trigeminal neuropathy, salivary gland enlargement, xerostomia, and melanotic pigmentation are all reported to be associated with HIV infection. It is essential that consultation with the patient's physician occurs before performing surgical procedures or initiating complex treatment plans.[51,94]

Prosthetic implants

Patients with prosthetic implants are frequently treated in dental practices. The question concerning the need for antibiotic prophylaxis to prevent infection of the prosthesis has been debated for many years. A brief review of the subject should of interest to clinicians.

A statement was issued jointly in 2003 by the American Dental Association (ADA; Chicago, IL) and the American Academy of Orthopaedic Surgeons (AAOS; Rosemont, IL) in an attempt to clarify the issue.[30] The statement concluded that scientific evidence does not support the need for antibiotic prophylaxis for dental procedures to prevent prosthetic joint infections. It should be noted that although endodontics has been shown to be a possible cause of bacteremia,[21,42] the risk is minimal in comparison with extractions, periodontal surgery, scaling, and prophylaxis.[75] Nevertheless, there had been numerous conflicting opinions about the need to premedicate such patients that were raised over the next 15 years after the publication of the original statement in 2003, such as the 2009 statement of the AAOS entitled Antibiotic Prophylaxis for Bacteremia in Patients with Joint Replacements, which recommended that clinicians consider antibiotic prophylaxis for all total joint replacement patients prior to any invasive procedure that may cause bacteremia. (See American Academy of Orthopaedic Surgeons: AAOS releases new statement on antibiotics after arthroplasty, www.aaos.org/news/aaosnow/may09/cover2.asp.) In 2012, an evidenced-based guideline was published that included recommendations of the AAOS-ADA clinical practice guideline for Prevention of Orthopaedic Implant Infection in Patients Undergoing Dental Procedures. This guideline stated that there is limited evidence for discontinuing the practice of routinely

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prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures. (See American Academy of Orthopaedic Surgeons: Prevention of orthopaedic implant infection in patients undergoing dental procedures, available at www.aaos.org/research/guidelines/PUDP/PUDP_guideline.pdf.) In 2015, the *Journal of the American Dental Association* published an article based on the review of prior research stating that "for patients with prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures to prevent prosthetic joint infection. The practitioner and patient should consider possible clinical circumstances that may suggest the presence of a significant medical risk in providing dental care without antibiotic prophylaxis, as well as the known risks of frequent or widespread antibiotic use."[103] The 2017 update of the AAE's Antibiotic Prophylaxis Reference Guide also reflect this position. (See https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/aae_antibiotic-prophylaxis-2017update.pdf.)

Consultation with the patient's physician on a case-by-case basis is advisable to assess the need for prophylaxis.

Behavioral and psychiatric disorders

Stress reduction is an important factor in the treatment of patients with behavioral and psychiatric disorders. Sensitivity to the patient's needs must be part of the dental team's approach. Significant drug interactions and side effects are associated with tricyclic antidepressants, monoamine oxidase inhibitors, and antianxiety medications.[51] Consultation with physicians in such cases is essential before using sedatives, hypnotics, antihistamines, or opioids.

Psychosocial evaluation

The initial visit, during which medical and dental histories are collected, provides an opportunity to consider the patient's psychosocial status and how they value their oral health. Although some patients may want to maintain a tooth with a questionable prognosis, others may lack the ability to comprehend the potential risks and benefits. It would be a mistake to lead patients beyond what they can understand and appreciate. Furthermore, patients should not be allowed to dictate a treatment plan that has a poor

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prognosis.

The clinician should also assess the patient's level of anxiety as an important part of preparation for the procedure to follow. It is reasonable to assume that most patients are anxious to some degree, especially when they are about to undergo endodontic treatment. A conversation describing the procedure and what the patient can expect is an important part of an anxiety-reduction protocol.

A study evaluated the most common pathways of fear and anxiety in patients who previously had root canal treatment or were planning to have that procedure. Cognitive conditioning and parental pathways seemed to be the primary cause ($P < .05$) of fear and anxiety with root canal treatment. Females were significantly more likely to be influenced by indirect conditioned experiences such as informative, parental, verbal threat, and vicarious pathways. It was concluded that the origin of patients' fears requires more attention to the importance of treating endodontic-related fear and anxiety. More knowledge about these factors would enable dentists to better manage patients in a multicultural society.[17] More information will be provided in the Anxiety segment of this chapter.

Development of the endodontic treatment plan

The long-term strategic value of a tooth should be considered before presenting an endodontic treatment plan. For example, a restorative plan could be endangered by including a tooth with a questionable endodontic prognosis. Currently, endodontic treatment options exist that were not previously supported by significant levels of evidence. The options include vital pulp therapy, regenerative procedures, and intentional replantation. Although some decisions may be straightforward, considering alternative treatment options can be challenging, as the clinician weighs multiple factors that will play a role in determining the ultimate success or failure of the case. For example, if initial endodontic nonsurgical or surgical procedure has failed, the most common alternative has been an implant or a fixed or removable prosthesis. However, there is increasing evidence that intentional replantation is a cost-effective procedure that may be accomplished, in select cases, with a good prognosis. It is important to note that even if replantation fails, a dental implant remains as an additional option.

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Referral of the patient to a specialist should be considered when the complexity of a procedure is beyond the ability of a clinician. The complexity can be due to restorative factors, unusual anatomy, and calcification of the chamber and/or the canal. Other factors that affect endodontic prognosis include periodontal, restorative, and the patient's systemic health.

Endodontic prognosis

Prognostic studies have identified a number of preoperative factors affecting the outcome of primary endodontic treatment. In a systematic review, it was determined that the absence of periapical radiolucency improves the outcome of root canal treatment significantly. The same study also showed that tooth vitality does not have an impact on the endodontic prognosis, as long as the periapex is healthy.[72] Studies have shown that the size of the radiolucency may also affect the outcome of endodontic treatment.[72,87] It was found that the existence of sinus tract, narrow but deep periodontal probing depth, pain, and discharging sinus have a significant effect on the outcome of nonsurgical root canal treatment.[69] Preoperative pain is not only one of the most important predictors of postoperative pain,[66] it also has an impact on tooth survival after endodontic treatment.[68]

These findings suggest that all preexisting signs and symptoms that may affect the prognosis of treatment, along with the prognostic factors associated with other disciplines—which will be discussed in the next segment—should be taken into consideration when developing a treatment plan. It is of utmost importance that the prognosis and risks and benefits of the treatment be relayed to the patient before the initiation of the treatment as well.

There is a general belief that the prognoses for retreatment cases are poorer than for primary treatment, but this is not universally supported. In a systematic review it was suggested that the outcomes of retreatment cases should be similar to treatment cases as long as access to the apical infection can be reestablished.[67] However, there is some evidence indicating that the incidence of postoperative pain and flare-up is higher in retreatment cases in comparison to treatments.[36] The presence of preoperative periapical lesion, apical extent of root filling, and quality of coronal restoration has been proved to significantly affect the outcome of retreatment cases.[67]

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Retreatment cases offer a particular set of challenges to the clinician (Figs. 5.3 and 5.4), and this topic is covered extensively in Chapter 10. Important questions to be considered before retreatment include the following:

- Why did the treatment fail?
- Can the point of bacterial entry to the canal space be identified?
- Are prior radiographs available for review?
- Is there an obvious endodontic procedural problem that can be corrected?
- Is the canal system readily accessible for reentry?
- There are additional factors (i.e., a significant periodontal defect or being under immunotherapy) that may have contributed to the failure.
- Is the tooth critical to the treatment plan? (Keeping in mind that including a tooth with a questionable endodontic or periodontal prognosis may endanger an entire prosthesis.)
- Does the patient understand the prognosis for the tooth and want to attempt retreatment? This question and its answer should be documented in the patient's record. The dentist could enter in the patient's record the following statement: "All reasonable options including extraction were presented to the patient and advantages and disadvantages of each were explained."

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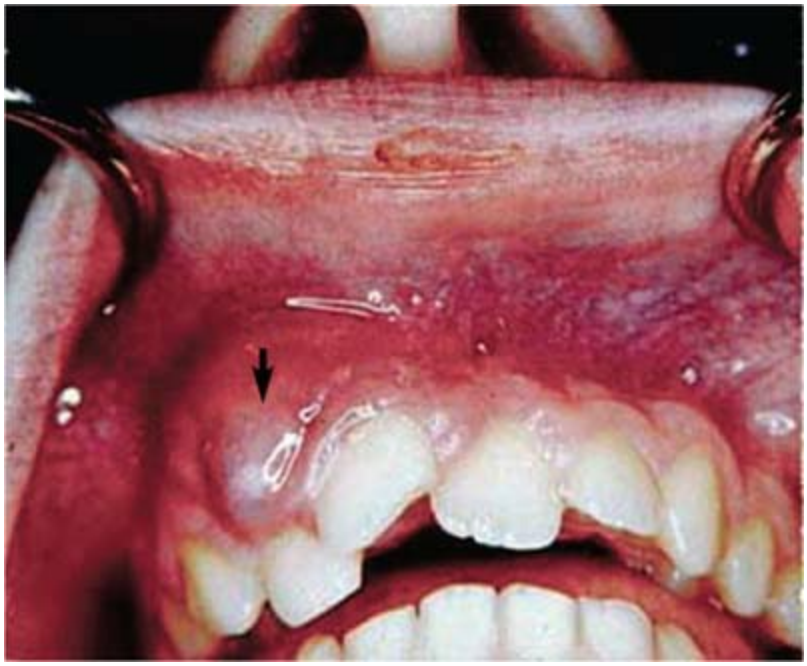

FIG. 5.3 Incision and drainage should be performed on this fluctuant swelling *(arrow)* in conjunction with canal instrumentation.

Close-up view shows an arrow pointing at gray swollen tissue on the gum line in the upper jaw with upper lips held by retractor.

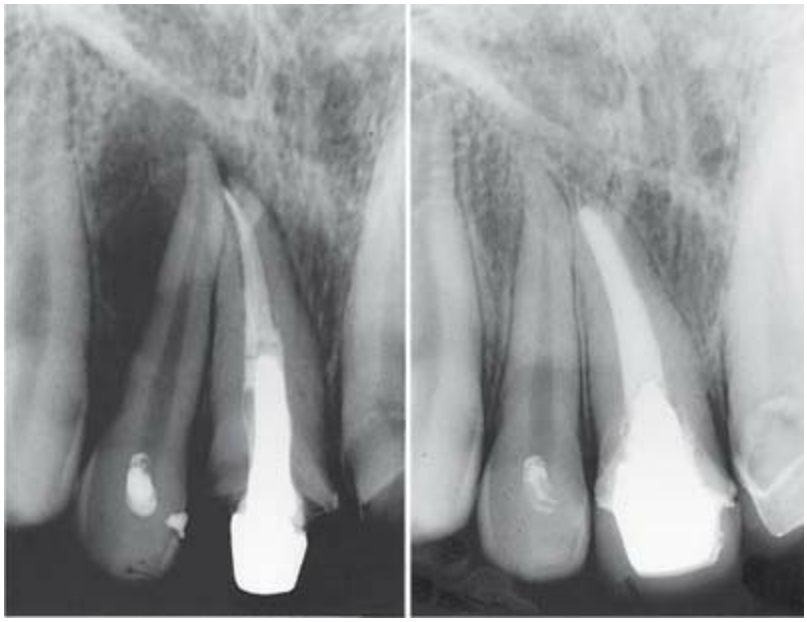

FIG. 5.4 Two years after endodontic therapy of tooth #8, the patient returned with pain and swelling. A clinician mistakenly began endodontic access on tooth #7, without confirming the apparent

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radiographic diagnosis by sensibility testing. Tooth #7 was vital, and tooth #8 was successfully retreated after removal of the post.

Radiograph on the left shows 7th tooth with radiolucent root canal with a small radiopaque patch in the pulp. 8th tooth radiopaque root canal and crown without enamel. The region near seventh tooth is extremely radiolucent extending from gum line to the root apex.

Radiograph on the right shows eighth tooth with radiopaque crown and root canal with radiolucent enamel. The tissue near seventh tooth has high-density.

Source: (Courtesy Dr. Leon Schertzer.)

A retreatment plan should be developed after the clinician has determined the cause of failure and weighed other factors that may affect the prognosis (e.g., root fracture, defective restoration) (Figs. 5.5 to 5.8). Retreatment cases may require surgical endodontics in combination with nonsurgical retreatment. Referral to a specialist is often helpful when planning treatment for complex cases. If retreatment (with or without surgery) of a tooth with a new restoration is being considered, it should be weighed against the possibility of intentional replantation or an implant. Many variables must be considered before a reasonable conclusion can be reached.

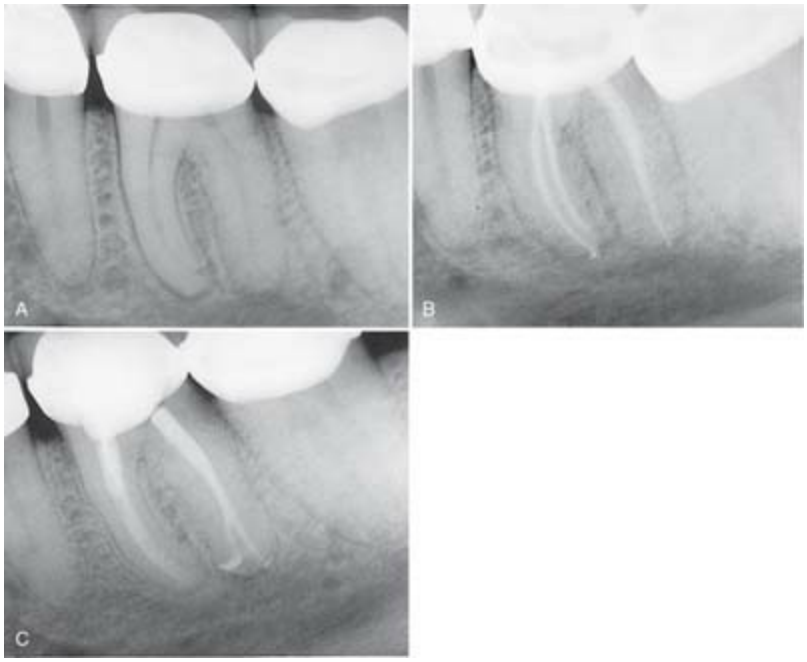

FIG. 5.5 Many years after endodontic treatment of tooth #19, the patient returned with a chief complaint of pain and an inability to chew

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with the tooth. Despite the radiographic appearance of excellent endodontic treatment, the tooth was retreated and the patient's pain disappeared. Note the unusual distal root anatomy, which was not apparent during the initial procedure. **A,** Initial radiograph. **B,** Completion of initial endodontic therapy. **C,** Retreatment.

A) Radiograph shows a tooth at the center with radiolucent root canals and radiopaque crown.

B) Radiograph shows the central tooth with radiopaque root canals and radiolucency near the root apex.

C) Radiograph is similar as that of B, except that there is a dense tissue near the root apex.

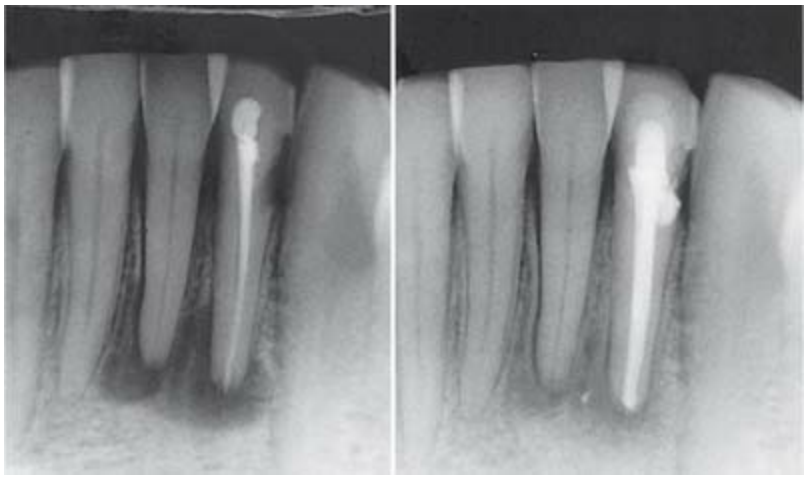

FIG. 5.6 Initial radiograph was misleading and implicated tooth #23 and tooth #24. Pulp testing indicated a vital pulp in tooth #24, and it was not treated. Retreatment of #23 resulted in healing of the periradicular lesion.

Radiograph on the left shows well-circumscribed dark patches around the root apices of two teeth. Radiograph on the right shows a trabecular tissue in place of the well-circumscribed dark patches.

Source: (Courtesy Dr. Leon Schertzer.)

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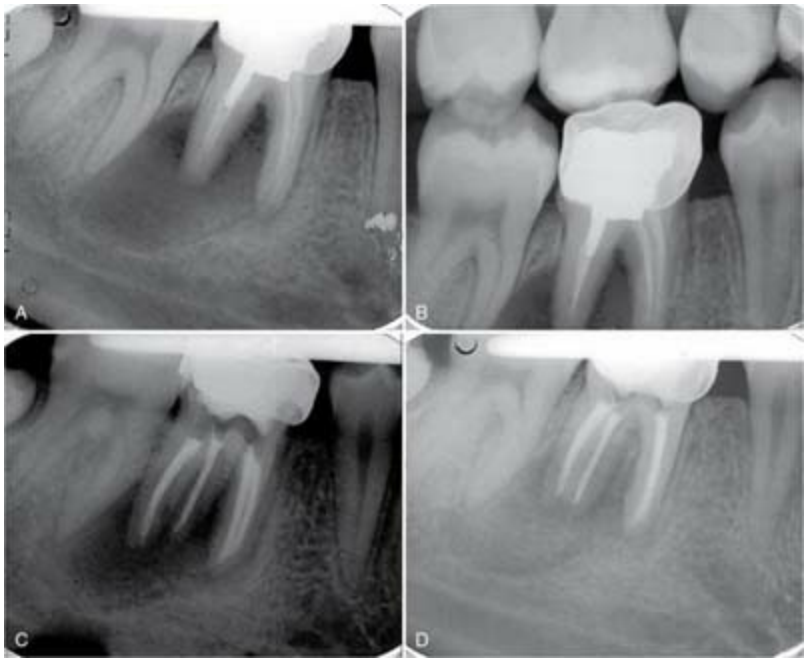

FIG. 5.7 Nonsurgical retreatment of tooth #30. An additional root was located and treated. **A,** Note inadequate endodontic treatment and large periapical lesion. **B,** Bitewing radiograph. **C,** Retreatment after post removal. **D,** Eighteen-month recall radiograph indicates periapical healing.

A) Radiograph shows a large oval-shaped, dark patch extending below the gum line to far lower end.

B) Radiograph shows lower and upper teeth touching each other with the large dark patch around the tooth in the lower jaw.

C) Radiograph shows tooth with four radiopaque root canals. The tissue around the root has less-density.

D) Radiograph shows tooth with radiopaque root canals and dense-tissue surrounding the root.

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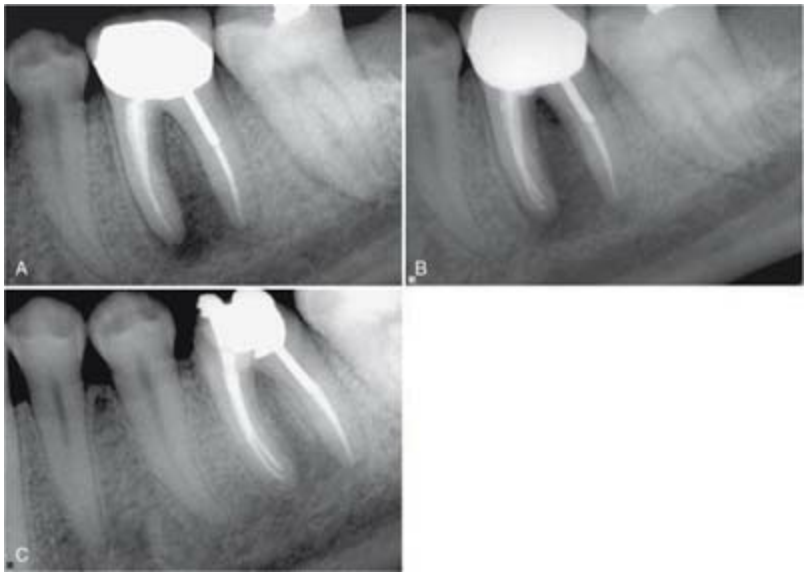

FIG. 5.8 Despite several exacerbations during endodontic retreatment, this case responded rapidly after the completion of therapy. Radiographic evaluation indicates a good periapical response after only 4 months. **A,** Radiograph taken 2 years after initial root canal therapy. **B,** Retreatment of the mesial root. **C,** Radiograph taken 4 months following retreatment.

A) Radiograph shows tooth at the center with radiopaque crown and root canals. The tissue is less dense at the center of root and root tips.

B) Radiograph shows porous tissue in the bifurcated root of tooth.

C) Radiograph shows slightly dense tissue at the center of root and root tips.

Single-visit versus multiple-visit treatment

Treatment planning includes considering a multiple or single visit approach. The number of roots, canal anatomy, time available, and the clinician's skills are among the factors to be considered. Severity of the patient's pretreatment symptoms is another important consideration. For example, a patient in severe pain, with or without swelling, is not a prime candidate for single visit endodontics. The initial goal for the patient in severe pain should be directed at alleviating pain, with filling of the canal deferred until the symptoms have been brought under control. The clinician's judgment of what the patient can comfortably tolerate (regarding duration of the visit) is made on a case-by-case basis. The second visit allows the clinician to determine the effect of the initial treatment and then complete instrumentation and filling if the tooth is

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no longer symptomatic.

Numerous studies have compared single and multiple visit cases and reached different conclusions about the outcomes of each approach. For example, some studies have reported less posttreatment pain in single-visit cases,[52,84,104] whereas a systematic review found that the incidence of postobturation discomfort was similar in the single- and multiple-visit approaches.[29] In another systematic review, it was concluded that there was a lack of compelling evidence, indicating a significantly different prevalence of posttreatment pain/flare-up with either single- or multiple-visit root canal treatment.[92] Differences in research methodology explain the conflict in these findings.

Regarding the healing rate of single- and multiple-visit cases, systematic reviews have found that there is no detectable difference in the effectiveness of root canal treatment in terms of radiologic success between single and multiple visits,[29] even in cases of infected teeth.[104]

Teeth with nonvital pulps and apical periodontitis pose a microbiologic problem. It is important to avoid pushing bacterial debris into the periapical tissues. Agreement is lacking concerning the appropriateness of single-visit endodontics for treating these patients. Some have postulated that the intervisit use of an antimicrobial dressing is essential to thoroughly disinfect the root canal system.[99,100,110] In contrast, other researchers have found no statistically significant difference in success when using the single-visit or multiple-visit approach to the nonvital tooth with apical periodontitis.[29,63,77,79,87,91,116]

Differences in research findings concerning single or multiple visits might be due to variations in research methodology, including sample size, duration of follow-up, and treatment methods.

It is possible that total elimination of bacteria may not be absolutely necessary for healing. Perhaps maximal reduction of bacteria, effective root canal filling, and a timely satisfactory coronal restoration can result in a high level of clinical success. However, regardless of the number of appointments, effective bacteriologic disinfection of the root canal system is critical.

Treatment planning for an endodontic case should be based on biologic considerations. Patients who present with acute symptoms present a different set of biologic and clinical issues than those with an asymptomatic tooth.

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Swelling associated with an abscess, cellulitis, or presence of a stoma represent signs of pathologic processes. The biologic significance of these conditions should be considered before determining specific goals for each visit.

Developing goals for each visit helps to organize the treatment. For example, an asymptomatic anatomically uncomplicated anterior, premolar, or molar tooth may be a candidate for a single-visit plan, depending on the skills of the practitioner. However, as the complexity of the case increases and/or symptoms become more severe, a multi-visit approach becomes more appropriate.

These recommendations have a biologic basis. Biologically, it is not reasonable to partially instrument root canal systems, thereby leaving residual inflamed pulpal remnants or necrotic debris in the canal, because such remnants may cause pain and be susceptible to infection. The clinician would be well advised to begin canal instrumentation only if time permits the extirpation of all pulp tissue and complete debridement of the root canal system.

In most cases, the clinical procedures required to complete endodontic treatment can be accomplished in a single visit. However, that does not mean it is the wiser course of treatment. What can be done and what should be done represent two very different approaches to endodontic treatment. The patient's systemic health, level of anxiety, and symptoms, as well as the complexity of the root canal system, and clinician's experience are all factors that must be considered.

A study concerning the outcome of initial endodontic treatment noted the complexity of treating apical periodontitis.[87] The author commented that treatment of this disease cannot be improved merely by changing treatment techniques. Because apical periodontitis results from interactions between microorganisms, their environment, and the host immune system, only the use of effective modifiers of any of these three factors might significantly improve the outcome of treatment.

Surgical endodontics

Surgical evaluation is particularly valuable in the diagnosis of lesions that may be nonodontogenic. Biopsy is the definitive means of diagnosing

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osseous pathosis, which may mimic a lesion of endodontic origin. When retreatment is being considered, the clinician must determine whether nonsurgical, surgical, or combined treatment is appropriate. This decision is influenced by the presence of complex restorations, posts, and the radiographic assessment of prior endodontic therapy.

Endodontic surgery is most often performed in an attempt to improve the apical seal and correct failure of nonsurgical therapy. Bacteria are the essential cause of failure. It is important that the clinician determine the bacterial path of ingress. For example, a deficient restoration or recurrent decay will result in microleakage into the root canal space. Unless that issue is addressed, the outcome of apical surgery may not be predictable (Fig. 5.9).

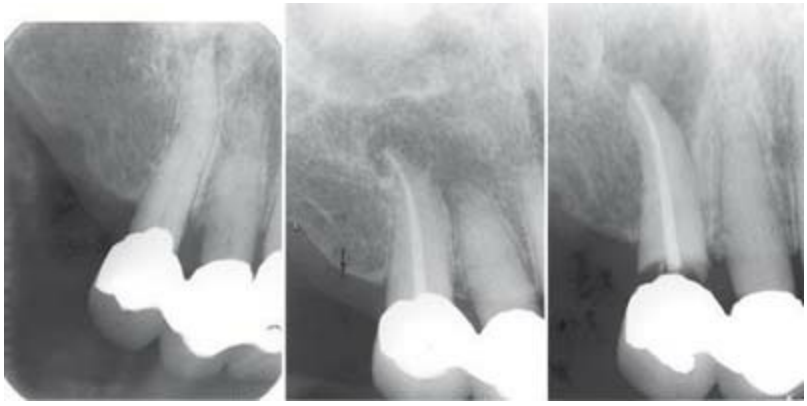

FIG. 5.9 Four years after endodontic therapy, the patient complained of pain and swelling associated with tooth #6. The initial impression was that apical surgery was indicated. However, further radiographs revealed the true cause of the endodontic failure. The initial endodontic access through the crown or caries damaged the coronal seal and recurrent decay followed.

Set of three radiographs are marked left to right as follows:

Radiograph shows 6th tooth with radiopaque crown and receding gum line. The tissue on one side of tooth is porous.

Second radiograph shows filled root canal of 6th root with slightly dense tissue near the gum line and less-dense tissue at the root tip. Third radiograph shows well-defined radiolucency in the neck region.

When a deficient restoration is identified, it must be replaced to prevent continued bacterial penetration. Endodontic surgery (see Chapter 11) may

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also be performed as a primary procedure when there are complications such as calcific metamorphosis. In those cases, by using surgery as primary therapy, an apical seal can be established while preserving the crown of the tooth. The treatment plan for these cases is determined after reviewing multiple radiographs and considering the possibility of completing nonsurgical therapy without destroying an otherwise functional crown or natural tooth. Endodontic surgery without prior nonsurgical therapy should be considered a treatment of last resort and only when nonsurgical treatment is not possible.

Reviewing the best available evidence for alternative treatments is an important aspect of treatment planning for a tooth with failed endodontics. Evidence concerning the healing potential after endodontic surgery is an important consideration in the management of posttreatment disease.[86] Numerous studies have examined the outcome of apical surgery and the results vary considerably.[111,114,121] This variability may reflect actual outcome differences or reflect variations in case selection techniques, recall periods, and methodology.

A prospective study indicated that there is an increased odds ratio for disease persistence for teeth with larger pretreatment lesions and pretreatment root canal filling of adequate length.[114] Another study found that patients presenting with pain at the initial examination before surgery had a significantly lower rate of healing at the 1-year follow-up compared with patients who did not have pain at the initial examination.[111]

It should also be noted that the periodontal condition of the tooth, including the interproximal bone levels and the amount of marginal bone loss, has been shown to significantly affect the long-term prognosis of periapical surgery.[112,115] Moreover, it has been shown that isolated endodontic lesions have much higher success rates at the 1- to 5-year follow-up (95.2%) in comparison with endodontic-periodontal combined lesions (77.5%).[46]

Dramatic changes in surgical technique and materials have occurred. The advent of microscopy, endoscopy, and ultrasonics, as well as improved retrograde filling materials, represents important modifications of surgical technique. An outcome study comparing traditional root-end surgery (TRS) and endodontic microsurgery (EMS) found that the probability of success for

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EMS was 1.58 times greater than the probability of success for TRS.[97] Another study comparing the EMS techniques with and without the use of higher magnification found that the difference of probability of success between the groups was statistically significant for molars, but no significant difference was found for the premolar or anterior teeth.[96] CBCT has proved to be of value in some surgical cases, producing three-dimensional images of a tooth, pathosis, and adjacent anatomic structures. It is beneficial for localizing the mandibular canal,[48] mental foramen, maxillary sinus, and nasal cavity.[18]

Intentional replantation

Intentional replantation is a procedure that has long been thought of as a measure of last resort and rarely considered during treatment planning. Its increasing popularity may be based on evidence indicating that its success rate, when specific guidelines are followed, may approach that of other endodontic procedures and dental implants. When compared to dental implants, the procedure offers a more cost-effective and timely means of maintaining a tooth that would otherwise be lost. In terms of patient-centered outcomes, replantation should be considered prior to extraction of previously endodontically treated teeth. While non-surgical or surgical endodontic treatment remains the preferred treatment for most patients, evidence is building supporting the concept that replantation of a tooth is a valuable option for some patients. It should be noted that in those cases, where replantation is not successful, a dental implant remains an option.

In 2017, a systematic review compared the survival of teeth intentionally replanted, using a modern technique and cost effectiveness, compared with single-tooth implants. Six studies met the inclusion criteria. Meta-analysis resulted in a survival rate of 89.1%. Compared with a single- tooth implant, intentional replantation was more cost-effective, even when custom post/core and crown were also needed. The authors concluded that based on the meta-analysis, there was a high survival rate for intentional replantation. Although the survival rate for implants was higher, intentional implantation is a more cost-effective treatment modality. The authors concluded that, “When nonsurgical and surgical retreatments of a tooth are not feasible or have a poor prognosis, intentional replantation with a modern technique is a reliable

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and cost-effective method of treating teeth. The option of intentional replantation should be discussed with patients as an alternative to extraction and implant placement."[53] (For further information please visit Chapter 11.)

Regenerative endodontics

Regenerative endodontics is a developing area in the treatment of immature teeth with infected root canals that has been described as a "paradigm shift"[22] in the management of these teeth and may result in continued root maturation and apical closure. Traditional approaches using calcium hydroxide for apexification and apical barrier techniques using mineral trioxide aggregate (MTA) have been used in the treatment of immature teeth with pulp necrosis. When using that technique, there is usually no further root development. The roots remain thin and fragile with a high risk of fracture and tooth loss.[81] It has been suggested that regenerative endodontic procedures (REPs) that utilize endogenous stem cells that are introduced in the canal by lacerating the periapical tissues to fill the canal with blood should be used for the treatment of immature teeth with pulp necrosis. Regenerative endodontics has been defined as "biologically based procedures designed to replace damaged structures, including dentin and root structures, as well as cells of the pulp-dentin complex."[22] The AAE recently updated the developed clinical considerations for regenerative procedures; this can be found on the AAE website (https://www.aae.org/specialty/wp-content/uploads/sites/2/2018/06/ConsiderationsForRegEndo_AsOfApril2018.
Most studies that have compared REPs with the traditional approaches of calcium hydroxide apexification and MTA apical barrier techniques have shown comparable outcomes.[3,44] However, a study that compared traditional and regenerative treatment protocols showed revascularization was associated with significantly greater increases in root length and thickness compared with calcium hydroxide apexification and MTA barrier placement as well as higher overall survival rates.[40]

There are several factors that must be taken into consideration when planning REP treatment for a tooth, such as: (a) remaining tooth structure, (b) tooth location, (c) level of tooth maturity, (d) number of visits, and (e) patient's compliance.

It must be noted that although REP is becoming more common, the

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traditional apexification techniques still have their own place in the treatment planning process for a premature tooth. For instance, the amount of remaining crown structure is one of the factors to be considered. Placing a post after a REP is usually difficult; therefore, if the tooth has lost most of its crown structure and may need a post in the future, other treatment modalities such as traditional $Ca(OH)_2$ or MTA plug apexification should be considered. Another factor to consider in the treatment planning stage is the location of the tooth. New materials are being introduced on a frequent basis, and each one may or may not be a suitable choice for all teeth. It is of utmost importance for the clinician to refer to the highest level of scientific evidence in treatment planning such cases. For example, MTA is the most common material used in REP; however, one of the common complications with MTA is tooth discoloration, which is a serious esthetic concern with anterior teeth. In these teeth, other materials, such as Biodentine, may be favored.[7] With regards to apical maturity, REP is mostly considered for immature teeth, since this area is rapidly developing, it is important for the clinician to have a broad knowledge on the protocols and biomaterials.

Tooth maturity is another factor to consider. Although it has been suggested that less mature teeth are more suitable for regenerative endodontics,[47] there have been reports of using REP in treating teeth with necrotic pulps and apical periodontitis.[106] Therefore, it is of utmost importance that the clinician discuss all treatment options prior to suggesting the definitive treatment.

The number of visits and patient's compliance are other critical elements in treatment planning for premature teeth. According to the AAE clinical protocol (mentioned earlier), RET usually requires two treatment and few follow-up visits; however, $Ca(OH)_2$ calcification requires multiple observation visits prior to definitive treatment session. It is important to discuss the number of sessions and their frequency with the patient or the parent/guardian in terms of patient's availability and compliance. Patients with fewer complaints may be more suitable for less technique-sensitive treatments or ones with fewer required number of sessions. (For more information refer to Chapter 12.)

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Interdisciplinary treatment planning

Periodontal considerations

Complex cases may require a multidimensional approach. For example, extensive periodontal lesions may complicate the prognosis of endodontic treatment. A periodontist and an endodontist might be able to add their insights to the prognosis of treatment and avoid future problems.

A 4-year retrospective study found that attachment loss and periodontal status affected endodontic prognosis of endodontically treated molars.[95] It is crucial that the dental practitioner be aware of the periodontal factors that may influence the prognosis of endodontic treatment, such as root perforations, bone loss, and clinical attachment loss.

When establishing the prognosis of a tooth with an endodontic/periodontal lesion, there are essential factors to be considered. Determination of pulp vitality and the extent of the periodontal defect are central to establishing the prognosis and developing a treatment plan for a tooth with an endodontic/periodontal lesion (see also Chapter 25).

In primary endodontic disease, the pulp is nonvital, whereas in primary periodontal disease, the pulp retains vitality. True combined endodontic-periodontal disease occurs less frequently. The combined lesion is found when the endodontic disease process advances coronally and joins with a periodontal pocket progressing apically. There is significant attachment loss with this type of lesion, and the prognosis is questionable.[85] The radiographic appearance of combined endodontic-periodontal lesions may be similar to that of a vertically fractured tooth. Therapy for true combined lesions requires both endodontic and periodontal therapy. Sequencing of treatment is based on addressing the initial chief complaint.

The prognosis and treatment of each type of endodontic-periodontal disease vary. Primary endodontic disease should be treated solely by endodontic therapy, and the prognosis is usually good (Figs. 5.10 and 5.11). Primary periodontal disease should be treated only by periodontal therapy, and the prognosis varies depending on the severity of the disease and patient's response to treatment.[85]

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FIG. 5.10 **A,** Inflamed, edematous interproximal tissue *(arrow)* caused by acute endodontic pathosis. **B,** Soft-tissue healing *(arrow)* 3 days after initiation of endodontic treatment. **C,** Periradicular pathosis. **D,** Completed endodontic therapy. **E,** Periradicular healing at 1-year recall.

A) Close-up view shows abscess in-between the root of two teeth in the lower jaw. Teeth have yellow stains at the margin and on the crown part.

B) Close-up view shows lower jaw. An arrow points toward the normal gum line between teeth and white margins.

C) Radiograph shows teeth with radiopaque crowns and radiolucent root canals. Porous tissue extends from gum line to below the root tips.

D) Radiograph shows teeth with radiopaque root canals. The tissue between teeth is slightly dense.

E) Radiograph shows normal teeth with dense tissue extending from gum lines to below the root tips.

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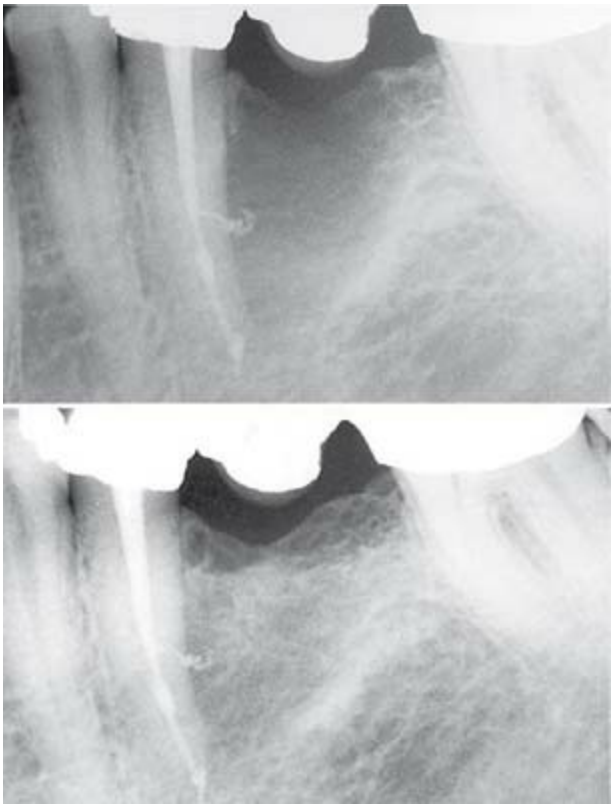

FIG. 5.11 A large, bony defect associated with tooth #20 healed after endodontic therapy. The tooth was nonvital, and no significant periodontal probing depth indicated pulpal disease.

Radiograph on the top shows well-defined radiolucency with receding gum line adjacent to tooth. Radiograph at the bottom shows dense tissue in place of the radiolucency.

Pathogenesis of the lesion can be better understood after sensibility testing, periodontal probing, radiographic assessment, and evaluation of dental history. When extensive prostheses are planned, the potential risk of including a tooth with a questionable prognosis must be considered. It is not prudent to incorporate a chronic problem into a new complex prosthesis (Fig. 5.12).

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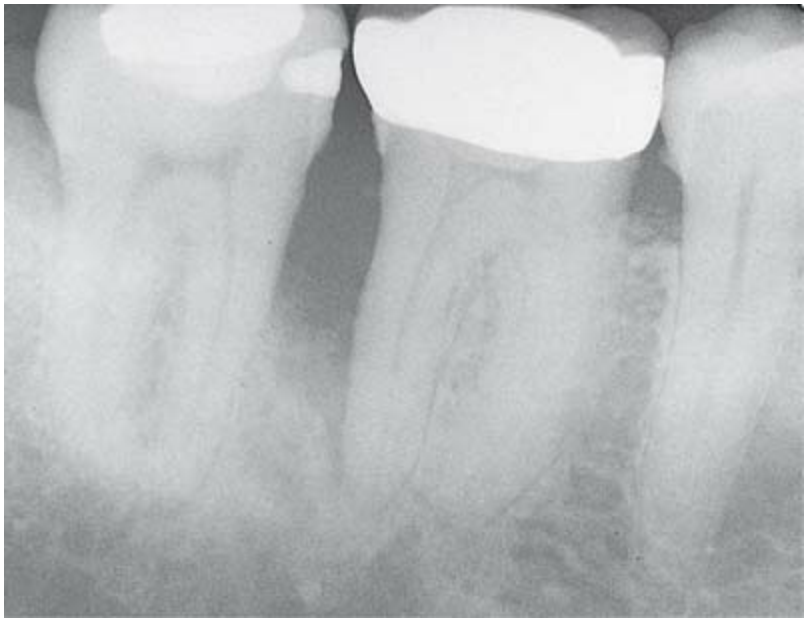

FIG. 5.12 Tooth #30 has a poor prognosis. Periodontal probing reached the apex of the distal root. Extraction is indicated and should be done as soon as possible to prevent further damage to the mesial bone associated with tooth #31. Implant site preservation is another consideration in treatment planning for this case.

Radiograph shows the central tooth with radiopaque crown and a perforated dark patch along the one side of root.

Restorative and prosthodontic considerations

A satisfactory restoration may be jeopardized by a number of factors. Subosseous root caries (perhaps requiring crown lengthening), poor crown-to-root ratio, and extensive periodontal defects or misalignment of teeth may have serious effects on the final restoration. These problems must be recognized before endodontic treatment is initiated. For complex cases, a restorative treatment plan should be in place before initiating endodontic treatment (see Chapter 23). Some teeth may be endodontically treatable but nonrestorable, or they may represent a potential restorative complication because of a large prosthesis. Reduced coronal tooth structure under a full-coverage restoration makes endodontic access more difficult because of reduced visibility and lack of radiographic information about the anatomy of the chamber. It is not unusual for restorations to be compromised during endodontic access (see Fig. 5.12). Whenever possible, restorations should be

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removed before endodontic treatment.

Full coverage restorations are usually suggested after endodontic treatment. In a systematic review on tooth survival following nonsurgical root canal treatment, four factors were found to be of significance in tooth survival[70]:

- A crown restoration after root canal treatment.
- Tooth having both mesial and distal proximal contacts.
- Tooth not functioning as an abutment for removable or fixed prosthesis.
- Tooth type or specifically nonmolar teeth.

Another systematic review found that the odds for healing of apical periodontitis increase with both adequate root canal treatment and adequate restorative treatment. However, poorer clinical outcomes may be expected with adequate root filling/inadequate coronal restoration and inadequate root filling/adequate coronal restoration, with no significant difference in the odds of healing between these two combinations.[34] These findings suggest that the quality of the coronal restoration is as important as the quality of the root canal treatment. Therefore, to increase the success of the treatment, it is strongly suggested that the clinician discuss the restorative plan of the tooth with both the patient and—if it is a referred patient—with the referring dentists before initiation of treatment.

Endodontic therapy or dental implant

The successful evolution of dental implants as a predictable replacement for missing teeth has had a positive impact on patient care. A clinician now has an additional possibility to consider when developing a treatment plan for a patient with a missing tooth or teeth. More challenging is the decision concerning whether or not to provide endodontic therapy for a tooth with a questionable prognosis or extract and use a single-tooth implant as a replacement or attempt replantation. Numerous studies have evaluated both the nonsurgical endodontic therapy[20,71,72,88,89,107] and endosseous dental implants.[1,19,38]

It is not possible to compare outcome studies because of variations in

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research methodologies, follow-up periods, and criteria associated with determining success or failure. A review of outcome studies points to the need for randomized controlled trials with standardized or similar methodologies that could provide a higher level of evidence to use in answering important clinical prognostic questions.

A synthesis of available evidence indicates that both primary root canal treatment and single-tooth implants are highly predictable procedures when treatment is appropriately planned and implemented. A study assessed clinical and radiographic success of initial endodontic therapy of 510 teeth over a 4- to 6-year period. It was found that 86% of teeth healed and 95% remained asymptomatic and functional.[20] Another study considered the outcomes of endodontic treatment on 1,462,936 teeth. More than 97% of teeth were retained after 8 years.[88] In a similar study, it was found that overall, 89% of the 4744 teeth were retained in the oral cavity 5 years after the endodontic retreatment.[89] A study concerning the outcome of endodontic therapy on 1312 patients in general practice with a mean follow-up time of 3.9 years found a combined failure rate of 19.1%, concluding that "failure rates for endodontic therapy are higher than previously reported in general practices, according to results of studies based on dental insurance claims data."[9]

The ADA's Council on Scientific Affairs has reported high survival rates for endosseous implants. An evaluation of 10 studies with more than 1400 implants demonstrated survival rates ranging from 94.4% to 99% with a mean survival rate of 96.7%.[1] In a recent systematic review, the authors claimed that with the mean range of 13.4 years follow-up, the survival rate of dental implants were 94.6% and the success rate for half of the cases, which used the same criterion with a mean period of follow-up of 15.7 years, was 89.7%, but a statistical analysis was not performed due to heterogenicity.[108] With such relatively high survival rates reported for endodontics and single-tooth implants, a clinician must consider a multiplicity of factors within the context of the best available evidence.

In a retrospective cross-sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants, it was suggested that restored endodontically treated teeth and single-tooth implant restorations have similar survival rates, although the implant group showed a longer average and

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median time to obtain function and a higher incidence of posttreatment complications requiring subsequent treatment intervention.[24] Aside from success, the clinician must also consider the success rate of dental implants, which is usually significantly lower than success rates. The term "success" usually involves more complex parameters and criteria and is associated with the health and quality of the implants.[108] Any treatment intervention due to postoperative complications will affect the success of the implant without directly affecting its survival. In a study on long-term survival and success of dental implants, the authors found that "The cumulative complication rate after an observation period of 10 to 16 years was 48.03%, which meant that substantial amounts of chair time were necessary following implant placement."[98] In a recent study of patients who have received both nonsurgical root canal treatment and single-tooth implants, it was concluded that "the number of adjunct and additional treatments, the number of appointments, the elapsed time before the final restoration, the number of prescribed medications, and the cost of the treatment were significantly higher for single tooth implants in comparison with non-surgical root canal treatment."[109]

A review summarized the best available evidence concerning factors influencing treatment planning involving preservation of a tooth with endodontic therapy or replacement by a single-tooth implant. Factors considered included prosthetic restorability of the natural tooth, quality of bone, aesthetic concerns, cost-to-benefit ratio, systemic factors, potential for adverse effects, and patient preferences.[38] The authors concluded that "endodontic treatment of teeth represents a feasible, practical and economical way to preserve function in a vast array of cases and that dental implants serve as a good alternative in selected indications in which prognosis is poor."[38]

Aside from treatment outcome, number of sessions, duration of the treatment, and additional adjunct treatments, there are other factors involved in any treatment that the practitioner has to consider when making a treatment planning decision. In a study evaluating the quality of life of endodontically treated versus implant treated patients, the results showed a high rate of satisfaction with both treatment modalities.[31] One study found that when compared to endodontic molar retreatment and fixed partial dentures,

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implant-supported restoration, despite its high survival rate, has been shown to be the least cost-effective treatment option.[48] Another study found that "endodontically treated natural teeth may provide more effective occlusal contact during masticatory function compared with implant-supported restorations, leading to more efficient mastication."[117]

Another important factor is the patient's health status. Implants require a surgical procedure that may not be possible due to the patient's medical status. One area of concern is diabetes mellitus. However, it has been shown that dental implant osseointegration can be accomplished in these subjects, as long as they have good glycemic control.[39]

It seems clear that patients are best served by retaining their natural dentition as long as the prognosis for long-term retention is positive. It is not reasonable to extract a tooth if endodontics with a good prognosis can be completed. It is also not reasonable for a patient to invest in root canal therapy, a post, and a crown if the prognosis is highly questionable and an implant with a good prognosis can be placed. An important advantage of providing endodontic therapy is to allow rapid return of the patient's compromised dentition to full function and aesthetics. This rapid return is in marked contrast to the use of provisional restorations associated with dental implants while waiting for osseous integration. The challenge is to weigh all pretreatment variables and reach a reasonable conclusion concerning the prognosis for tooth retention or implant placement.

Interestingly, some endodontic advanced education programs are now including implant training in their curricula. This training will enable the endodontist to provide more value to the patient and referring the dental clinician as treatment plans are determined. Such dually trained endodontists will be well positioned to provide endodontic therapy or place an implant as best serves the patient.

Finally, the AAE Position Statement on Implants referring to the Ethics of Clinical Practice states: "Inappropriate treatment, such as: performing endodontic therapy on nonrestorable or periodontally hopeless teeth; or placing single-tooth implants when the natural tooth could predictably be retained, would also be considered unethical. Failure to adhere to these principles not only violates the trust placed in the dental profession, but leaves the dentist vulnerable to litigation" (see

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https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/implantsstatement.pdf).

Other factors that may influence endodontic case selection

A variety of factors may complicate proposed endodontic therapy. Calcifications, dilacerations, or resorptive defects may compromise endodontic treatment of a tooth with potentially strategic value (Fig. 5.13).

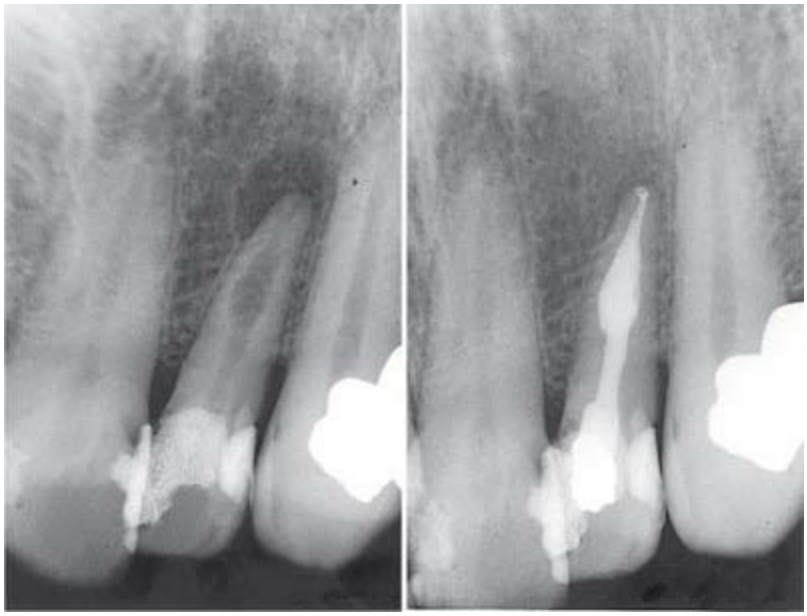

FIG. 5.13 Resorptive defects can be successfully treated. Early intervention, before there is perforation of the root, increases the chance of success.

Radiograph on the left shows three teeth where the central tooth has radiolucency at the center of root with a porous tissue at the root apex. Radiograph on the right shows radiopaque root canal of the central tooth with slightly dense tissue at the root apex.

Source: (Courtesy Dr. Leon Schertzer.)

Diagnosis and treatment of resorptive defects may be challenging at times. Distinguishing external resorption from internal resorption is usually possible with routine radiographs using different angles; but in order to detect the

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exact location and extension of certain types of resorption, especially external cervical resorption (ECR), the use of the CBCT is paramount for treatment planning purposes.[28,76] Resorptive lesions are usually asymptomatic, and therefore, are often found accidentally when reviewing radiographs. As a result, in many cases, when the lesion is visible on a radiograph, a large portion of the tooth is already affected, making the treatment extremely difficult, if not impossible. In addition, when one tooth in a patient is affected, it is possible that other teeth may also be affected in the same dentition,[33] making the use of CBCT more justified in these cases (Fig. 5.14). Treatment of these lesions usually includes a combination of nonsurgical and surgical endodontic techniques (such as osteotomy and root repair, with or without intentional replantation) (for more information see Chapter 18).

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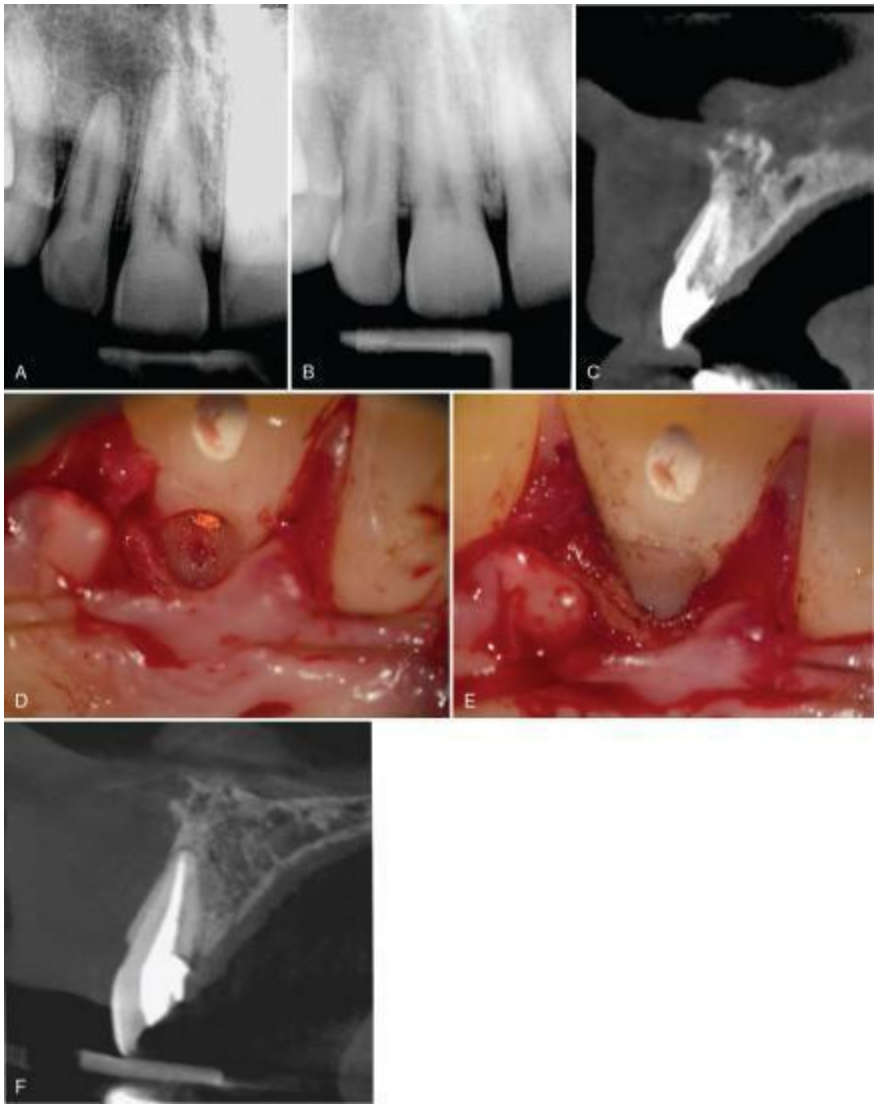

FIG. 5.14 **A,** Invasive cervical resorption of tooth #8 was detected on a periapical radiograph. **B,** A radiograph taken 9 years prior to the current examination. **C,** CBCT provided an accurate view of the location and the extent of the lesion. **D,** During surgery, following root canal therapy, the resorptive defect was excavated. **E,** The surgical site after restoration with Geristore. **F,** A 2-year follow-up CBCT shows that the lesion has not continued to extend.

A) Radiograph shows teeth with radiolucent root canals and low-density tissue surrounding the roots. The tooth at the center has radiolucent patches. Beside this, there is an L-shaped framework below the teeth.

B) Radiograph shows teeth with wedge-shaped radiolucency in the root and slightly dense tissue surrounding the teeth.

C) Radiograph shows the single tooth with translucent dental tissue. It is surrounded by porous tissue.

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D) Close-up view shows central tooth with oval-shaped light patch and a lesion below the root.

E) Close-up view shows tooth with filling at the root.

F) Radiograph shows tooth with filled root canal and filled dental tissue.

The inability to isolate a tooth is also a problem and may result in bacterial contamination of the root canal system. Extra roots and canals pose a particular anatomic challenge that radiographs do not always reveal (Fig. 5.15). A bitewing radiograph is useful in providing an accurate image of the pulp chambers of posterior teeth.

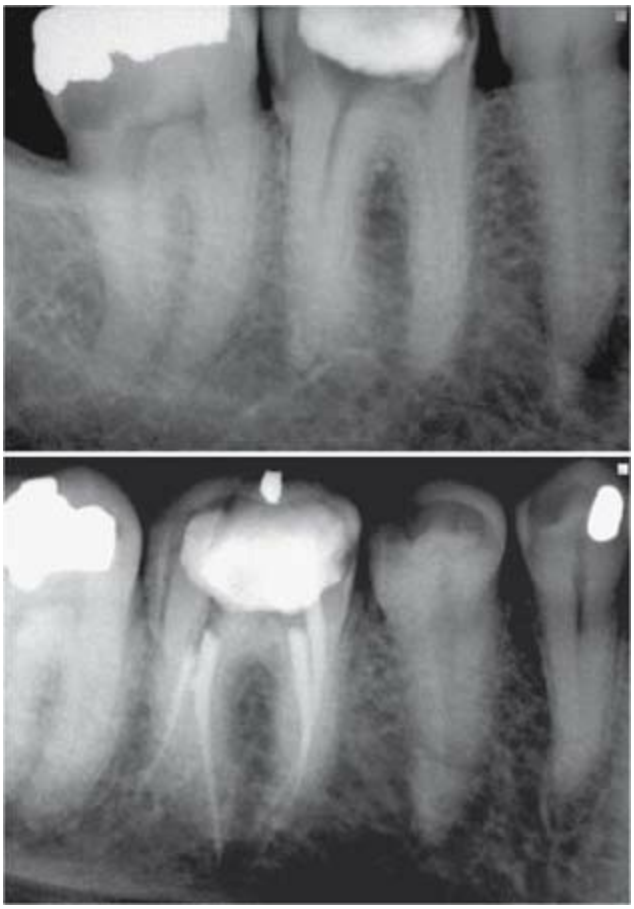

FIG. 5.15 The presence of curved roots and multiple canals is a complicating factor.

Radiograph on the top shows tooth with radiolucency in the dentine below radiopaque enamel and in-between bifurcated root. Radiograph at the bottom shows the same tooth with filled root canals with minor radiolucency at the

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The clinician should recognize these potential problems and be able to manage and factor them into the decision concerning the tooth's prognosis, including the possibility that the patient should be referred to a specialist.

As mentioned, CBCT is a tool that when used judicially can be helpful in identifying roots that are not visible with conventional radiography. For more information on clinical applications of CBCT, refer to the 2015/2016 AAE and American Academy of Oral and Maxillofacial Radiology (AAOMR) Joint Position Statement (available at https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/conebeamstatement.pdf). The statement provides 11 specific recommendations and supporting evidence for when CBCT should be considered, and emphasizes that CBCT should not be routinely used for endodontic diagnosis or screening in the absence of clinical symptoms. Consistent with the principles of ALARA—keeping patient radiation doses "As Low as Reasonably Achievable"—the statement notes that the patient's history and clinical examination must justify the use of CBCT. (Refer to https://f3f142zs0k2w1kg84k5p9i1o-wpengine.netdna-ssl.com/specialty/wp-content/uploads/sites/2/2017/06/conebeamstatement.pdf.)

The revised position statement concludes with the following advisory: "CBCT is an emerging technology that is revolutionizing the approach to endodontic care of dental patients. The guidance in this statement is not intended to substitute for a clinician's independent judgment in patient care. The use of limited field of view (FOV) CBCT should be considered on a case-by-case basis, with due consideration given to the risks and benefits of exposing the patient to ionizing radiation, the patient's history, clinical findings and pre- existing radiographs so that superior treatment can be provided to the general public in need of endodontic care."

Another consideration is the stage of maturity of the tooth. Primary and immature permanent teeth may have a pulpal pathosis caused by caries or trauma; preserving these young teeth is essential. Premature loss of an anterior tooth can lead to malocclusion, predispose the patient to tongue habits, impair aesthetics, and damage the patient's self-esteem. (See Chapter 21 on dental traumatic injuries.)

Some clinicians use a simple formula for determining which endodontic

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cases they treat and which they refer to a specialist. The number of roots may be the determining factor in a decision concerning referral, or the key factor may be the chronic or acute status of the case. Others consider the complexity of the ultimate prosthesis as a factor in considering an endodontic referral. The most important variables in determining whether to refer a patient to a specialist are the skills of the clinician and the complexity of the case. The AAE developed guidelines for assessing endodontic case difficulty (available at https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/10/2006casedifficultyassessmentformb_edited20 The AAE Endodontic Case Difficulty Assessment Form enables a clinician to assign a level of difficulty to a particular case. The form describes cases with minimal, moderate, and high degrees of difficulty. This form lists criteria that can be used to identify cases that should be referred to a specialist. The use of surgical operating microscopes, endoscopes, and ultrasonics enables the specialist to predictably treat teeth that would not previously have been treatable.

Anxiety

Anxiety presents a problem at many levels of dental care. Avoidance of dental treatment due to anxiety appears to be associated with significant deterioration of oral and dental health.[120] Even at the diagnostic stage, severe anxiety may confuse the process.[25] Several studies support the hypothesis that pain or fear of pain is a primary source of anxiety as well as an obstacle to seeking dental care.[50,120] Also, highly anxious patients appear to be more sensitive to pain.[26,49] High levels of anxiety have been found to negatively affect clinical procedures including local anesthesia.[64] In 2009, Binkley tested the hypothesis that having natural red hair color, which is caused by variants of the melanocortin-1 receptor *(MC1R)* gene, could predict a patient's experiencing dental care–related anxiety and dental care avoidance.[10] She found that participants with *MC1R* gene variants reported significantly more dental care–related anxiety and fear of dental pain than did participants with no *MC1R* gene variants. A more recent study confirmed the relationship between the red hair phenotype and anxiety. However, this study found no association between the red hair phenotype and the success of local anesthesia.[23]

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It has been demonstrated that dental anxiety and expectation of pain had a profound effect on a patient's ability to understand information provided.[27] A person's cognitive ability to process information is significantly affected by stress.[27] A study found that 40% of patients who had minor oral surgery did not remember receiving both written and verbal instructions, contributing to 67% noncompliance with antibiotic prescriptions.[11] Patients' anxiety can compromise their understanding of complex treatment plans. Decisions made by a patient concerning options involving tooth retention or loss may be markedly affected by anxiety. In the field of medicine, there strong evidence to show that behavioral intervention for the highly anxious patient before treatment decreases anxiety before and after surgery, reduces posttreatment pain, and accelerates recovery.[45]

Unfortunately, the impact that a high level of anxiety can have on patient's cognition, local anesthesia, and intraoperative and postoperative experiences is not always recognized. A landmark medical study found that pretreatment discussion of surgical treatments and associated discomfort reduced by 50% the need for posttreatment morphine and reduced the time to discharge.[24] Existing research has focused primarily on the effect of pretreatment information on reducing anxiety and stress during surgery.[27]

Calm setting, reassurance by the clinician and explanation of the treatment plan, as well as a discussion about pain prevention strategies are all important steps even before treatment starts.[65] A written description as well as a verbal description of the proposed treatment are helpful. It may also be of value to have a family member or friend accompany the patient for a discussion of the treatment plan.

Scheduling considerations

If a vital case is to be treated by a multivisit approach, it is suggested that the clinician allow 5 to 7 days between canal instrumentation and obturation to allow periradicular tissues to recover. When a vital case is to be treated in a single visit, adequate time must be scheduled so that the clinician can comfortably complete the procedure. Because profound inferior alveolar nerve block anesthesia may require approximately 15 to 20 minutes, it is wise to include that time when scheduling a patient's appointment (see also Chapter 6 on pain control and local anesthesia).

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Appointments to fill nonvital cases should be scheduled approximately 1 week after instrumentation to maximize the antimicrobial effect of the intracanal dressing when calcium hydroxide is used.[2,99,100] Acute (pain or swelling) nonvital cases should be seen every 24 to 48 hours to monitor the patient's progress and bring the acute symptoms under control. Further cleaning and shaping are important components of the treatment as the clinician seeks to eliminate persistent microbes in the canal system. Long delays between visits contribute to the development of resistant microbial strains and should be avoided.

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References

1. American Dental Association Council of Scientific Affairs. *Dental endosseous implants: an update*, J Am Dent Assoc 2004;135: 92.
2. Bergenholtz GH, Hörsted-Bindslev P, Reit C. Textbook of endodontology 2003; Blackwell Hoboken, NJ.
3. Alobaid AS, Cortes LM, Lo J, et al: *Radiographic and clinical outcomes of the treatment of immature permanent teeth by revascularization or apexification: a pilot retrospective cohort study*, J Endod 8, 2014;40: 1063-1070.
4. Scialli AR, Ang R, Breitmeyer J, et al: *Childhood asthma and use during pregnancy of acetaminophen. A critical review*, Reprod Toxicol 4, 2010;30: 508-519.
5. Vernillo AT. *Diabetes mellitus: Relevance to dental treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 263.
6. Bader JD, Bonito AJ, Shugars DA. *A systematic review of cardiovascular effects of epinephrine on hypertensive dental patients*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 648.
7. Bakhtiar H, Esmaeili S, Fakhr Tabatabayi S, et al: *Second-generation platelet concentrate (platelet-rich fibrin) as a scaffold in regenerative endodontics: a case series*, J Endod 3, 2017;43: 401-408.
8. Sanchez-Dominguez B, López-López J, Jané-Salas E, et al: *Glycated hemoglobin levels and prelalence of apical periodontitis in type 2 diabetic patients*, J Endod 2015;41: 601-606.
9. Bernstein SD, Horowitz AJ, Man M, et al: *Outcomes of*

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www.konkur.in

endodontic therapy in general practice: a study by the practitioners engaged in applied research and learning network, J Am Dent Assoc 2012;43: 478.

10. Binkley CJ, Beacham A, Neace W, et al: *Genetic variations associated with red hair color and fear of dental pain, anxiety regarding dental care and avoidance of dental care*, J Am Dent Assoc 2009;140: 896.
11. Blinder D, Rotenberg L, Peleg M, et al: *Patient compliance to instructions after oral surgical procedures,* Int J Oral Maxillofac Surg 2001;30: 216.
12. Brennan MT, Valerin MA, Noll JL, et al: *Aspirin use and post-operative bleeding from dental extractions*, J Dent Res 2008;87: 740.
13. Briggs GG, Freeman RK, Yaffe SJ. Drugs in pregnancy and lactation: a reference guide in fetal and neonatal risk ed 8 2009; Lippincott Williams & Wilkins Philadelphia.
14. Brown RS, Rhodus NL. *Epinephrine and local anesthesia revisited*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 401.
15. Burket LW, Greenberg MS, Glick M, et al: Burket's oral medicine ed 11 2008; PMPH Six Forks, NC.
16. Tibúrcio-Machado CD, Bello MC, Maier J, et al: *Influence of diabetes in the development of apical periodontitis: a critical literature review of human studies*, J Endod 2017;43: 370-376.
17. Carter AE, Carter G, George R. *Pathways of fear and anxiety in endodontic patients*, Int Endod 6, 2015;48: 528-532.
18. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J Endod 2007;33: 1121.
19. Creugers NH, Kreulen CM, Snoek PA, et al: *A systematic*

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review of single-tooth restorations supported by implants, J Dent 2000;28: 209.
20. de Chevigny C, Dao TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study—phase 4: initial treatment*, J Endod 2008;34: 258.
21. Debelian GJ, Olsen I, Tronstad L. *Bacteremia in conjunction with endodontic therapy*, Endod Dent Traumatol 1995;11: 142.
22. Diogenes A, Ruparel NB, Shiloah Y, et al: *Regenerative endodontics: A way forward*, J Am Dent Assoc 5, 2016;147: 372-380.
23. Droll B, Drum M, Nusstein J, et al: *Anesthetic efficacy of the inferior alveolar nerve block in red-haired women*, J Endod 2012;38: 1564.
24. Egbert LD, Battit GE, Welch CS, et al: *Reduction of postoperative pain by encouragement and instruction of patients. a study of doctor-patient rapport*, N Engl J Med 1964;270: 825.
25. Eli I. *Dental anxiety: a cause for possible misdiagnosis of tooth vitality*, Int Endod 1993;26: 251.
26. Eli I, Schwartz-Arad D, Baht R, et al: *Effect of anxiety on the experience of pain in implant insertion*, Clin Oral Implants Res 2003;14: 115.
27. Eli I, Schwartz-Arad D, Bartal Y. *Anxiety and ability to recognize clinical information in dentistry*, J Dent Res 2008;87: 65.
28. Estrela C, Bueno MR, De Alencar AHG, et al: *Method to evaluate inflammatory root resorption by using cone beam computed tomography*, J Endod 11, 2009;35: 1491-1497.
29. Figini L, Lodi G, Gorni F, et al: *Single versus multiple visits for endodontic treatment of permanent teeth*, J Endod 2008;34: 1041.

Telegram: @dental_k

www.konkur.in

30. Fouad AF, Burleson J. *The effect of diabetes mellitus on endodontic treatment outcome: data from an electronic patient record*, JADA 2003;134: 43-51.
31. Gatten DL, Riedy CA, Hong SK, et al: *Quality of life of endodontically treated versus implant treated patients: a university-based qualitative research study*, J Endod 2002;37: 903.
32. Gaur AH, Dominguez KL, Kalish ML, et al: *Practice of feeding premasticated food to infants: a potential risk factor for HIV transmission*, Pediatrics 2009;124: 658.
33. Heithersay GS. *Clinical, radiologic, and histopathologic features of invasive cervical resorption*, Quintessence Int 1999;30: 27-37.
34. Gillen BM, Looney SW, Gu LS, et al: *Impact of the quality of coronal restoration versus the quality of root canal fillings on success of root canal treatment: a systematic review and meta-analysis*, J Endod 2011;37: 865.
35. Goodchild JH, Glick M. *A different approach to medical risk assessment*, Endod Top 2003;4: 1.
36. Imura N, Zuolo M. *Factors associated with endodontic flare-ups: a prospective study*, Int Endod 1995;28: 261.
37. Interrante JD, Ailes EC, Lind JN, et al: *Risk comparison for prenatal use of analgesics and selected birth defects, national birth defects prevention study 1997–2011*, Ann Epidemiol 10, 2017;27: 645-653.e642.
38. Iqbal MK, Kim S. *A review of factors influencing treatment planning decisions of single-tooth implants versus preserving natural teeth with nonsurgical endodontic therapy*, J Endod 2008;34: 519.
39. Javed F, Romanos GE. *Impact of diabetes mellitus and glycemic control on the osseointegration of dental*

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www.konkur.in

implants: a systematic literature review, J Periodontol 2009;80: 1719.

40. Jeeruphan T, Jantarat J, Yanpiset K, et al: *Mahidol study 1: comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: a retrospective study*, J Endod 10, 2012;38: 1330-1336.
41. Jeske AH, Suchko GD. *Lack of a scientific basis for routine discontinuation of oral anticoagulation therapy before dental treatment*, J Am Dent Assoc 2003;134: 1492.
42. Siqueira JF. *Endodontic infections: concepts, paradigms, and perspectives*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 281.
43. Lee JM, Shin TJ. *Use of local anesthetics for dental treatment during pregnancy; safety for parturient*, J Dent Anesth Pain Med 2, 2017;17: 81-90.
44. Kahler B, Rossi-Fedele G, Chugal N, et al: *An evidence-based review of the efficacy of treatment approaches for immature permanent teeth with pulp necrosis*, J Endod 7, 2017;43: 1052-1057.
45. Kiecolt-Glaser JK, Page GG, Marucha PT, et al: *Psychological influences on surgical recovery: perspectives from psychoneuroimmunology*, Am Psychol 1998;53: 1209-1218.
46. Kim E, Song JS, Jung IY, et al: *Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal-endodontic origin*, J Endod 2008;34: 546.
47. Kim SG, Malek M, Sigurdsson A, et al: *Regenerative endodontics: a comprehensive review*, Int Endod J 12,

Telegram: @dental_k

www.konkur.in

2018;51: 1367-1388.
48. Kim TS, Caruso JM, Christensen H, et al: *A comparison of cone-beam computed tomography and direct measurement in the examination of the mandibular canal and adjacent structures*, J Endod 2010;36: 1191.
49. Klages US, Kianifard S, Ulusoy O, et al: *Anxiety sensitivity as predictor of pain in patients undergoing restorative dental procedures*, Community Dent Oral Epidemiol 2006;34: 139.
50. Lahmann C, Schoen R, Henningsen P, et al: *Brief relaxation versus music distraction in the treatment of dental anxiety: a randomized controlled clinical trial*, J Am Dent Assoc 2008;139: 317.
51. Little JW, Miller CS, Rhodus NL. Dental management of the medically compromised patient ed 9 2018; Elsevier St. Louis.
52. Fava LR. *One-appointment root canal treatment: incidence of postoperative pain using a modified double-flared technique*, Int Endod J 1991;24: 258.
53. Mainkar A. *A systematic review of the survival of teeth intentionally replanted with a modern technique and cost-effectiveness compared with single-tooth implants*, J Endod 2017;43: 1963.
54. Markiewicz MR, Margarone JE, Campbell JH, et al: *Bisphosphonate-associated osteonecrosis of the jaws: a review of current knowledge*, J Am Dent Assoc 2005;136: 1669.
55. Marotta PS, Fontes TV, Armada L, et al: *Type 2 diabetes mellitus and the prevalence of apical periodontitis and endodontic treatment in an adult Brazilian population*, J Endod 2012;38: 297.
56. McDaniel RK, Luna MA, Stimson PG. *Metastatic tumors*

Telegram: @dental_k

www.konkur.in

in the jaws, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1971;31: 380.

57. Melo MD, Obeid G. *Osteonecrosis of the jaws in patients with a history of receiving bisphosphonate therapy: strategies for prevention and early recognition*, J Am Dent Assoc 2005;136: 1675.
58. Michalowicz BS, DiAngelis AJ, Novak MJ, et al: *Examining the safety of dental treatment in pregnant women*, J Am Dent Assoc 2008;139: 685.
59. Migliorati CA, Casiglia J, Epstein J, et al: *Managing the care of patients with bisphosphonate-associated osteonecrosis: an American Academy of Oral Medicine position paper*, J Am Dent Assoc 2005;136: 1658.
60. Miley DD, Terezhalmy GT. *The patient with diabetes mellitus: etiology, epidemiology, principles of medical management, oral disease burden, and principles of dental management*, Quintessence Int 2005;36: 779.
61. Mitchell AA, Gilboa SM, Werler MM, et al: *Medication use during pregnancy, with particular focus on prescription drugs: 1976-2008*, Am J Obstet Gynecol 1, 2011;205: 51 e51-58.
62. Moinzadeh AT, Shemesh H, Neircynk NA, et al: *Bisphosphonates and their clinical implications in endodontic therapy*, Int Endod 2012;46: 391.
63. Molander A, Warfvinge J, Reit C, et al: *Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical periodontitis: a randomized clinical trial*, J Endod 2007;33: 1145.
64. Nakai Y, Milgrom P, Mancl L, et al: *Effectiveness of local anesthesia in pediatric dental practice*, J Am Dent Assoc 2000;131: 1699.

Telegram: @dental_k

www.konkur.in

65. Ng SK, Chau AW, Leung WK. *The effect of pre-operative information in relieving anxiety in oral surgery patients*, Community Dent Oral Epidemiol 2004;32: 227.
66. Ng YL, Glennon JP, Setchell DJ, et al: *Prevalence of and factors affecting post-obturation pain in patients undergoing root canal treatment*, Int Endod 2004;37: 381.
67. Ng YL, Mann V, Gulabivala K. *Outcome of secondary root canal treatment: a systematic review of the literature*, Int Endod J 2008;41: 1026.
68. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2: tooth survival*, Int Endod J 2011;44: 610.
69. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment—part 1: periapical health*, Int Endod J 2011;44: 583.
70. Ng YL, Mann V, Gulabivala K. *Tooth survival following non-surgical root canal treatment: a systematic review of the literature*, Int Endod J 2010;43: 171.
71. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature—Part 1. Effects of study characteristics on probability of success*, Int Endod J 2007;40: 921.
72. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature—Part 2. Influence of clinical factors*, Int Endod J 2008;41: 6.
73. Nishimura RA, Otto CM, Bonow RO, et al: *2017 AHA/ACC focused update of the 2014 AHA/ACC guidelines for the management of patients with valvular heart disease: a report of the American College of*

Telegram: @dental_k

www.konkur.in

Cardiology/American Heart Association Task Force on Clinical Practice, Circulation 2017;135: 115.

74. Moore PA. *Selecting drugs for the pregnant dental patient*, J Am Dent Assoc 1998;129: 1281.
75. Pallasch TJ, Slots J. *Antibiotic prophylaxis and the medically compromised patient*, Periodontol 1996;10: 107.
76. Patel K, Mannocci F, Patel S. *The assessment and management of external cervical resorption with periapical radiographs and cone-beam computed tomography: a clinical study*, J Endod 10, 2016;42: 1435-1440.
77. Penesis VA, Fitzgerald PI, Fayad MI, et al: *Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: a randomized controlled trial with one-year evaluation*, J Endod 2008;34: 251.
78. Persky V, Piorkowski J, Hernandez E, et al: *Prenatal exposure to acetaminophen and respiratory symptoms in the first year of life*, Ann Allergy Asthma Immunol 2008;101: 271.
79. Peters LB, Wesselink PR. *Periapical healing of endodontically treated teeth in one and two visits obturated in the presence or absence of detectable microorganisms*, Int Endod 2002;35: 660.
80. Quesnell BT, Alves M, Hawkinson RW Jr, et al: *The effect of human immunodeficiency virus on endodontic treatment outcome*, J Endod 2005;31: 633.
81. Rafter M. *Apexification: a review*, Dent Traumatol 1, 2005;21: 1-8.
82. Rayburn WF, Amanze AC. *Prescribing medications safely during pregnancy*, Med Clin North Am 2008;92: 1227.
83. Rhodus NL, Vibeto BM, Hamamoto DT. *Glycemic*

Telegram: @dental_k

www.konkur.in

control in patients with diabetes mellitus upon admission to a dental clinic: considerations for dental management, Quintessence Int 2005;36: 474.

84. Roane JB, Dryden JA, Grimes EW. *Incidence of postoperative pain after single- and multiple-visit endodontic procedures*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1983;55: 68.
85. Rotstein I, Simon JH. *The endo-perio lesion: a critical appraisal of the disease condition*, Endod Top 2006;13: 34.
86. Friedman S. *Considerations and concepts of case selection in the management of post-treatment endodontic disease (treatment failure)*, Endod Top 2002;1: 54.
87. Friedman S. *Prognosis of initial endodontic therapy*, Endod Top 2002;1: 54.
88. Salehrabi R, Rotstein I. *Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study*, J Endod 2004;30: 846.
89. Salehrabi R, Rotstein I. *Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment*, J Endod 2010;36: 790.
90. Ruggiero SL, Dodson TB, Fantasia J, et al: *American Association of Oral and Maxillofarial Sugeons position paper on medication-related osteonecrosis of the jaw – 2014 Update*, J Oral Maxillofac Surg 10, 2014;72:.
91. Sathorn C, Parashos P, Messer HH. *Effectiveness of single- versus multiple-visit endodontic treatment of teeth with apical periodontitis: a systematic review and meta-analysis*, Int Endod J 2005;38: 347.
92. Sathorn C, Parashos P, Messer HH. *The prevalence of postoperative pain and flare-up in single- and multiple-visit endodontic treatment: a systematic review*, Int Endod

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www.konkur.in

J 2008;41: 91.
93. White SC. *1992 assessment of radiation risk from dental radiography*, Dentomaxillofac Radiol 1992;21: 118.
94. Scully C, Cawson RA. Medical problems in dentistry ed 5 2005; Churchill Livingstone Edinburgh.
95. Setzer FC, Boyer KR, Jeppson JR, et al: *Long-term prognosis of endodontically treated teeth: a retrospective analysis of preoperative factors in molars*, J Endod 2011;37: 21.
96. Setzer FC, Kohli MR, Shah SB, et al: *Outcome of endodontic surgery: a meta-analysis of the literature—Part 2: comparison of endodontic microsurgical techniques with and without the use of higher magnification*, J Endod 2012;38: 1.
97. Setzer FC, Shah SB, Kohli MR, et al: *Outcome of endodontic surgery: a meta-analysis of the literature—part 1: comparison of traditional root-end surgery and endodontic microsurgery*, J Endod 2010;36: 1757.
98. Simonis P, Dufour T, Tenenbaum H. *Long-term implant survival and success: a 10–16-year follow-up of non-submerged dental implants*, Clin Oral Impl Res 7, 2010;21: 772-777.
99. Sjögren U, Figdor D, Perrson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int Endod J 1997;30: 297.
100. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498.
101. Skaar D, O'Connor H, Lunos S, et al: *Dental procedures and risk of experiencing a second vascular event in a medicare population*, J Am Dent Assoc 2012;143: 1190.

Telegram: @dental_k

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102. Smidt A, Lipovetsky-Adler M, Sharon E. *Forced eruption as an alternative to tooth extraction in long-term use of oral bisphosphonates: review, risks and technique*, J Am Dent Assoc 2012;143: 1303.
103. Sollecito TP, Abt E, Lockhart PB, et al: *The use of prophylactic antibiotics prior to dental procedures in patients with prosthetic joints: evidence-based clinical practice guideline for dental practitioners—a report of the American Dental Association Council on Scientific Affairs*, J Am Dent Assoc 1, 2015.e18;146: 11-16.
104. Su Y, Wang C, Ye L. *Healing rate and post-obturation pain of single- versus multiple-visit endodontic treatment for infected root canals: a systematic review*, J Endod 2011;37: 125.
105. Arya S, Duhan J, Tewari S, et al: *healing of apical periodontitis after nonsurgical treatment in patients with type 2 diabetes*, J Endod 2017;43: 1623-1627.
106. Saoud TM, Martin G, Chen YH, et al: *Treatment of mature permanent teeth with necrotic pulps and apical periodontitis using regenerative endodontic procedures: a case series*, J Endod 1, 2016;42: 57-65.
107. Torabinejad M, Goodacre CJ. *Endodontic or dental implant therapy: the factors affecting treatment planning*, J Am Dent Assoc 2006;137: 973.
108. Moraschini V, Poubel LA, Ferreira VF, et al: *Evaluation of survival and success rates of dental implants reported in longitudinal studies with a follow-up period of at least 10 years: a systematic review*, Int J Oral Maxillofac Surg 3, 2015;44: 377-388.
109. Vahdati SA, Torabinejad M, Handysides R, et al: *A retrospective comparison of outcome in patients who received both nonsurgical root canal treatment and single-*

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www.konkur.in

tooth implants, J Endod 2, 2019;45: 99-103.

110. Vera J, Siqueira JF, Ricucci D, et al: *One- versus two-visit endodontic treatment of teeth with apical periodontitis: a histobacteriologic study*, J Endod 2012;38: 1040.
111. von Arx T, Jensen SS, Hanni S. *Clinical and radiographic assessment of various predictors for healing outcome 1 year after periapical surgery*, J Endod 2007;33: 123.
112. von Arx T, Jensen SS, Hanni S. *Five-year longitudinal assessment of the prognosis of apical microsurgery*, J Endod 2012;38: 570.
113. Wang CH, Chueh LH, Chen SC, et al: *Impact of diabetes mellitus, hypertension, and coronary artery disease on tooth extraction after nonsurgical endodontic treatment*, J Endod 2011;37: 1.
114. Wang N, Knight K, Dao T, et al: *Treatment outcome in endodontics—The Toronto study. Phases I and II: apical surgery*, J Endod 2004;30: 751.
115. Wang Q, Cheung GSP, Ng RPY. *Survival of surgical endodontic treatment performed in a dental teaching hospital: a cohort study*, Int Endod 2004;37: 764.
116. Weiger R, Rosendahl R, Lost C. *Influence of calcium hydroxide intracanal dressings on the prognosis of teeth with endodontically induced periapical lesions*, Int Endod 2000;33: 219.
117. Woodmansey KF, Ayik M, Buschang PH, et al: *Differences in masticatory function in patients with endodontically treated teeth and single-implant-supported prostheses: a pilot study*, J Endod 2009;35: 10.
118. Wynn RL, Meiller TF, Crossley HL. Drug information handbook for dentistry: including oral medicine for

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www.konkur.in

medically-compromised patients & specific oral conditions ed 18 2012; Lexi-Comp Hudson, OH.

119. Xu J, Murphy SL, Kochanek KD, et al: *Deaths: Preliminary data for 2016*, Natl Vital Stat Rep 2018;67: 5.

120. Yu SM, Bellamy HA, Kogan MD, et al: *Factors that influence receipt of recommended preventive pediatric health and dental care*, Pediatrics 2002;110: 73.

121. Zuolo ML, Ferreira MO, Gutmann JL. *Prognosis in periradicular surgery: a clinical prospective study*, Int Endod J 2000;33: 91.

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6: Pain control*

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CHAPTER OUTLINE

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Local anesthesia

Effective local anesthesia is the foundation of pain control in dentistry and especially endodontics. Regardless of the clinician's skills, both treatment and patient management are difficult or impossible to deliver without effective pain control. This chapter reviews the pharmacology of local anesthetics and the relative advantages and limitations of various anesthetics and routes of administration. Other chapters in this book provide complementary information on the use of local anesthetics in diagnosis (see Chapter 1) and the treatment of emergency patients (see Chapter 19). The authors assume that the reader is familiar with various anesthetic injection techniques; several excellent texts are available for review regarding this topic.[165,213,277]

Mechanisms of action for anesthetics

Most dental pharmacology courses teach that local anesthetics block sodium channels by partitioning into two types: the uncharged basic form of the molecule (RN), which crosses cell membranes, and the charged acid form of the molecule (RNH^+), which binds to the inner pore of the sodium channel. As a first approximation, this model is reasonably accurate. However, molecular research has demonstrated the existence of at least nine subtypes of voltage-gated sodium channels (VGSCs) that differ in their expression pattern, biophysical properties, and roles in mediating peripheral pain (Table 6.1). These channels have a clear clinical relevance.[30,146,198] Indeed, several groups of patients have been described with genetic mutations to a VGSC, with significant reported effects on pain sensitivity.

Table 6.1

Voltage-Gated Sodium Channels and Pain

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Channel Subtype	Tissue Expression	Tetrodotoxin Sensitive	Peripheral Role in Pain
Na_v 1.1	Central nervous system (CNS), sensory neurons	Yes	?
Na_v 1.2	CNS	Yes	No
Na_v 1.3	CNS	Yes	No
Na_v 1.4	Muscle	Yes	No
Na_v 1.5	Heart	Somewhat	No
Na_v 1.6	CNS, sensory neurons	Yes	?
Na_v 1.7	CNS, sensory neurons	Yes	?
Na_v 1.8	Sensory neurons	No	Yes
Na_v 1.9	Sensory neurons	No	Yes

The broad class of VGSCs can be divided into channels that are blocked by a toxin (tetrodotoxin [TTX]) and those that are resistant to the toxin (TTX-R). Most TTX-R channels are found primarily on nociceptors (e.g., Na_v 1.8 and Na_v 1.9).[349] These channels also are relatively resistant to local anesthetics and are sensitized by prostaglandins (PGs).[127] As explained later in this chapter, the presence of TTX-R sodium channels may explain why local anesthetics are less effective when they are administered to patients with odontalgia. Many of the adverse effects of local anesthetics are attributed to their ability to block other VGSCs expressed in the central nervous system (CNS) or heart (see Table 6.1).

VGSCs consist of an alpha and a beta subunit. The alpha subunit serves as a voltage sensor, leading to channel activation and sodium ion passage when the channel detects an electrical field. The biologic basis for an electric pulp tester (EPT), therefore, is the generation of a small electrical field across the dental pulp that can activate VGSCs.[146] Interestingly, sensitization of TTX-R channels by PGs lowers the activation threshold and increases the number of sodium ions that flow through the channel.[129] Put another way, an inflammation-induced elevation in PG levels sensitizes TTX-R channels, leading to greater activation with weaker stimuli. This may explain the increased responsiveness to electrical pulp testing seen in patients with irreversible pulpitis.

Local anesthetics have other mechanisms that may contribute to their pharmacology for treating odontogenic pain. For example, local anesthetics modulate certain G protein–coupled receptors (GPCRs). The GPCRs are a major class of cell membrane receptors, and many classes of dental drugs

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(e.g., opioids, catecholamines) and endogenous mediators produce their effects by activating specific GPCRs and their related second messenger pathways. Studies suggest that local anesthetics inhibit the G-alpha-q (Gaq) class of GPCRs, which includes receptors activated by inflammatory mediators such as bradykinin.[157] A local anesthetic may therefore block the actions of a major hyperalgesic agent.

Other studies have indicated that local anesthetics potentiate the actions of the G-alpha-i (Gai) class of GPCRs.[22] This could have a major effect in potentiating the actions of vasoconstrictors, including the newly recognized analgesic role that vasoconstrictors play in inhibiting pulpal nociceptors.[33,145] Prolonged alteration of GPCR function might explain why analgesia obtained with long-acting local anesthetics persists well beyond the period of anesthesia.[72,91,237] More research is needed on this exciting aspect of local anesthetic pharmacology.

Clinically available local anesthetics

The most common forms of injectable local anesthetics are in the amide class. In 2003, the American Dental Association specified a uniform color code for dental cartridges to prevent confusion among brands (Table 6.2). Local anesthetics can be divided roughly into three types: short duration (30 minutes of pulpal anesthesia), intermediate duration (60 minutes of pulpal anesthesia), and long duration (over 90 minutes of pulpal anesthesia). However, clinical anesthesia does not always follow these guidelines, as it depends on whether the local anesthetic is used as a block or for infiltration. For example, bupivacaine is classified as a long-acting agent; when it is used in an inferior alveolar nerve (IAN) block, this is true.[98] However, when it is used for infiltration for anterior teeth, it has a shorter duration of anesthetic action than 2% lidocaine with 1:100,000 epinephrine[74,134] (this is discussed in more detail later in this chapter).

Table 6.2

Local Anesthetics Available in the United States*

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Anesthetic	Vasoconstrictor	Dental Cartridge Color Codes†	Maximum Allowable Dose	Typical Maximum Dose
2% Lidocaine	1:100,000 epinephrine	Red	13	8
2% Lidocaine	1:50,000 epinephrine	Green	13	8
2% Lidocaine	Plain (no vasoconstrictor)	Light blue	8	8
2% Mepivacaine	1:20,000 levonordefrin	Brown	11	8
3% Mepivacaine	Plain (no vasoconstrictor)	Tan	7	5½
4% Prilocaine	1:200,000 epinephrine	Yellow	5½	5½
4% Prilocaine	Plain (no vasoconstrictor)	Black	5½	5½
0.5% Bupivacaine	1:200,00 epinephrine	Blue	10	10
4% Articaine	1:100,000 epinephrine	Gold	7	7
4% Articaine	1:200,000 epinephrine	Silver	7	7

*This table provides the maximum dosage in two formats. The maximum allowable dose is generally approached only with complex oral and maxillofacial surgical procedures. The typical maximum dose is the usual outer envelope of drug dosage for most endodontic, surgical, and restorative dental procedures. Both columns show the number of cartridges that would be required for an adult weighing 67.5 kg (150 pounds).

†Uniform dental cartridge color codes were mandated by the American Dental Association in June 2003.

Selection of a local anesthetic: Possible adverse effects and medical history

Possible adverse effects

Possible adverse reactions to local anesthetics can be divided into six major categories: cardiovascular reactions, systemic effects, methemoglobinemia, peripheral nerve paresthesia, allergic reactions to the anesthetic and/or latex, and reactions to anesthetics containing a sulfite antioxidant. These reactions range from fairly common (e.g., tachycardia after intraosseous [IO] injection of 2% lidocaine with 1:100,000 epinephrine) to extremely rare (e.g., allergic reactions to lidocaine).

Cardiovascular reactions

Although classic research studies have reported that large dosages or intravenous injections of local anesthetics were required to produce cardiovascular effects,[163,336] it now is well recognized that even comparatively small amounts of epinephrine can induce measurable

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tachycardia after nerve block or IO injection.[99,129,282] Several authors have reported increases in heart rate with infiltration injections and nerve blocks using 2% lidocaine with 1:100,000 epinephrine[2,149,195,298,335]; others have reported that no significant changes in heart rate occurred or that the changes were clinically insignificant.[228,330,338] When specific information was given on dosing and heart rate increases, several studies found mean heart rate increases.[2,149,194,335] Two studies found increases on average of about 4 beats/min with approximately 20 µg of epinephrine[149,194]; three studies recorded increases of 10 to 15 beats/min with 45 to 80 µg of epinephrine[2,194,298]; and one study found increases of approximately 21 beats/min using 144 µg.[335] Increasing the amount of epinephrine in an infiltration or block injection, therefore, increases the likelihood of an elevated heart rate.

Tachycardia after injection is primarily a pharmacologic effect. The cardiovascular effects are the result of alpha-adrenoceptor stimulation by systemic distribution of the vasoconstrictor throughout the vascular compartment. The patient may also report heart palpitations associated with anxiety or fear and may experience transient tachycardia and changes in blood pressure. Large doses or inadvertent intravenous injection may lead to lidocaine toxicity and CNS depression.[100,244] To reduce this risk, the clinician should always aspirate before making the injection, inject slowly, and use dosages within accepted guidelines. The maximal dosages for local anesthetics are listed in Table 6.2.

Systemic effects

Acute toxicity from an overdose of a local anesthetic is often the result of inadvertent intravenous administration or of a cumulative large dose (e.g., repeated injections). As shown in Table 6.1, VGSCs are found in the CNS and the myocardium, the two major sites of anesthetic-induced toxicity. Although systemic effects from a local anesthetic are rare, they can include an initial excitatory phase (e.g., muscle twitching, tremors, grand mal convulsions) and a subsequent depressive phase (e.g., sedation, hypotension, and respiratory arrest).[78,100] It should be noted that symptomatic management (possibly including cardiopulmonary resuscitation [CPR], airway support, and supplemental oxygen) is the primary response to this adverse

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event.[191,193] An acute hypotensive crisis with respiratory failure has also been interpreted as the result of hypersensitivity to local anesthetics[55]; these patients should be evaluated with allergy testing. To reduce the risk of systemic effects from anesthetics, the clinician must always aspirate before giving the injection and must use dosages within accepted guidelines (see Table 6.2). Finder and Moore[100] proposed a "rule of 25" as a simple means of remembering maximal local anesthetic dosages: with currently formulated local anesthetic cartridges, it is generally safe to use one cartridge of local anesthetic for every 25 pounds of patient weight (e.g., six cartridges for a patient weighing 150 pounds [67.5 kg]).

Methemoglobinemia

The metabolism of certain local anesthetics (e.g., prilocaine, benzocaine, articaine, and to a lesser extent lidocaine) can produce a metabolite that causes methemoglobinemia; this effect often occurs several hours after injection of the local anesthetic.[214,354] Typical signs and symptoms include cyanosis, dyspnea, emesis, and headache. In a study on benzocaine-induced methemoglobinemia, 67% of reported adverse effects with benzocaine were associated with methemoglobinemia; of these events, 93% occurred with spray formulations and only one case involved the gel formulation.[239] To reduce the risk of methemoglobinemia, clinicians should take care to refrain from giving excessive dosages of local anesthetics.

Peripheral nerve paresthesia

Postinjection paresthesia is a rare adverse effect of local anesthetics.[136,214,365] The incidence of paresthesia (which involved the lip and/or tongue) associated with articaine and prilocaine was higher than that found with either lidocaine or mepivacaine.[114,121,136] Another study evaluated patients referred with a diagnosis of damage to the inferior alveolar and/or lingual nerve that could only have resulted from an IAN block; it was found that 35% were caused by lidocaine and 30% by articaine.[272] The conclusion was that there was not a disproportionate involvement from articaine, although this interpretation does not account for large differences in clinical use of the two solutions. This same author published a paper on IAN block involvement with the following proportions: 25% lidocaine, 33% articaine, and 34%

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prilocaine.[271] Another investigative group also noted more occurrences of paresthesia with articaine and prilocaine.[269] Paresthesia can be caused by any anesthetic agent. Since paresthesia is noted in the literature to be higher with articaine and because articaine does not have higher pulpal anesthetic success rates when given as an IAN block, articaine use may be best reserved for infiltrations. With any alteration in nerve sensation, documentation of the area of altered sensation (patient reported and clinically tested), the type of altered sensation (e.g., anesthesia, paresthesia, dysesthesia), and regular follow-up are important.[371]

Allergic reactions

The amide local anesthetics appear to have little immunogenicity and therefore have an extremely low rate of true allergic reactions,[20,244,303,292] but case reports of hypersensitivity reactions after the administration of local anesthetics have been published.[32,55,141,240,303,310,318] Referral for allergy testing (with dental anesthetic cartridges) or deep sedation may be indicated in patients with true allergic reactions (anaphylaxis, etc.).[303]

Local anesthetic formulations that contain vasoconstrictors also contain a sulfite to prevent oxidation of the agent. The reaction to sulfites in dental anesthetics may be different from the reaction caused by foods.[318] This reaction is unlikely to occur, but the use of local anesthetics without vasoconstrictors is a possible alternative in patients with severe sulfite allergies.

Effects of systemic diseases or conditions on local anesthetics

It has been stated that vasoconstrictors should be avoided in patients with high blood pressure (higher than 180 mm Hg systolic or 120 mm Hg diastolic), cardiac dysrhythmias, unstable angina, less than 6 months since myocardial infarction or cerebrovascular accident, or severe cardiovascular disease.[213] However, these conditions are contraindications to *routine* dental treatment.

Local anesthetics may interact with a patient's medications, so a thorough review of the medical history is an absolute requirement. Potential drug-drug

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interactions occur primarily with the vasoconstrictors in local anesthetic formulations (Table 6.3). Judicious use of local anesthetic solutions without vasoconstrictors (e.g., 3% mepivacaine) is a reasonable alternative for those adult patients.

Table 6.3

Possible Drug Interactions With Vasoconstrictors

Drugs	Possible Adverse Effects	Recommendations
TRICYCLIC ANTIDEPRESSANTS		
Amitriptyline, doxepin	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
NONSELECTIVE BETA BLOCKERS		
Nadolol, propranolol	Hypertension, bradycardia	Reduce or eliminate vasoconstrictors
RECREATIONAL DRUGS		
Cocaine	Hypertension, myocardial infarction, dysrhythmias	Instruct patient to abstain from drug use for 48 h before procedure; do not use vasoconstrictors
COMT INHIBITORS		
Entacapone, tolcapone	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
ANTIADRENERGIC DRUGS		
Guanadrel, guanethidine	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
NONSELECTIVE ALPHA-ADRENERGIC BLOCKERS		
Chlorpromazine, clozapine, haloperidol	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
DIGITALIS		
Digoxin	Dysrhythmias (especially with large dosage of vasoconstrictor)	Reduce or eliminate vasoconstrictor
HORMONE		
Levothyroxine	Dysrhythmias (especially with large dosage of vasoconstrictor)	*Euthyroid:* No precaution *Hyperthyroid:* Reduce or eliminate vasoconstrictors
MONOAMINE OXIDASE INHIBITORS		
Furazolidone, linezolid, selegiline, tranylcypromine	No interaction	None

COMT, Catecholamine *O*-methyl transferase.

Modified from Naftalin L, Yagiela JA: Vasoconstrictors: indications and precautions, *Dent Clin North Am* 46:733, 2002.

Many of the commonly available local anesthetics are safe for use in pregnant or lactating women.[137] The US Food and Drug Administration (FDA) has classified lidocaine and prilocaine as category B and mepivacaine, bupivacaine, and articaine as category C. The point in pregnancy (trimester)

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and emergent need for care need to be considered. The most important aspect of care with pregnant patients is to eliminate the source of pain by performing the indicated endodontic treatment.[137]

Clinical factors

Traditional methods of confirming anesthesia usually involve questioning the patient, probing the soft tissue, and/or starting treatment and waiting for the patient's reaction. These approaches may not be effective for determining pulpal anesthesia.[53,154,225,340]

Anesthesia in asymptomatic vital teeth can be measured more objectively by applying a cold refrigerant (Fig. 6.1) or by using an EPT, as described in Chapter 1 (Fig. 6.2). Application of cold or the EPT can be used to test the tooth under treatment for pulpal anesthesia before a clinical procedure is started.[49,88,170,198,210]

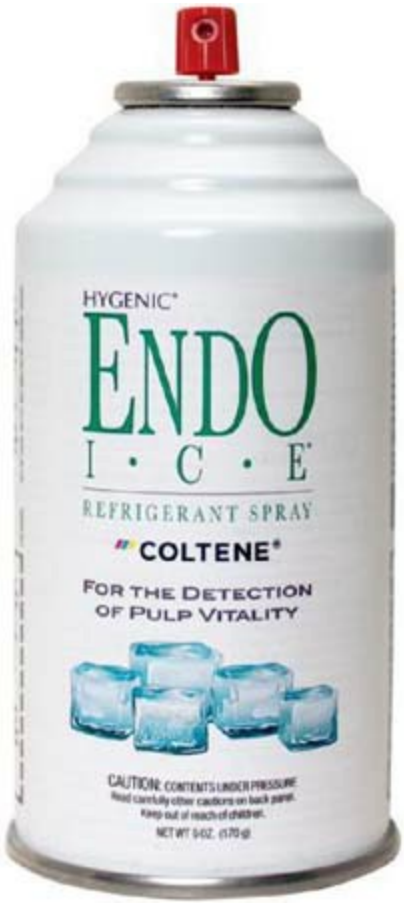

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FIG. 6.1 A cold refrigerant may be used to test for pulpal anesthesia before the start of a clinical procedure.

The bottle of a cold refrigerant named Endo Ice made by colleen is shown that is used for the detection of pulp vitality. Text on the bottle reads, hygienic ice refrigerant spray.

Source: (Courtesy Coltène/Whaledent Inc., Cuyahoga Falls, OH.)

FIG. 6.2 An electric pulp tester also may be used to test for pulpal anesthesia before a clinical procedure is started.

A wired vitality scanner manufactured by SybronEndo with a probe. Display reads, 06.

Source: (Courtesy SybronEndo, Corporation Orange, CA.)

In symptomatic vital teeth (after administration of a local anesthetic), testing with a cold refrigerant or an EPT can also be used to evaluate pulpal anesthesia before starting a treatment.[62,88,253,357] If there is a positive response to testing, supplemental anesthesia will be necessary. However, in these particular patients, a negative response does not guarantee pulpal anesthesia,[92,259,349] and supplemental anesthesia may still be necessary. If the pulp chamber contains necrotic tissue and the canals have vital tissue, no objective test can predict the level of pulpal anesthesia.

Why patients in pain have difficulty with anesthesia is a complicated

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question. If there was an easy answer, we would have an easy way to provide predictable profound anesthesia for every patient every time. Theories related to pH, hyperalgesia, altered resting potentials and lowered excitability thresholds, increased sodium channel expression, TTX-R (resistant) sodium channels, and lowered pain thresholds have been suggested as potentially compromising the efficacy of local anesthetics.[146,44,343,230,293,320,347,349] This is different from the central core theory, which has been offered as an explanation for failure with the IAN block in asymptomatic vital teeth.[77,325] This theory relates to the inner portion of the nerve bundle supplying the anterior teeth and the outer portion of the nerve bundle supplying the posterior teeth (Fig. 6.3), explaining part of the higher failure rates in anterior teeth.

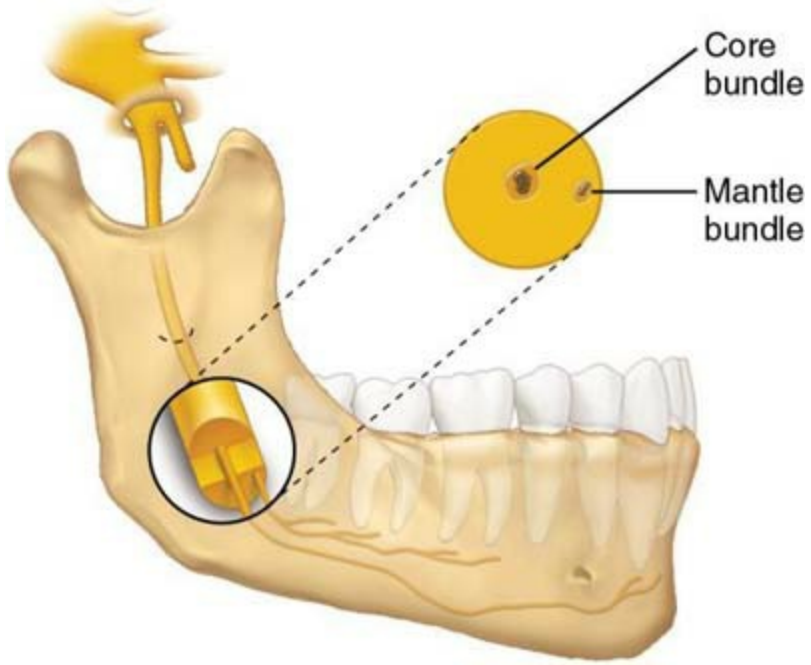

FIG. 6.3 The central core theory. The axons in the mantle bundle supply the molar teeth, and those in the core bundle supply the anterior teeth. The extraneural local anesthetic solution diffuses from the mantle to the core.

Part of mandible at the left end is enlarged that shows a core bundle inside a circle with a circular structure marked mantle bundle near the periphery.

Source: (Modified and redrawn from De Jong RH: *Local anesthetics,* St Louis, 1994, Mosby.)

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Inferior alveolar nerve block

2% lidocaine and 1:100,000 epinephrine

Because failure occurs most often with the IAN block,[180] factors that modify mandibular anesthesia must be carefully reviewed. The technique for administering an IAN block can be reviewed in available textbooks.[165,213] The following discussion reviews the expected outcomes after administration of a conventional IAN block to asymptomatic patients using 1.8 mL of 2% lidocaine with 1:100,000 epinephrine (Xylocaine, Lignospan, Octocaine). Although anesthesia requirements vary among dental procedures, the following discussion concentrates on *pulpal* anesthesia in asymptomatic patients.

Anesthetic success, duration, and difficulty

Anesthetic success for nerve blocks has been defined in the majority of studies as the percentage of subjects who achieve two consecutive nonresponsive readings on electrical pulp testing within 15 minutes and continuously sustain this lack of responsiveness for 60 minutes. The clinical condition would be pulpal anesthesia within 15 minutes that lasts for 1 hour. Using this criterion, the percentage of cases in which anesthesia was obtained after IAN block injections ranged from 10% (central incisor) to 65% (second molar).[53,140,154,225,252,277,340] In all of these studies, patients reported profound lip numbness, meaning that these are not “missed” blocks. Profound lip numbness *does not* predict pulpal anesthesia. However, lack of soft-tissue anesthesia is a useful indicator that the block injection was not administered accurately for that patient. This will occur approximately 4% to 6% of the time in asymptomatic patients.[106] The authors also found that administration of a two-cartridge volume was significantly better for avoiding a missed block than a one-cartridge volume in asymptomatic subjects. It is important to note, however, that a two-cartridge volume does not provide a higher success rate for pulpal anesthesia.[106,341,366] Once pulpal anesthesia does occur, it usually persists for approximately 2½ hours.[98] Fig. 6.4 depicts the time course for complete pulpal anesthesia for an asymptomatic first

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molar, as defined by the percentage of patients who did not respond to a stimulus (EPT) across time for 60 minutes. Most patients achieved pulpal anesthesia within 15 minutes and had a duration of anesthesia of at least 1 hour, but the success rate was not 100%.

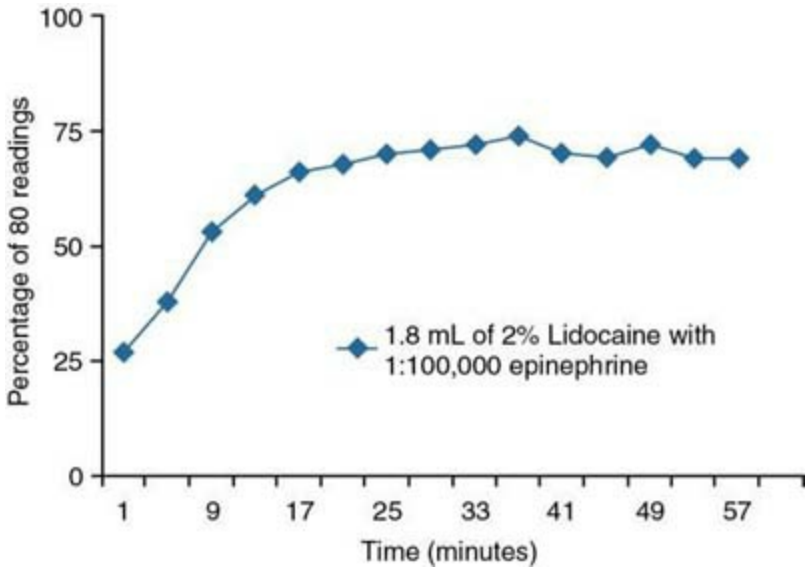

FIG. 6.4 Incidence of first mandibular molar anesthesia as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100 in increments of 25. The horizontal axis ranges from 1 to 57. The data points for 1.8 milliliters of 2 percent Lidocaine with 1 to100,000 epinephrine are represented by diamond shapes. A line connects the data points. The line originates from the point 25 on the vertical axis, rises, and reaches at (70, 17), from where the line remains constant, starts fluctuating after (33, 74), and terminates on the extreme right below the point 75. All data are approximate.

Anesthetic failure can be defined as the percentage of subjects who never achieved two consecutive nonresponsive EPT readings at any time during a 60-minute period. Using this criterion, anesthetic failure rates range from 17% (second molar) to 58% (central incisor).[53,98,140,154,225,252,277,309,340,341]

Another presentation of mandibular anesthesia is noncontinuous anesthesia, which may be related to the action of the anesthetic solution on the nerve membrane (blocking and unblocking the sodium channels). This occurs in about 12% to 20% of patients.[53,135,140,154,225,252,277,340]

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After a conventional IAN block injection, the onset of pulpal anesthesia occurs within 10 to 15 minutes in most cases (see Fig. 6.4).[53,98,140,154,225,252,277,309,340,341] Slow onset of anesthesia can occur in a percentage of subjects who are nonresponsive to the EPT after 15 minutes. In mandibular teeth, slow onset occurs in 12% to 20% of patients.

Alternative anesthetic solutions for the inferior alveolar nerve block

Solutions without epinephrine

Plain solutions (mepivacaine, prilocaine) have been found to obtain pulpal anesthesia for an IAN block at rates similar to those for lidocaine with 1:100,000 epinephrine.[62] These findings support the selection of 3% mepivacaine as a local anesthetic when medical conditions or drug therapies suggest caution in the administration of solutions containing epinephrine; however, the duration of pulpal anesthesia is reduced to approximately 50 minutes.[225]

Articaine with 1:100,000 or 1:200,000 epinephrine

Articaine is classified as an amide. It has a thiophene ring (instead of a benzene ring like the other amide local anesthetics) and an extra ester linkage, which results in hydrolysis of articaine by plasma esterases.[219] Lidocaine and articaine have the same maximal dose of 500 mg for adult patients (recommended dose, 6.6 to 7 mg/kg), but the maximum number of cartridges is different because of the differences in drug concentration (see Table 6.2).[213]

Many clinical trials and some meta-analyses have failed to demonstrate any statistical superiority of articaine over lidocaine for IAN blocks in both symptomatic and asymptomatic patients.[17,35,57,197,273,311,319,332]

Long-acting anesthetics

Clinical trials with bupivacaine (Marcaine) have been conducted in patients undergoing oral surgery,[76,292] endodontic treatment,[91,98,237,262,299], and

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periodontic treatment.[71,207] Bupivacaine was found to have a slower onset of pulpal anesthesia than lidocaine for IAN blocks.[98] Generally, bupivacaine provides prolonged analgesia and is indicated when postoperative pain is anticipated, but not all patients want lip numbness for an extended period.[292] Patients should be questioned about their preference. Although bupivacaine has a somewhat slower onset than lidocaine, its duration of pulpal anesthesia in the mandible is almost twice as long (4 hours) (Fig. 6.5).[98]

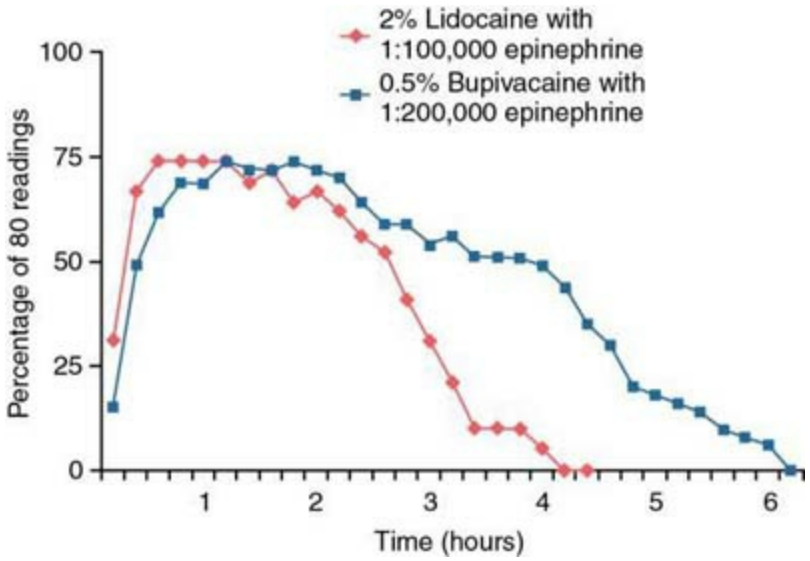

FIG. 6.5 Incidence of first mandibular molar anesthesia: comparison of 0.5% bupivacaine with 1:200,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 6 hours. The bupivacaine solution showed a longer duration of anesthesia than the lidocaine solution.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 0 to 6. The data points for 2 percent Lidocaine with 1 to 100,000 epinephrine and 0.5 percent Bupivacaine with 1:200,000 epinephrine are represented by diamond and square shapes, respectively. Two lines are fitted to both diamond and square data points. The line for 0.5 percent Bupivacaine originates from (0, 18), reaches at a height (1.2, 75), fluctuates while slopes downward, and terminates at (6, 0). The line for 2 percent Lidocaine originates from (0, 28), reaches at a height (0.5, 75), intersects the line of 0.5 percent Bupivacaine, fluctuates while slopes down, and terminates at (4.3, 0). All data are approximate.

Ropivacaine (Naropin), a relatively new long-acting local anesthetic, is a

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structural homolog of bupivacaine.[187] It is currently unavailable in dental cartridge form. A number of studies have shown that ropivacaine has a lower potential for toxic CNS and cardiovascular effects than bupivacaine but produces equivalent pharmacologic effects.[187] Ropivacaine and levobupivacaine are being developed as potentially new local anesthetics based on their stereochemistry. Both are S-isomers and are thought to cause less toxicity than the racemic mixture of bupivacaine currently marketed.[312] A medical clinical trial has indicated that levobupivacaine showed significantly better postoperative pain control at 4 and 24 hours after infiltration injection than ropivacaine.[259,215]

Liposomal bupivacaine (Exparel, Pacira Pharmaceuticals, San Diego, CA) has been approved for infiltration use by the FDA. The formulation is a preservative-free aqueous suspension of multivesicular liposomes (DepoFoam) containing bupivacaine at a concentration of 13.3 mg/mL (expressed as anhydrous bupivacaine hydrochloride [HCl] equivalent).[258,275] A small amount (3%) is free bupivacaine, which provides immediate numbness, whereas the remaining drug is released from the liposomes over time.[275] The FDA has *not* approved Exparel for nerve blocks. In clinical endodontic studies, Exparel did not significantly reduce symptomatic irreversible pulpitis pain or pain medication usage as compared with bupivacaine in untreated cases.[41] In another endodontic clinical trial using symptomatic patients diagnosed with pulpal necrosis who were experiencing moderate to severe preoperative pain and receiving endodontic treatment, a 4.0-mL infiltration of liposomal bupivacaine did not result in a statistically significant decrease in posttreatment pain compared with an infiltration of 4.0 mL of 0.5% bupivacaine with 1:200,000 epinephrine.[126]

Buffered lidocaine

Buffering lidocaine using sodium bicarbonate raises the pH of the anesthetic solution. In the medical literature, there is evidence that the use of buffered lidocaine results in less pain during the injection.[48,139] In the dental literature, some studies[11,12,34,177,296] found that buffered lidocaine produced less pain on injection and a faster onset of anesthesia. However, other dental studies[274,351,296,155] did not find less pain on injection or a faster onset with

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buffered lidocaine for IAN block. In symptomatic patients with a diagnosis of pulpal necrosis and associated acute swelling, no significant decrease in pain of infiltrations or significant decrease in the pain of an incision-and-drainage procedure was found when the buffered anesthetic formulation was used.[30,148]

Diphenhydramine as a local anesthetic agent

Diphenhydramine (Benadryl) has been advocated for patients who are allergic to local anesthetics. Multiple studies have found lower effectiveness as well as more pain and adverse effects with injection of this drug.[355,59] Diphenhydramine should not be used as an alternative to local anesthetics.

Alternative injection sites

Gow-gates and vazirani-akinosi techniques

Some clinicians have reported that the Gow-Gates technique[132] has a higher success rate than the conventional IAN block injection,[211,213] but controlled experimental studies have failed to show superiority of the Gow-Gates technique.[8,128,233,329,297] Studies have found no superiority of Gow-Gates for pulpal anesthesia in cases of symptomatic irreversible pulpitis.[4,306,60] A recent study indicated that a combination of Gow-Gates and IAN block injection was significantly superior to the Gow-Gates or IAN blocks alone.[297] The Vazirani-Akinosi technique[9,146,213] also has not been found to be superior to the standard inferior alveolar injection.[146,219,314,329,369] The Vazirani-Akinosi technique is indicated for cases involving a limited mandibular opening to assist with further opening. After using the Vazirani-Akinosi technique, conventional IAN blocks should be given to ensure pulpal anesthesia.

Articaine infiltrations

Articaine is significantly better than lidocaine for buccal infiltration of the mandibular first molar.[69,171,173,285] However, articaine alone does not predictably provide pulpal anesthesia of the mandibular first molar. There is

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no difference between 4% articaine with 1:100,000 and 1:200,000 epinephrine for buccal infiltration for pulpal anesthesia.[223]

In anterior teeth, buccal and lingual infiltrations of articaine provide initial pulpal anesthesia, but the anesthesia declines over 60 minutes.[161,256]

Volume and concentration

Increasing the volume of 2% lidocaine with epinephrine to 3.6 mL (two cartridges) does not increase the incidence of pulpal anesthesia with the IAN block (Fig. 6.6).[105,252,366] Also, no advantage has been seen in using a higher concentration (1:50,000 versus 1:100,000) of epinephrine.[73,341]

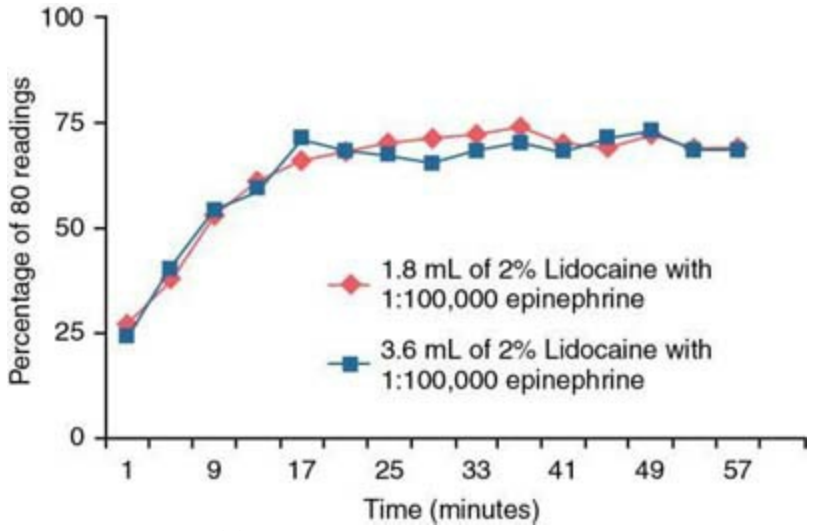

FIG. 6.6 Incidence of first mandibular molar anesthesia: comparison of 1.8 and 3.6 mL of 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two volumes was noted.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 1 (near the origin) to 57. The data points for 1.8 milliliters of 2 percent Lidocaine with 1 to100,000 epinephrine and 3.6 milliliters of 2 percent Lidocaine with 1 to100,000 epinephrine are represented by diamond and square shapes, respectively. Two lines are fitted to both diamond and square data points. The line for 1.8 milliliters of 2 percent Lidocaine originates from (1, 25), increases in a concave down manner, fluctuates, and terminates at (57, 65). The line for 3.6 milliliters of 2 percent Lidocaine originates from (0, 26), merges with the

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first line, reaches at a height (17, 73), fluctuates while crossing the first line, and terminates at (57, 65). All data are approximate.

Factors in failure of the inferior alveolar nerve block

Many theories abound to explain the failure to achieve pulpal anesthesia with an IAN block; these include accessory and cross innervation, injection accuracy, needle beveling, and needle deflection. The mylohyoid nerve is the accessory nerve most often cited as a cause of failure of mandibular anesthesia.[111,366] A controlled clinical trial compared the IAN block alone with a combination of the IAN block and a mylohyoid nerve block using a peripheral nerve stimulator (Fig. 6.7); it was found that the mylohyoid injection did not significantly enhance pulpal anesthesia of the IAN block (Fig. 6.8).[58] Experimentally, cross innervation has been implicated in the failure of anterior pulpal anesthesia. Although this does occur in incisors,[289,368] it plays a very small role in the failure of an IAN block. It has also been theorized that an inaccurate injection contributes to inadequate mandibular anesthesia, but a number of studies have determined that the use of ultrasound, a peripheral nerve stimulator, or radiographs to guide needle placement for IAN blocks did not result in more successful pulpal anesthesia.[26,116,140,309] Needle deflection has also been proposed as a cause of failure with the IAN block.[64,75,156] Several in vitro studies have shown that beveled needles tend to deflect toward the nonbeveled side (i.e., away from the bevel).[10,64,75,156,168,286] In one clinical investigation, however, no significant difference in anesthetic success was seen when the needle bevel was oriented away from the mandibular ramus (so that the needle would deflect toward the mandibular foramen [90% success]) compared with the needle bevel oriented toward the mandibular ramus (92% success).[324]

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FIG. 6.7 Injection site for the mylohyoid nerve block.

Mandible shows an injection of 2 percent xylo is being administrated to the nerve block above the last molar.

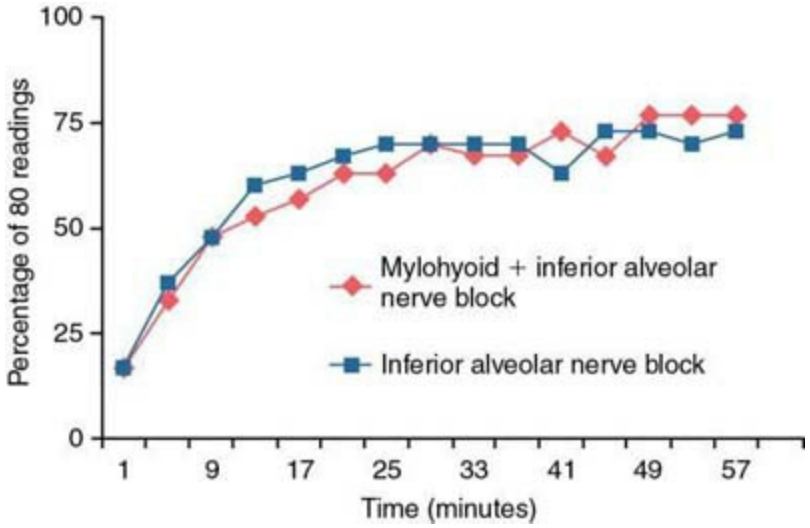

FIG. 6.8 Incidence of first mandibular molar anesthesia: comparison of the combination mylohyoid infiltration plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two techniques was noted.

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Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 1 (near the origin) to 57. The data points for mylohyoid plus inferior alveolar nerve block and inferior alveolar nerve block are represented by diamond and square shapes, respectively. Two lines connect the data points. The first line of mylohyoid plus inferior alveolar nerve block originates from (1, 18), rises up, fluctuates, and terminates at (57, 75). The second line of inferior alveolar nerve block originates from the same point as that of the first line, crosses the first line, increases in a concave down manner, remains constant, again crosses the first line, and terminates just below the first line. All data are approximate.

Speed of injection and success

A slow IAN block is more successful than a fast injection,[172] but not for patients diagnosed with irreversible pulpitis.[6]

Supplemental anesthesia for vital pulps in the mandible

Indications

A supplemental injection is used if the standard injection is not effective. It is useful to repeat an initial injection only if the patient is not exhibiting the classic signs of soft-tissue anesthesia, such as profound lip numbness. Generally, if the classic signs are present, reinjection is not very effective.[175] For example, after the IAN block, the patient develops lip, chin, and tongue numbness and quadrant “deadness” of the teeth. A useful procedure, as previously described, is to pulp test the tooth with a cold refrigerant or an EPT before the cavity preparation is begun.[49,88] If the patient feels pain to cold, a supplemental injection is indicated.

Supplemental injections include the infiltration injection, the IO injection, the intraligament (IL) injection (formerly called the periodontal ligament [PDL] injection), the intraseptal (IS) injection, and the intrapulpal (IP) injection.

Infiltrations

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Supplemental mandibular buccal, lingual, or buccal plus lingual infiltrations of articaine

Although the infiltration of articaine is effective in restorative dentistry as a supplemental technique (after the IAN block),[138] its use in endodontically involved teeth does not result in predictable pulpal anesthesia.[104,221,257,304,310] A buccal infiltration of lidocaine is also not effective for profound, predictable pulpal anesthesia. An important clinical finding is that an articaine infiltration of the first molar, premolars, and anterior teeth after an IAN block should provide pulpal anesthesia for approximately 1 hour.[138,174,256]

Buccal infiltration alone and buccal plus lingual infiltrations alone (or after an IAN block) do not result in complete pulpal anesthesia.[3,4,7,87,95,273,305] Therefore IO and IL injections are the preferred techniques for supplemental anesthesia, and the IP injection is indicated when the other injections are not successful.

Intraosseous anesthesia

The IO injection is a supplemental technique that has been shown to be effective by substantial research and clinical use. It is particularly useful in conjunction with an IAN block when it is likely that supplemental anesthesia will be necessary (e.g., in mandibular second molar teeth).[90,118,135] The IO injection allows placement of a local anesthetic directly into the cancellous bone adjacent to the tooth. When a primary IO injection was compared with an infiltration injection, the IO technique showed a quicker onset and a shorter duration of anesthesia (Fig. 6.9).[254]

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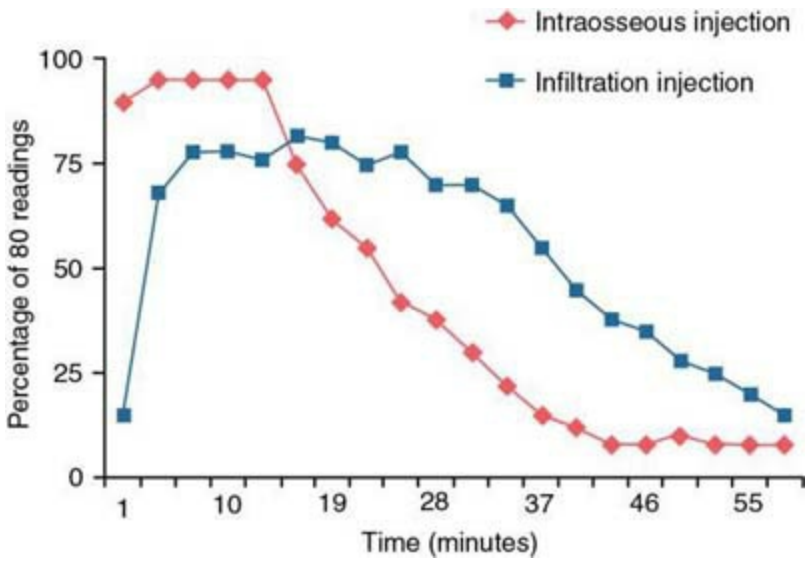

FIG. 6.9 Incidence of anesthesia for intraosseous and infiltration injections. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The intraosseous injection showed a quicker onset and a shorter duration of anesthesia.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 1 (near the origin) to 55. The data points for intraosseous injection and infiltration injection are represented by diamond and square blocks, respectively. Two lines connect the data points. The first line for intraosseous injection originates from (1, 93), stays constant until (13, 94), slops down in a concave up decreasing manner, and terminates at (57, 10). The second line for infiltration injection originates from (1, 18), reaches at a height (9, 76), intersects the first line (17, 76), fluctuates while slopes down, and terminates at (57, 17). All data are approximate.

The Stabident IO system (Fairfax Dental Inc., Miami, FL) utilizes a slow-speed, handpiece-driven perforator and a solid 27-gauge beveled wire that drills a small hole through the cortical plate (Fig. 6.10). The anesthetic solution is delivered into cancellous bone through a 27-gauge ultrashort needle placed into the perforation made by the perforator (Fig. 6.11).

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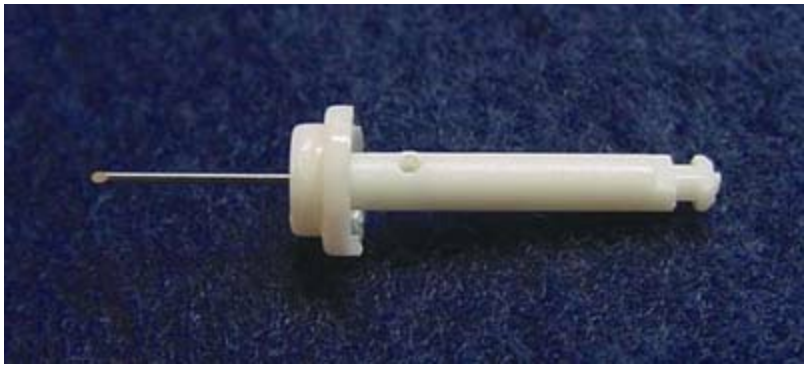

FIG. 6.10 The Stabident perforator, a solid 27-gauge wire with a beveled end that is placed in a slow-speed handpiece.

Stabident shows a plastic handle with a large needle at the center.

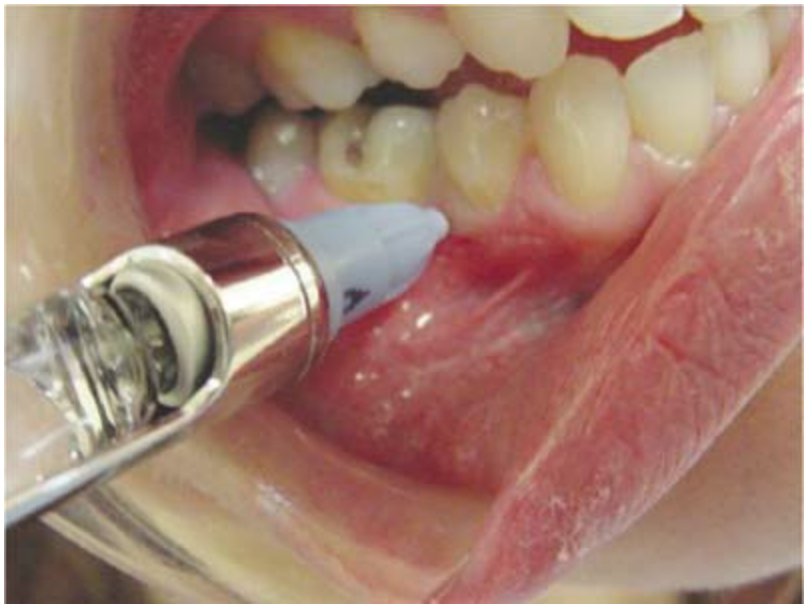

FIG. 6.11 The anesthetic solution is delivered to the cancellous bone through a needle placed into the hole made by the perforator.

Close-up view of open mouth shows an injection being inserted near the gum line in-between teeth in the lower right quadrant. The tooth adjacent to injection site has a cavity.

The X-Tip IO anesthetic delivery system (Dentsply International, York, PA) consists of an X-tip that separates into two parts, the drill and the guide sleeve (Fig. 6.12). The drill, a special hollow needle, leads the guide sleeve through the cortical plate; it is then separated from the guide sleeve and

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withdrawn. The remaining guide sleeve is sized to receive a 27-gauge needle for deposition of the anesthetic solution (Fig. 6.13). The guide sleeve is removed after the IO injection is complete.

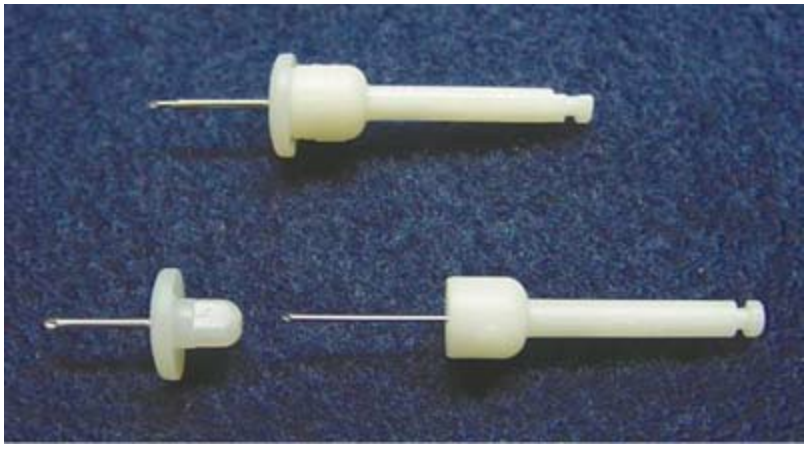

FIG. 6.12 The X-Tip anesthetic delivery system consists of an X-Tip *(top)* that separates into two parts: the drill (a special hollow needle) and the guide sleeve component *(bottom).*

Three X-tips are shown. Two X-tips have Y-shaped handle and a needle. One X-tip shows U-shaped handle with a flat bottom at the base and a needle at the center.

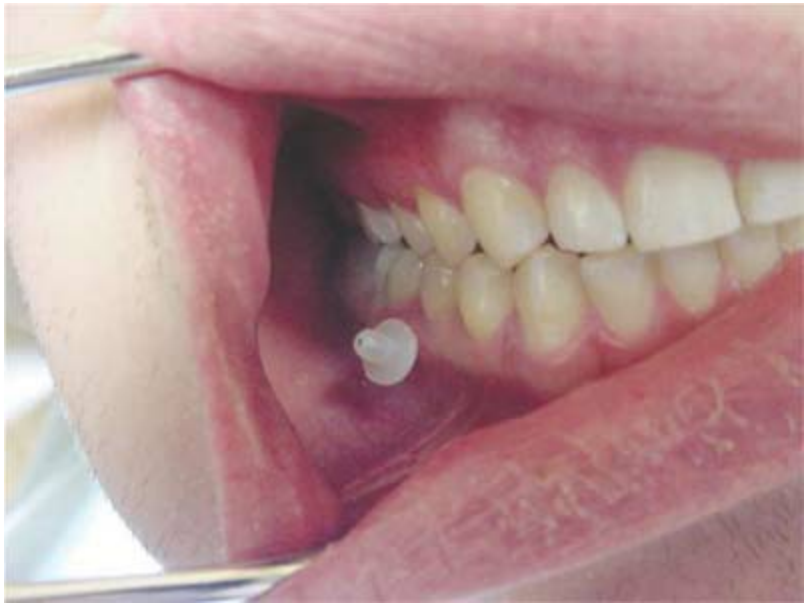

FIG. 6.13 The anesthetic solution is injected through the X-Tip guide

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sleeve.

Close-up view shows an X-tip being inserted below the gum line in the lower right quadrant.

Another technique available is the TuttleNumbNow (TNN) system. It consists of a Septodont Evolution needle (needle with a secondary scalpel bevel) (Fig. 6.14) and a TNN needle guide that allows the needle to be safely bent by 90 degrees (Fig. 6.15). Instead of a motor-driven perforator, the needle is rotated back and forth 180 degrees as pressure is applied by hand via the anesthetic syringe. The beveled needle is claimed to perforate the cortical bone and allows for delivery of anesthetic into the cancellous bone. It is important to note that this is a system that is intended to deliver anesthetic intraosseously without a separate perforator.

FIG. 6.14 Septodont Evolution needle used for the Tuttle Numb Now intraosseous injection technique.

Box of septodont anesthesia needles is shown. Needles with a scalpel bevel are placed near by.

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FIG. 6.15 Septodont needle sleeve, which allows safe bending of the needle for the Tuttle Numb Now intraosseous injection technique.

Septodont needle sleeve shows a hollow structure with a rim at the base. The needle is bent at the top.

The techniques for IO injection of anesthetic using the Stabident, X-Tip, or TNN system can be reviewed in the systems' instruction manuals or in published papers or books.[61,119,280–282]

Success and duration

Supplemental IO injections of lidocaine and mepivacaine with vasoconstrictors allow quick onset and increase the success of the IAN block for approximately 60 minutes (Fig. 6.16).[90,135] The addition of a supplemental IO injection reduced the incidence of slow onset of pulpal anesthesia to zero compared with the IAN block alone (18% incidence).[90] Using 3% mepivacaine plain results in pulpal anesthesia for approximately 30 minutes (Fig. 6.17).[118] Repeating the IO injection—using 1.4 mL of 2% lidocaine with 1:100,000 epinephrine 30 minutes after the initial IO injection—provides an additional 15 to 20 minutes of pulpal anesthesia, similar to the

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duration of the initial IO injection.[167]

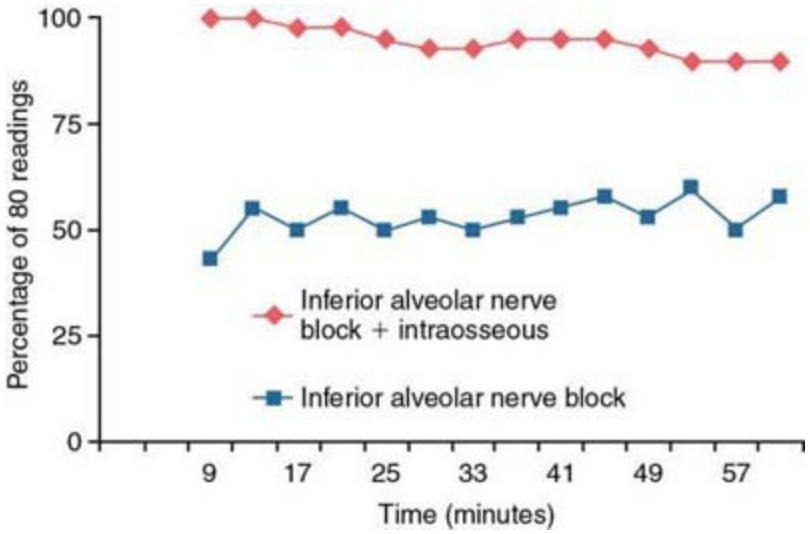

FIG. 6.16 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection of 2% lidocaine with 1:100,000 epinephrine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique was significantly better at all postinjection times.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 9 (slightly away from the origin) to 57. The data points for inferior alveolar nerve block plus intraosseous and inferior alveolar nerve block are represented by diamond and square blocks, respectively. Two lines connect the data points. The first line of inferior alveolar nerve block plus intraosseous originates from (9, 100), fluctuates slightly, and terminates at (59, 98). The second line of inferior alveolar nerve block originates from (9, 43), rises up to (13, 52), fluctuates, and terminates at (59, 52). All data are approximate.

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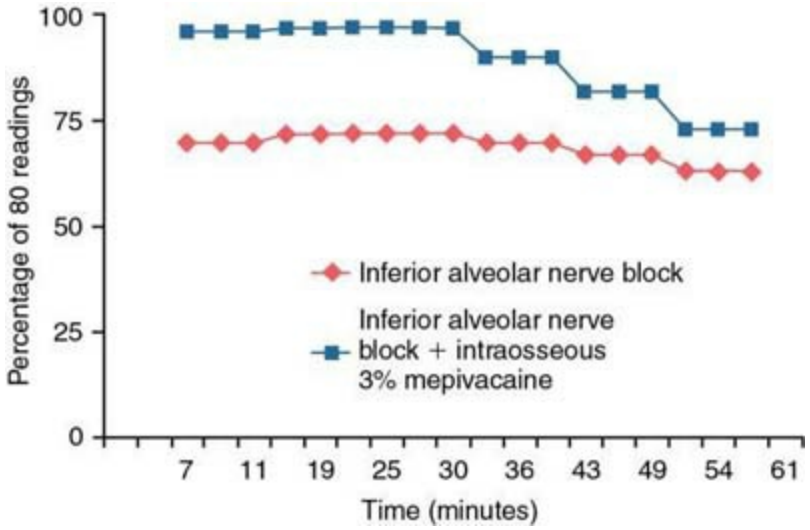

FIG. 6.17 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection with 3% mepivacaine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique proved to be significantly better for approximately 30 minutes.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 7 (slightly away from the origin) to 61. The data points for inferior alveolar nerve block and inferior alveolar nerve block plus intraosseous 3 percent mepivacaineare represented by diamond and square blocks, respectively. Two lines connect the data points. The first line of inferior alveolar nerve block originates from (7, 98), moves constantly upto (32, 98), slopes down, forms ladder-like steps, and finally terminates at (57, 75). The second line of inferior alveolar nerve block plus intraosseous originates from (7, 72), fluctuates a little bit, and finally terminates at (57, 70). All data are approximate.

Long-acting anesthetics do not show an extended duration of anesthesia when injected by the IO route.[159,322] Bupivacaine has cardiotoxic effects[18] and for IO anesthesia offers no advantage, clinically, to 2% lidocaine with epinephrine in terms of efficacy, duration, and heart rate effects. Therefore bupivacaine should not be used for IO anesthesia.

Articaine use in the IO injection has also been found not to provide superior results compared with lidocaine.[27]

Success in symptomatic irreversible pulpitis

High success rates (around 90%) have been reported when the IO injection

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was used as a supplemental injection with irreversible pulpitis.[27,221,250,253,257] Three percent mepivacaine has an 80% success rate, which increases to 98% with a second IO injection of 3% mepivacaine.[349]

An initial supplemental IO injection of 0.45 to 0.9 mL of 2% lidocaine with 1:100,000 epinephrine was successful in 79% of posterior mandibular teeth.[260] A second IO injection of the remaining cartridge increased the success rate to 91%. Therefore, initially giving a quarter to a half cartridge of 2% lidocaine with 1:100,000 epinephrine was less successful than initially giving a full cartridge.

Although some authors[264,278] have suggested an IO injection alone successfully anesthetizes patients presenting with irreversible pulpitis, it is very doubtful that this would be successful.[27,96,221,250,253,257]

Complications/contraindications.

During an IO injection, if the anesthetic solution flows out of the perforation site (backflow), anesthesia will not occur.[120] Reperforation or use of another perforation site is a practical way to gain access to the cancellous bone in such cases. In fewer than 10% of cases, constricted cancellous spaces may limit the distribution of the anesthetic solution around the apices of the teeth.[61,90,120,135,250,253,279–282,322] In such cases, failure may result even if the anesthetic solution is delivered intraosseously. Reports have shown that approximately 1% of Stabident cases had the metal perforator separate from the plastic shank of the Stabident system during use.[61,90,120,250,280–282] The metal perforator can be easily removed with a hemostat but may require a small gingival flap to access the separated component. Separation usually occurs during a difficult perforation (e.g., dense cortical bone) and may be caused by the wire being heated excessively due to friction, thus causing the plastic hub to melt. No perforator breakage (metal perforator breaking into parts) has been reported in numerous published clinical studies.[61,90,120,250,280–282] However, excessive torqueing of the perforator laterally by an inexperienced operator may result in breakage. When the IO injection is used as a primary injection, pain is experienced about 25% of the time.[61,119,280–282] When the IO injection is used as a supplemental injection, fewer patients experience pain.[90,119,135,280,322] With the Stabident system, fewer than 5% of patients develop swelling or exudate at the site of bone

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perforation.[61,135,280–282] The X-Tip system may show a higher incidence of postoperative swelling.[119] With both systems, the swelling and/or exudate may persist for several weeks after the injection, but should resolve with time.[63,119,135,280–282] Slow healing of perforation sites may be the result of overheating of the bone caused by pressure during perforation.

With both the Stabident and X-Tip systems, approximately 4% to 15% of patients may report that the tooth "feels high" during chewing.[63,119,135,280–282] This sensation is probably due to an increased awareness to pressure due to soreness in the treated area caused by damage from perforation or inflammation of the bone. The incidence of this effect is lower for the IO injection than that reported for the IL injection (36% to 49%).[72,300] When IO injections are given with the Stabident system, either as a primary or a supplemental technique, most patients report no or only mild pain; approximately 2% to 15% report moderate pain.[63,135,280–282] Less postoperative discomfort is reported for the Stabident IO injection than for IL injection.[300]

One study found that significantly more men experienced postoperative pain with the X-Tip system than with the Stabident system.[119] The authors interpreted these results as being due to the denser, more mineralized bone in the posterior mandible in men and to the fact that the diameter of the X-Tip perforating system is larger than that of the Stabident perforator, meaning the X-Tip system generates more frictional heat during perforation. No clinical research has been published about the TNN system.

Systemic effects

A transient increase in heart rate has been reported in 46% to 93% of cases involving Stabident and X-Tip IO injection of solutions containing epinephrine or levonordefrin.[61,90,120,135,250,253,280–282,322] Four clinical trials using techniques such as objective electrocardiographic recordings and pulse oximetry have shown that subjects experienced a transient tachycardia (mean increase of 12 to 32 beats/min) after Stabident IO injection of 1.8 mL of 2% lidocaine with 1:100,000 epinephrine, 1.8 mL of 2% mepivacaine with 1:20,000 levonordefrin, or 1.8 mL of 1.5% etidocaine with 1:200,000 epinephrine.[50,135,283,322] Another clinical trial reported transient tachycardia after IO injection, but not with infiltration injection, of 1.8 mL of 2%

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lidocaine with 1:100,000 epinephrine in the maxillary anterior region.[358] All of these studies showed that the heart rate returned to original baseline readings within 4 minutes in most patients. Therefore IO injection of anesthetic solutions containing vasoconstrictors with either the Stabident or X-Tip system results in a transient tachycardia. No research has been conducted with the TNN system, although similar results would be expected. However, no significant changes in diastolic, systolic, or mean arterial blood pressure has been reported with the IO injection of 2% lidocaine with 1:100,000 epinephrine.[50,283]

Although the patient is likely to notice the transient tachycardia that occurs after Stabident or X-Tip IO injection of 2% lidocaine with 1:100,000 epinephrine, it is generally not clinically significant in healthy patients.[283] The clinical significance, cardiovascular effects, and contraindications to the use of vasoconstrictors in IO injections have been reviewed.[283]

No significant tachycardia occurs when 3% mepivacaine is used for IO anesthesia.[118,283] Clinicians should keep in mind that this anesthetic is an alternative for IO injection in patients whose medical condition or drug therapy suggests caution in the use of solutions containing epinephrine or levonordefrin.[118,283]

Plasma levels of lidocaine after intraosseous injection.

Some authors have warned that administration of a large volume of local anesthetic via an IO injection could lead to overdose reactions.[160] One experimental study using volunteers found that injection of 1.8 mL of 2% lidocaine with 1:100,000 epinephrine produced venous plasma levels of lidocaine that were the same for maxillary anterior IO and infiltration injections (Fig. 6.18).[358] Although systemic concentrations of vasoconstrictors have a short-lived effect on the heart rate, the plasma concentration of lidocaine delivered with IO injection is similar to that delivered via infiltration, so the IO technique should not be considered an intravascular injection with regard to lidocaine. Therefore the precautions for the maximum amount of lidocaine for an infiltration injection would seem to apply to an IO injection.

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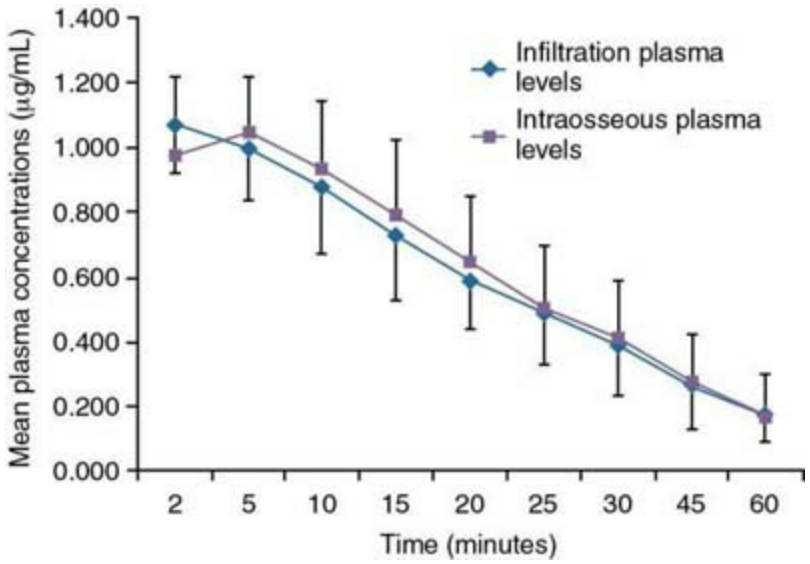

FIG. 6.18 Mean plasma concentrations of lidocaine for the intraosseous and infiltration injection techniques. No statistical differences were seen between the two techniques at any time period.

Graph shows mean plasma concentrations in micro grams per milliliters against time in minutes. The vertical axis ranges from 0.0 0 0 to 1.4 0 0. The horizontal axis ranges from 2 (slightly away from the origin) to 60. The data points for infiltration plasma levels and intraosseous plasma levels are represented by diamond and square blocks, respectively. Two lines connect the data points. The first line of infiltration plasma levels originates from (2, 1.0 4 0), slopes down, and terminates at (60, 0.1 6 0). The second line of intraosseous plasma levels originates from (2, 0.9 8 0), intersects the first line at (4, 1.0 0 0), moves further, slopes down, and again merged with the first line. The second line ends with the first line at the same point. All data are approximate.

Intraligament anesthesia

The IL injection is another technique that is used if a conventional injection is unsuccessful.[317,344] The technique for the IL injection of anesthesia is reviewed in a number of published papers and textbooks.

Success, onset, and duration

For use as a primary injection, IL injections have a reported success rate of about 75% in mandibular posterior teeth, with a duration of pulpal anesthesia of 10 to 15 minutes.[300,352] Success rates are low in anterior teeth.[224,300,352]

For use as a supplemental injection (e.g., when standard techniques have

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failed to provide adequate anesthesia), good success rates are achieved, but the duration of pulpal anesthesia is approximately only 23 minutes.[54]

The onset of anesthesia is immediate with an IL injection,[72,324,300,317,344,352] which means that no waiting period is required for the anesthesia to take effect. If anesthesia is still not adequate, reinjection may be necessary.

Success in symptomatic irreversible pulpitis

The overall success of supplemental IL injections in achieving pulpal anesthesia for endodontic procedures has been reported to be 50% to 96%.[63,212,317,344] If a first IL injection fails, reinjection results in a success rate of 92%.[344] Similar results have been reported by other investigators.[317] IL injection is not successful in mandibular anterior teeth.[226,352] In addition, supplemental IL injections are not as successful as supplemental IO injections.[175,370]

Mechanism of action

An IL injection forces anesthetic solutions through the cribriform plate into the cancellous bone around the tooth.[88,113,276,316,346] The primary route of anesthetic is *not* via the PDL, and unlike the IP injection,[329,339] the mechanism of action is not a pressure anesthesia.[92,234] The IL injection should be considered an IO injection.

Back pressure and amount of solution delivered

Studies have shown that the most important factor for anesthetic success with an IL injection is delivery of anesthetic under *strong back pressure*.[316,344] Pressure is necessary to force the solution into the marrow spaces. Usually about 0.2 mL of solution is deposited with each injection and the use of traditional or pressure syringes. However, the exact amount of anesthetic delivered is not always known because some of the anesthetic solution may escape from the injection site.

Anesthetic solutions

A vasoconstrictor significantly increases the efficacy of an IL

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injection.[133,179,190,226,300] Anesthetic solutions with reduced vasoconstrictor concentrations are not very effective with the IL technique.[133,169,179] Articaine is equivalent to lidocaine.[5,25]

Injection discomfort.

When the IL injection is used as a primary injection, needle insertion and injection have the potential to be painful about one third of the time.[234,300,352] The IL injection may be quite painful in maxillary anterior teeth[352] and should not be used in these teeth; infiltration is preferred. The potential for pain with the IL technique is greatly reduced when the injection is used as a supplemental injection after an IAN block.[54]

Selective anesthesia.

Although some authors have reported that the IL injection can be used to differentially diagnose pulpally involved teeth,[209] clinical studies have shown that adjacent teeth can also become anesthetized with the IL injection.[234,300,352] Therefore the IL injection should *not* be used for differential diagnosis.

Postoperative discomfort

When the IL injection is used as a primary technique, postoperative pain occurs in one third to three quarters of patients, with a duration of 14 hours to 3 days.[72,234,300,317,344,352] The discomfort is related to damage from needle insertion rather than to the pressure of depositing the solution.[72] About one fourth of patients report that their tooth feels “high” in occlusion.[300,352]

Systemic effects

When a high-pressure syringe was used in dogs, IL injection of solutions containing epinephrine caused cardiovascular responses similar to those seen with intravenous injections.[315] Clinical studies using a high-pressure syringe in human beings found that IL injections of such solutions did not significantly change the heart rate, rhythm, or amplitude or the blood pressure.[46,247] These studies support the conclusion that IL injections do not cause significant changes in heart rate in human beings.

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Other factors

Different needle gauges (25, 27, or 30) are equally effective.[344] Special pressure syringes have been marketed but have not been proven more effective than a standard syringe.[300,344,352]

Safety of the periodontium and pulp

Clinical and animal studies have demonstrated the safety of the IL injection technique.[36,112,113,117,208,234,266,300,344,352] Minor transient damage occurs only at the site of needle penetration, but the tissue subsequently undergoes repair. In rare cases, periodontal abscesses and deep pocket formation have occurred after IL injections.[50,352] A small clinical risk of periodontal abscess formation and bone loss exists with this technique, and although these effects are rare, the clinician should be aware of them. Localized areas of root resorption after IL injections have also been reported.[265,284]

IL injections have been shown to be safe in cases of mild to moderate gingival inflammation or incipient periodontitis.[70]

Studies have shown that IL injections have no permanent impact on the pulp.[205,234,267,284,300,352] However, IL injection of a solution containing epinephrine does produce a rapid and prolonged decrease in blood flow to the pulp tissue.[190] Some have suggested that using the IL injection technique during restorative procedures could result in an accumulation of inflammatory mediators in the pulp that could not be effectively removed because of the decreased blood flow.[190] This hypothesis was directly tested, and the IL injection of an anesthetic solution containing a vasoconstrictor in conjunction with a deep cavity preparation did *not* produce a more severe pulpal reaction than in controls (cavity preparation only).[270] It was reported that the depth of the cavity preparation was the biggest factor dictating pulpal responses. IL injections are therefore unlikely to cause pulpal necrosis.

Intraseptal injection

IS injections are somewhat similar to IL injections in that anesthetic is deposited directly into the interdental septum and the anesthetic gains access to the cancellous bone surrounding the tooth. The injection technique involves placing the needle in the center of the papillary triangle at a 45-

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degree angle and advancing the needle until it contacts the bony crest.[295,359] The needle may then be pressed into the IS bone and anesthetic delivered with the same back pressure as achieved during an IL injection.

Clinical research has shown the anesthetic success to range from 35% to 90% as a primary injection when used for extractions, restorations, or anesthetic evaluations.[28,31,38] For patients diagnosed with symptomatic irreversible pulpitis, Webster and coworkers[348] reported that the success of the IS injection as a supplement to the IAN block was only 29%.

Intrapulpal injection

In about 5% to 10% of mandibular posterior teeth with irreversible pulpitis, supplemental IO or IL injections, even when repeated, do not produce profound pulpal anesthesia and pain is experienced by the patient when the pulp is entered. This is an indication for the IP injection.

The major drawback of the IP injection technique is that the needle and injection are made directly into vital and very responsive pulp tissue; this injection is quite often moderately to severely painful.[253] The endodontic literature offers more efficacious methods of supplemental pulpal anesthesia; therefore the IP injection should be given only after all other supplemental anesthetic techniques have failed. Another disadvantage of the technique is the duration of pulpal anesthesia (15 to 20 minutes). The pulpal tissue must be removed quickly and completely to prevent a recurrence of pain during instrumentation. Also, in order to give the IP injection, the pulp must be exposed to allow direct injection. Anesthetic problems frequently occur before exposure while the clinician is still working in dentin; thus patients may experience pain immediately prior to exposure of the pulp.[221,250,251,253,263,279,310]

The advantage of the IP injection is that it produces profound anesthesia if it is given under back pressure.[29,339] The onset of anesthesia is immediate, and no special syringes or needles are required. The methods for this technique can be found in many endodontic textbooks. As mentioned previously, strong back pressure is the major factor in achieving successful IP anesthesia.[29,339] Simply depositing the anesthetic solution into the chamber will be insufficient because the anesthetic solution will not diffuse throughout the pulp tissue.

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Maxillary anesthesia

Descriptions of conventional techniques for maxillary anesthesia are available for review in numerous articles and textbooks.[165,213] The most commonly used injection for anesthetizing maxillary teeth is infiltration with a cartridge of 2% lidocaine with 1:100,000 epinephrine. Infiltration results in a fairly high incidence of successful pulpal anesthesia (around 87% to 92%).[40,94,134,178,202,220,231,254,261,277,302] However, some patients may not be anesthetized because of individual variations in response to the drug administered, operator differences, and variations of anatomy and tooth position. Pulpal anesthesia usually occurs within 3 to 5 minutes.[40,94,134,178,202,220,231,254,261,277,302] The duration of pulpal anesthesia is a problem with maxillary infiltrations.[40,94,134,178,202,220,236,254,261,277,302] Pulpal anesthesia of the anterior teeth declines after about 30 minutes, with most losing anesthesia by 60 minutes.[40,94,134,178,202,220,236,254,261,277,302] In premolars and first molars, pulpal anesthesia is good until about 40 to 45 minutes and then it starts to decline.[40,94,134,178,202,220,236,254,261,277,302] Additional local anesthetic should be administered depending on the duration of the procedure and the tooth group affected.

Alternative anesthetic solutions for maxillary infiltrations

Plain solutions: 3% mepivacaine (carbocaine, polocaine, scandonest) and 4% prilocaine (citanest plain)

Anesthesia duration is shorter with these solutions.[178,220] Therefore these are best used for procedures of short duration (10 to 15 minutes) (Fig. 6.19). They are generally not as safe as anesthetic solutions with vasoconstrictors, especially if *large* volumes are administered. They are rapidly absorbed systemically, thus resulting in excessive plasma concentrations and possible toxic reactions.[213]

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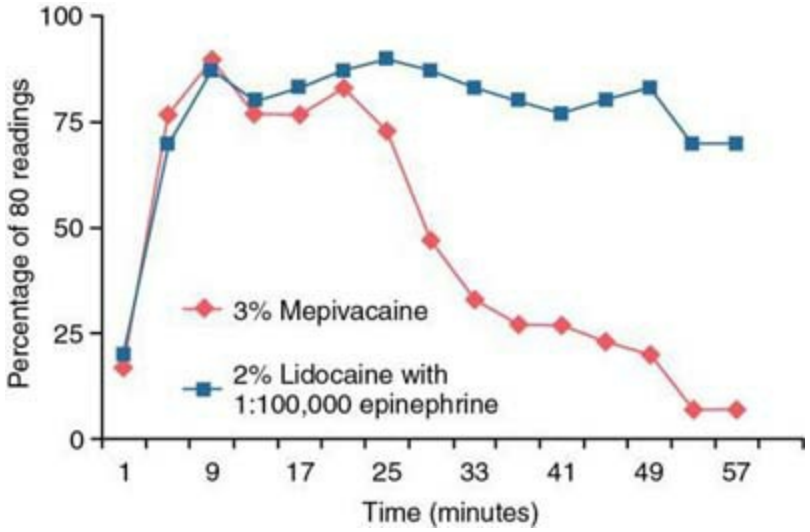

FIG. 6.19 Incidence of first maxillary molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The 3% mepivacaine showed a shorter duration of anesthesia than the lidocaine solution.

Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 1 (near the origin) to 57. The data points for 3 percent Mepivacaine and 2 percent Lidocaine with 1 to 100,000 epinephrine are represented by diamond and square shapes, respectively. Two lines connect the data points. The first line representing 3 percent Mepivacaine originates from (1, 20), moves up, and reaches at a height (9, 90), slopes down, fluctuates, and terminates at (57, 8). The second line representing 2 percent Lidocaine with epinephrine originates just above the first line, merges with the first line, reaches a height at (9, 90), fluctuates horizontally, and terminates at (57, 75). All data are approximate.

4% prilocaine with 1:200,000 epinephrine (citanest forte), 2% mepivacaine with 1:20,000 levonordefrin (carbocaine with neo-cobefrin), and 4% articaine with 1:100,000 epinephrine (septocaine, articadent, zorcaine)

These formulations are similar to 2% lidocaine with 1:100,000 epinephrine.[94,178,202]

0.5% bupivacaine with epinephrine (marcaine)

Success rates with bupivacaine range from 80% to 95% in the maxillary

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lateral incisor compared with 50% in the maxillary second premolars.[73,134,187,328] Although bupivacaine can provide long-term anesthesia with the IAN block, it *does not* provide prolonged pulpal anesthesia with maxillary infiltration injection.[74,134,187]

Extending the duration of pulpal anesthesia for maxillary teeth

A two-cartridge volume of 2% lidocaine with epinephrine extends the duration of pulpal anesthesia, but not for 60 minutes.[231] Adding another cartridge of 2% lidocaine with epinephrine at 30 minutes in anterior teeth and 45 minutes in posterior teeth significantly improves the duration of pulpal anesthesia and may be the best way to extend the duration of pulpal anesthesia (Fig. 6.20).[302]

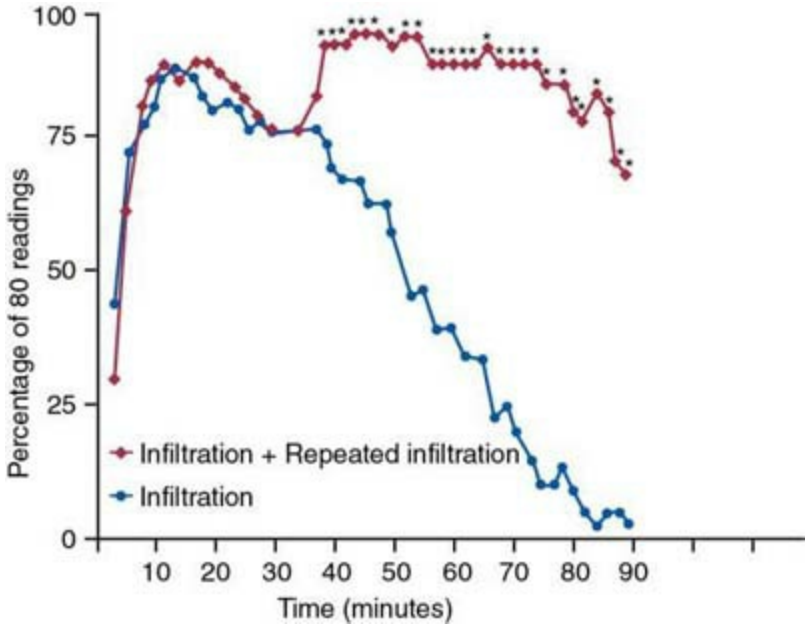

FIG. 6.20 Incidence of maxillary lateral incisor pulpal anesthesia using an initial infiltration and a repeated infiltration 30 minutes later (both infiltrations used 1.8 mL of 2% lidocaine with 1:100,000 epinephrine). Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings). The repeated infiltration injection significantly prolonged the duration of pulpal anesthesia.

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Graph shows percentage of 80 readings against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 10 (away from the origin) to 90. The data points for infiltration plus repeated infiltration and infiltration are represented by diamond and square shapes, respectively. Two lines connect the data points. The first line representing infiltration plus repeated infiltration originates from (3, 29), reaches a height at (10, 88), forms a U-shape, fluctuates, and terminates at (90, 70). The data points are marked with asterisks from (40, 92) to (90, 70). The second line representing infiltration originates from (2, 45), reaches a height at (11, 88), fluctuates while slopes down, and terminates at (90, 2). All data are approximate.

Alternative maxillary injection techniques

For routine restorative and endodontic procedures, posterior superior alveolar (PSA),[209,268] anterior middle superior alveolar (AMSA),[107,108,110,127,203,251] palatal-anterior superior alveolar (P-ASA),[44,109,110,248] infraorbital,[24,176] and second division nerve blocks[39,101] are not advocated. Infiltrations of the area are preferred for pulpal anesthesia.

Kovanaze is an intranasal 3% tetracaine plus 0.05% oxymetazoline formulation designed to provide anesthesia of the maxillary anterior and premolar teeth. Kovanaze is a needle-free anesthetic delivered as an intranasal atomized plume of solution. It was developed as an alternative to needle delivery systems because of the anxiety, fear, and needle sticks associated with traditional systems. Kovanaze has already been studied for its cardiovascular and pharmacokinetic effects[56,152,124] and in restorative procedures in maxillary anterior and posterior teeth.[56,153,152] Studies evaluating pulpal anesthesia are likely to follow to determine its future use in endodontics.

Management of anesthesia in endodontic cases

The management of anesthesia in endodontic cases generally includes use of the solutions, injection types, and supplemental anesthesia modalities mentioned throughout this chapter. Anesthesia types and indications will differ depending on pulpal diagnosis. For a thorough review of each clinical

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scenario with clinical flowcharts, the reader is directed to more in-depth texts on this topic.[277]

Summary and future directions for effective anesthesia

The emergence of evidence-based dentistry has rapidly drawn interest across all fields of dentistry. Fortunately, the large number of high-quality randomized controlled studies in anesthesia and analgesia provide a wealth of information supporting evidence-based recommendations. The practitioner must focus on high-quality research when clinical decisions are being made.

The prudent practitioner knows that there is no “magic bullet” in pain control. Rather, the clinician must be knowledgeable about the advantages and disadvantages of several anesthetic-vasoconstrictor combinations and the various routes of injection. Clearly, judicious selection of local anesthetics, delivered through multiple sites of injection, is likely to provide predictable anesthesia with minimal exposure to side effects.

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Analgesics and therapeutic recommendations

Nonnarcotic analgesics

Management of endodontic pain is multifactorial and directed at reducing the peripheral and central components of hyperalgesia and allodynia through combined endodontic procedures and pharmacotherapy. A major class of drugs for managing endodontic pain comprises the nonnarcotic analgesics, which include both nonsteroidal antiinflammatory drugs (NSAIDs) and acetaminophen. NSAIDs have been shown to be very effective in managing pain of inflammatory origin and, by virtue of their binding to plasma proteins, actually exhibit increased delivery to inflamed tissue via the extravasation of plasma proteins.[33,82,147] Although these drugs classically are thought to produce analgesia through peripheral mechanisms, the CNS is believed to be an additional site of action.[216,326] NSAIDs inhibit the synthesis of PGs by blocking the enzyme cyclooxygenase (COX), which has two known isoforms, COX-1 and COX-2. Some researchers have proposed that a splice variant of COX-1 (i.e., COX-3) is expressed predominantly in the CNS and is the major site of action of acetaminophen.[52,192,255,301] However, recent studies[151] indicate that the antipyretic and analgesic effects of acetaminophen do not involve inhibition of COX-3; they are more likely exerted through effects of an active metabolite on CNS cannabinoid receptors,[14] and this metabolite appears to act by blocking a calcium channel ($Ca_V3.2$).[188]

Numerous NSAIDs are available for the management of pain and inflammation (Table 6.4). Ibuprofen is generally considered the prototype of contemporary NSAIDs and has a well-documented efficacy and safety profile.[80] New formulations of ibuprofen include ibuprofen sodium dihydrate, which is an effective analgesic and has a faster onset of action than ibuprofen acid. Other NSAIDs may offer certain advantages over ibuprofen.[327] For example, etodolac (i.e., Lodine) causes less gastrointestinal (GI) irritation,[16] and ketoprofen (i.e., Orudis) has been shown in some studies to be somewhat

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more analgesic than ibuprofen.[67] An intranasal formulation of ketorolac tromethamine (Sprix, Regency Therapeutics, Shirley, NY) is now available and provides significant pain relief within 30 minutes of administration in patients with endodontic pain.[337] Recent studies suggest that in addition to inhibiting COX, ketorolac and diclofenac inhibit peripheral *N*-methyl-d-aspartate (NMDA) receptors, which may contribute to their analgesic effects.[47,85] The advantages of NSAIDs include their well-established analgesic efficacy for inflammatory pain. Many of the NSAIDs listed in Table 6.4 have been shown to be more effective than traditional acetaminophen and opioid combinations such as acetaminophen with codeine.[66,80,334]

Table 6.4

Summary of Selected Nonnarcotic Analgesics

Analgesic	Trade Name	Dose Range (mg)	Daily Dose (mg)
Acetaminophen	Tylenol and others	325–650	4000
Aspirin	Many	325–1000	4000
Diclofenac potassium	Cataflam	50–100	150–200
Diflunisal	Dolobid	250–1000	1500
Etodolac	Lodine	200–400	1200
Fenoprofen	Nalfon	200	1200
Flurbiprofen	Ansaid	50–100	200–300
Ibuprofen acid	Motrin et al.	200–800	2400 (Rx)
Ibuprofen sodium dihydrate	Fast-acting Advil	256–512	1536
Ketoprofen	Orudis	25–75	300 (Rx)
Ketorolac*	Toradol	30–60 (oral)	60
	Sprix	31.5	126 mg
Naproxen	Naprosyn	250–500	1500
Naproxen Na	Anaprox and others	220–550	1650 (Rx)

*A new package insert for ketorolac tablets includes the instructions that the drug should be used only as a transition from injectable ketorolac and for no longer than 5 days. The package insert for intranasal ketorolac states that for patients 65 years of age or older, renally impaired patients, and patients weighing less than 50 kg, the dose should be limited to 15.75 mg and the maximum daily dose should be limited to 63 mg.

Note: Cyclooxygenase-2 (COX-2) inhibitors are not included (see text). Rx, Prescription strength

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Modified from Cooper SA: Treating acute dental pain, *Postgrad Dent* 2:7, 1995.

A 2002 paper represents the first systematic review comparing all endodontic pain studies evaluating oral NSAIDs.[158] The authors concluded that NSAIDs combined with other drugs (e.g., flurbiprofen with tramadol[86]) or pretreatment and posttreatment application of NSAIDs provides effective pain control. Table 6.5 lists the results of a large ongoing systematic review of the relative efficacy of analgesics in inflammatory pain conditions. Importantly, the data are generated based on postoperative patients having moderate to severe pain, and the number needed to treat (NNT) is based on the relative superiority of the analgesic over placebo for producing a 50% relief in pain. These data, therefore, constitute important, clinically relevant information for clinicians who want to compare the relative efficacy of posttreatment analgesics. Of course other issues, such as the potential adverse effects of drugs and the patient's medical history, must be considered when a treatment plan for postendodontic pain is developed.

Table 6.5

Oxford League Table of Analgesic Efficacy*

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Analgesic†	Number of Patients in Comparison	Percentage With at Least 50% Pain Relief	Number Needed to Treat	Lower Confidence Interval	Higher Confidence Interval
Ibuprofen (800 mg)	76	100	1.6	1.3	2.2
Ketorolac (60 mg) (IM)	116	56	1.8	1.5	2.3
Diclofenac (100 mg)	548	69	1.8	1.6	2.1
Oxycodone IR (5 mg) + acetaminophen (500 mg)	150	60	2.2	1.7	3.2
Diclofenac (50 mg)	738	63	2.3	2	2.7
Naproxen (440 mg)	257	50	2.3	2	2.9
Oxycodone IR (15 mg)	60	73	2.3	1.5	4.9
Ibuprofen (600 mg)	203	79	2.4	2	4.2
Ibuprofen (400 mg)	5456	55	2.5	2.4	2.7
Aspirin (1200 mg)	279	61	2.4	1.9	3.2
Oxycodone IR (10 mg)	315	66	2.6	2	3.5
Ketorolac (10 mg) + acetaminophen (650 mg)	790	50	2.6	2.3	3.1
Ibuprofen (200 mg)	3248	48	2.7	2.5	2.9
Naproxen (500/550)	784	52	2.7	2.3	3.3
Diclofenac (50 mg)	1296	57	2.7	2.4	3.1
Diclofenac (25 mg)	204	54	2.8	2.1	4.3
Demerol (100 mg) (IM)	364	54	2.9	2.3	3.9
Tramadol (150 mg)	561	48	2.9	2.4	3.6

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Morphine (10 mg) (IM)	946	50	2.9	2.6	3.6
Naproxen (500/550 mg)	784	52	2.7	2.3	3.3
Naproxen (220/250 mg)	202	45	3.4	2.4	5.8
Ketorolac (30 mg) (IM)	359	53	3.4	2.5	4.9
Acetaminophen (500 mg)	561	61	3.5	2.2	13.3
Acetaminophen (600/650 mg) + codeine (60 mg)	1123	42	4.2	3.4	5.3
Acetaminophen (650 mg) + dextropropoxyphene (65 mg hydrochloride or 100 mg napsylate)	963	38	4.4	3.5	5.6
Aspirin (600/650 mg)	5061	38	4.4	4	4.9
Acetaminophen (600/650 mg)	1886	38	4.6	3.9	5.5
Tramadol (100 mg)	882	30	4.8	3.8	6.1
Tramadol (75 mg)	563	32	5.3	3.9	8.2
Aspirin (650 mg) + codeine (60 mg)	598	25	5.3	4.1	7.4
Oxycodone IR (5 mg) + acetaminophen (325 mg)	149	24	5.5	3.4	
Ketorolac (10 mg) (IM)	142	48	5.7	3	53
Acetaminophen (300 mg) + codeine (30 mg)	379	26	5.7	4	9.8
Tramadol (50 mg)	770	19	8.3	6	13
Codeine (60 mg)	1305	15	16.7	11	48
Placebo	>10,000	18	N/A	N/A	N/A

*Analgesics are listed in descending order from most to least effective based on the NNT. The NNT reflects the superiority of an analgesic over a placebo treatment; therefore an analgesic with a lower NNT has greater efficacy than an analgesic with a higher NNT. The NNT is calculated for the proportion of patients with at least 50% pain relief over 4 to 6 hours compared with placebo in randomized, double-blind, single-dose studies in patients with moderate to severe pain. The 95% confidence interval contains the upper and lower estimates of the NNT with a 95% chance of accuracy.

†Drugs were given orally except where noted.

IM, Intramuscular; *IR*, immediate release; *NA*, not applicable

Modified from http://www.bandolier.org.uk/booth/painpag/Acutrev/Analgesics/lftab.html.

The introduction of selective inhibitors of COX-2 offered the potential for both analgesic and anti-inflammatory benefits and reduced GI irritation.[81,189]

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However, most selective COX-2 inhibitors have been withdrawn from the market due to prothrombic adverse effects, and the only coxib still available, celecoxib, has not received FDA approval for the treatment of acute inflammatory pain. Concern has been raised that the COX-2 inhibitors may also cause at least some GI irritation in patients with preexisting GI disease.[342]

Another major concern centers on the recognized prothrombic adverse effects of the COX-2 inhibitors. Two meta-analyses have examined the cardiovascular (CV) safety of traditional NSAIDs and COX-2 inhibitors. Kearney et al.[182] conducted a meta-analysis of 138 randomized trials, and McGettigan and Henry[224] conducted a meta-analysis of 23 controlled observational studies.[182,224] Kearney et al.[182] estimated a relative risk for CV events associated with COX-2 to be 1.42 (95% CI, 1.64 to 2.91). Naproxen was found to have no significant adverse effects on the CV system in either meta-analysis. Diclofenac (Voltaren) is a relatively COX-2–selective drug and seems to have a degree of COX-2 selectivity similar to that of celecoxib. Diclofenac was associated with increased CV events. In the randomized trial analysis, there was an increase in CV risk with high-dose ibuprofen. Based on the available data, the FDA has requested that manufacturers of all prescription products containing nonselective NSAIDs revise their product labeling to include (1) a boxed warning regarding the potential serious adverse CV events and the serious, potentially life-threatening GI adverse events associated with the use of this class of drugs; (2) a contraindication to use in patients who have recently undergone coronary artery bypass surgery; and (3) a medication guide for patients regarding the potential for CV and GI adverse events associated with the use of this class of drugs. The available data do not suggest an increased risk of serious CV events for the short-term, low-dose use of NSAIDs available over the counter, but the FDA has requested changes to the label to better inform consumers about the safe use of these products. Given this situation and reasonable alternative NSAIDs, we recommend against the use of COX-2 inhibitors for the treatment of patients with routine endodontic pain.

Limitations and drug interactions

Clinicians should educate themselves not only on the efficacy of the

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nonnarcotic analgesics but also on their limitations and interactions with other drugs.[45] For example, NSAIDs exhibit an analgesic ceiling that limits the maximal level of analgesia; they also induce side effects, including those affecting the GI system (3% to 11% incidence) and the CNS (1% to 9% incidence of dizziness and headache). NSAIDs are contraindicated in patients with ulcers and aspirin hypersensitivity.[199,357] They are also associated with severe GI complications, and the risk of adverse effects increases with an increasing lifetime-accumulated dose of these drugs and the concurrent intake of aspirin, steroids, or coumadin.[15,83,199,357] A strategy for avoiding GI bleeding associated with NSAIDs is to use a proton pump inhibitor (PPI). A combination of a PPI (esomeprazole magnesium) with naproxen (Vimovo, Patheon/Astrozenica, Wilmington, DE) is now available. Although this is a cost-effective approach, it does not protect the lower GI tract.[321] Another approach is to use a histamine receptor H_2 antagonist in combination with an NSAID. The combination of famotidine (an H_2-receptor antagonist) and ibuprofen (Duexis, Horizon Pharma, Northbrook, IL) was recently approved by the FDA. Also, as mentioned earlier, there is an increased CV risk with some NSAIDs, including ibuprofen.

The NSAIDs have been reported to interact with a number of other drugs (Table 6.6). Acetaminophen and opioid combination drugs are alternatives for patients unable to take NSAIDs.[65] Further information is available on the pharmacology and adverse effects of this important class of drugs.[45,63,83,115,364] Other resources are also available for the evaluation of drug interactions, including internet drug search engines such as *rxlist.com*, *Epocrates.com*, and *Endodontics.UTHSCSA.edu*.

Table 6.6

Summary of Selected Drug Interactions With Nonsteroidal Anti-inflammatory Drugs

Drug	Possible Effect
Angiotensin-converting enzyme (ACE) inhibitors	Reduced antihypertensive effectiveness of captopril (and especially indomethacin)
Anticoagulants	Increase in prothrombin time or bleeding with

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	anticoagulants (e.g., coumarins)
Beta blockers	Reduced antihypertensive effects (e.g., propranolol, atenolol, pindolol)
Cyclosporine	Increased risk of nephrotoxicity
Digoxin	Increase in serum digoxin levels (especially ibuprofen, indomethacin)
Dipyridamole	Increased water retention (especially indomethacin)
Hydantoins	Increased serum levels of phenytoin
Lithium	Increased serum levels of lithium
Loop diuretics	Reduced effectiveness of loop diuretics (e.g., furosemide, bumetanide)
Methotrexate	Increased risk of toxicity (e.g., stomatitis, bone marrow suppression)
Penicillamine	Increased bioavailability (especially indomethacin)
Sympathomimetics	Increased blood pressure (especially indomethacin with phenylpropanolamine)
Thiazide diuretics	Reduced antihypertensive effectiveness

Data from Facts and Comparisons: *Drug facts and comparisons*, ed 54, St Louis, 2000, Facts and Comparisons; Gage T, Pickett F: *Mosby's dental drug reference*, ed 5, St Louis, 2000, Mosby; and Wynn R, Meiller T, Crossley H: *Drug information handbook for dentistry*, Hudson, OH, 2000, Lexi-Comp.

Acetaminophen

Acetaminophen (*N*-acetyl *p*-aminophenol) is one of the most commonly used analgesic and antipyretic drugs. Acetaminophen alone is not comparable with ibuprofen in relieving moderate to severe pain; however, a combination of ibuprofen and acetaminophen provides greater pain relief than either drug alone, as evidenced by large systematic reviews.[238] In a randomized double-blind placebo-controlled study, patients who had undergone pulpectomy were administered a single dose of either a combination of acetaminophen (1000 mg) and ibuprofen (600 mg) or ibuprofen (600 mg) alone. The combination of the two analgesics provided greater pain relief in the immediate postoperative period (8 hours) than ibuprofen (600 mg) alone.[230]

Acetaminophen is one of the most common drugs found in combination products for the relief of pain and symptoms of cold or flu. It is considered safe when taken at normal doses; in higher doses, however, acetaminophen causes liver toxicity and is associated with nearly half of the cases of acute

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liver failure in the United States.[201] Most acetaminophen is conjugated in the liver to form inactive metabolites. A small portion is metabolized by the cytochrome P450 system to form *N*-acetyl-*p*-benzoquinone imine (NAPQI), which is very toxic but is generally detoxified by glutathione and converted into nontoxic compounds. Large doses of acetaminophen saturate the main route of metabolism, causing more acetaminophen to be converted to NAPQI. Liver injury occurs once glutathione becomes depleted and NAPQI is allowed to accumulate. To minimize this risk, it has been recommended that healthy adults not take more than 3 g (3000 mg) of acetaminophen in a 24-hour period (*www.tylenolprofessional.com/extra-strength-tylenol-dosage-faq.html*). The FDA requires a boxed warning to be added to the label of all oral prescription drug products that contain acetaminophen. (A boxed warning, the strongest warning that the FDA requires, indicates that the drug carries a significant risk of serious adverse effects.)

Opioid analgesics

Opioids are potent analgesics and are often used in dentistry in combination with acetaminophen, aspirin, or ibuprofen. Most clinically available opioids activate mu opioid receptors at several important sites in the brain and on afferent neurons. Studies indicate that they activate peripheral opioid receptors in dental pulp and that IL injection of morphine significantly reduces pain in endodontic patients and in other inflammatory pain states.[84,97,143] Gender-dependent differences appear to exist in response to at least the kappa opioid agonists.[122] In a randomized controlled clinical trial, women taking a combination of pentazocine and naloxone had significantly less postoperative endodontic pain than men taking the same medications.[294]

Although opioids are effective as analgesics for moderate to severe pain, it is important to remember that they have no anti-inflammatory effects and as such will have no effect on the inflamed periapical tissues. The current opioid epidemic in the United States highlights the risks associated with even a single prescription of opioids—that is, opioid diversion, their misuse, and the development of opioid use disorders. Adverse effects associated with these drugs include nausea, emesis, dizziness, drowsiness, and the potential for respiratory depression and constipation. Because the dosage is limited by side

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effects, these medications are almost always used in combination drugs to manage dental pain. A combination formulation is preferred because it permits a lower dose of the opioid, thereby reducing side effects (Table 6.7).

Table 6.7

Selected Opioid Combination Analgesic Drugs

Formulation	Trade Name*	Possible Rx
APAP 300 mg and codeine 30 mg	Tylenol with codeine no. 3	2 tabs q4h
APAP 500 mg and hydrocodone 5 mg	Vicodin, Lortab 5/500	1–2 tabs q6h
APAP 325 mg and oxycodone 5 mg	Percocet	1 tab q6h
APAP 500 mg and oxycodone 5 mg	Tylox	1 tab q6h
ASA 325 mg and codeine 30 mg	Empirin with codeine no. 3	2 tabs q4h
ASA 325 mg and oxycodone 5 mg	Percodan	1 tab q6h

*Several generics are available for most formulations.

APAP, Acetaminophen; *ASA*, acetylsalicylic acid (aspirin); *Rx*, prescription.

Codeine is often considered the prototype opioid for orally available combination drugs. Most studies have found that the 60-mg dose of codeine (the amount in two tablets of Tylenol with codeine no. 3) produces significantly more analgesia than placebo, although it often produces less analgesia than either aspirin 650 mg or acetaminophen 600 mg.[65,66] In general, patients taking only 30 mg of codeine report about as much analgesia as those taking a placebo.[22,334] Table 6.8 provides comparable doses of other opioids equivalent to 60 mg of codeine.

Table 6.8

Analgesic Doses of Representative Opioids

Opioid	Dose Equivalent to Codeine 60 mg
Codeine	60 mg
Oxycodone	5–6 mg
Hydrocodone	10 mg
Dihydrocodeine	

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	60 mg
Propoxyphene HCl	102 mg
Propoxyphene-N	146 mg
Meperidine	90 mg
Tramadol	50 mg

HCl, hydrochloride; *N*, napsylate

Modified from Troullos E, Freeman R, Dionne RA: The scientific basis for analgesic use in dentistry, *Anesth Prog* 33:123, 1986.

Corticosteroids

Posttreatment pain or a flare-up after endodontic treatment can be attributed to inflammation, infection, or both in the periradicular tissues. Establishing patency and subsequently debriding and shaping the root canal system may irritate the periradicular tissues and inadvertently introduce bacteria, bacterial products, necrotic pulp tissue, or caustic irrigating solution through apical foramina.

In response to this irritation, inflammatory mediators (e.g., PGs, leukotrienes, bradykinin, platelet-activating factor, substance P) are released into the tissues surrounding the apical area of the tooth. As a result, pain fibers are directly stimulated or sensitized, and an increase in vascular dilation and permeability results in edema and increased interstitial tissue pressure.

Glucocorticosteroids are known to reduce the acute inflammatory response by suppressing vasodilation, the migration of polymorphonuclear (PMN) leukocytes, and phagocytosis and by inhibiting the formation of arachidonic acid from neutrophil and macrophage cell membrane phospholipids, thus blocking the COX and lipoxygenase pathways and respective synthesis of PGs and leukotrienes. It is not surprising that a number of investigations have evaluated the efficacy of corticosteroids (administered via either intracanal or systemic routes) in the prevention or control of postoperative endodontic pain or flare-ups.[217]

Intracanal administration

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Several studies have evaluated the intracanal administration of steroids.[51,243,333] A large-scale clinical trial of 223 patients reported significantly less posttreatment pain in patients after intracanal administration of Ledermix (triamcinolone acetonide and demeclocycline HCl) compared with either calcium hydroxide or no intracanal dressing.[93] Intracanal steroids appear to have significant effects for reducing postoperative pain.[287]

Systemic administration

Some studies have evaluated the systemic route of administration of corticosteroids on posttreatment pain or flare-ups.[181,204,218]

In a double-blind placebo-controlled study, patients with irreversible pulpitis were given 4 mg of dexamethasone or placebo by means of a supraperiosteal injection at the apex of the treated tooth after pulpectomy.[229] This is an injection technique that most clinicians would be familiar with (as opposed to intramuscular injection). Posttreatment pain was significantly reduced in the steroid group during the first 24 hours. There was no difference at 48 hours.

Yet another study evaluated the effect of IO injection of methylprednisolone or placebo in patients with irreversible pulpitis. Highly significant pain reduction in the steroid group was maintained for 7 days after a single injection.[123]

Animal studies have histologically evaluated the anti-inflammatory effects of corticosteroids on inflamed periradicular tissues. In one study, after an acute inflammatory reaction was induced in the molar teeth of rats by overextending endodontic instruments, sterile saline or dexamethasone was infiltrated supraperiosteally into the buccal vestibule adjacent to the treated teeth. Dexamethasone significantly reduced the number of neutrophils present and thus had an anti-inflammatory effect on the periradicular tissues of the treated teeth.[246]

Other studies of systemic administration have evaluated the effect of oral administration of corticosteroids on the incidence and severity of posttreatment endodontic pain. In a double-blind controlled clinical trial, 50 patients randomly received either 0.75 mg of dexamethasone or a placebo tablet orally after initial endodontic treatment.[196] Oral dexamethasone significantly reduced posttreatment pain after 8 and 24 hours compared with

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placebo. A follow-up study evaluated the effect a larger oral dose of dexamethasone (i.e., 12 mg given every 4 hours) on the severity of posttreatment endodontic pain.[125] Results showed that dexamethasone was effective in reducing posttreatment endodontic pain up to 8 hours after the treatment was completed; no effect on the severity of pain was seen at 24 and 48 hours after treatment. In a double-blind study comparing etodolac and dexamethasone, the perioperative administration of oral dexamethasone was similar to that of etodolac in reducing pain after endodontic surgery. In a randomized clinical trial,[206] 40 patients received a single dose of prednisolone (30 mg) or placebo 30 minutes before initiation of endodontic treatment. Prednisolone significantly reduced posttreatment pain at 6, 12, and 24 hours compared to placebo.[164]

Collectively, these studies on systemic steroid administration indicate that corticosteroids reduce the severity of posttreatment endodontic pain compared with placebo treatment. However, given the relative safety/efficacy relationship between steroids and NSAIDs, most investigators choose an NSAID as the drug of first choice for postoperative pain control.

Of note is an interesting study reporting that 17.7% of patients on steroid therapy developed dentin hypersensitivity–like pain.[308] A positive correlation was noted between the steroid dose and pain scores. Discontinuation of the steroids resolved the pain in all the study subjects.

Antibiotics

Because bacteria are involved in endodontic cases with apical periodontitis, the incidence of a posttreatment infection or flare-up is a concern to clinicians providing endodontic treatment. Prescribing an antibiotic to prevent such an occurrence might make sense, but the use of antibiotics is controversial for several reasons.[102] First, the overprescribing of antibiotics, especially when these drugs are not indicated, has led to increased bacterial resistance and patient sensitization. Second, antibiotics have been mistakenly prescribed for patients with severe pain who have a vital tooth (i.e., when bacteria are unlikely to be a causative factor in periradicular pain).[367] Third, even when bacteria are likely to be present, data from controlled clinical trials provide little or no support for the hypothesis that antibiotics reduce pain.[183]

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A series of clinical studies evaluated the efficacy of prophylactically administered systemic antibiotics for preventing posttreatment endodontic flare-ups. Working on the premise that the incidence of infectious flare-ups after endodontic treatment is 15%, Morse et al.[242] randomly prescribed a prophylactic dose of either penicillin or erythromycin to patients after endodontic treatment of teeth with a diagnosis of necrotic pulp and chronic periradicular periodontitis (no placebo was used). The results showed that the overall incidence of flare-ups was 2.2%, with no difference between penicillin and erythromycin. Similar results were obtained in a study in which dental students (instead of private practitioners) provided the endodontic treatment.[1] Outcomes found a 2.6% incidence of flare-ups, with no statistically significant differences between penicillin and erythromycin. However, neither the original nor the follow-up study was a randomized placebo-controlled clinical trial. This observation appears to be highly significant for clinical recommendations because in general, randomized controlled studies fail to detect any analgesic benefit from antibiotics, whereas open-label or historical control studies often report profound effects.[102]

To determine whether the timing of administration of an antibiotic altered the occurrence of flare-ups and pain unassociated with flare-ups, researchers analyzed components of two separate prospective studies of patients undergoing endodontic treatment for teeth with necrotic pulps and chronic periradicular periodontitis. In the first study, prophylactic penicillin was provided; in the second study, patients were instructed to take penicillin (or erythromycin if they were allergic to penicillin) at the first sign of swelling.[241,242] The authors concluded that prophylactic administration of antibiotics is preferable to having the patient take antibiotics at the first sign of an infection.

In a multicenter two-part clinical study, 588 consecutive patients received one of nine medications or placebos and were monitored for 72 hours after treatment.[331] The results showed that ibuprofen, ketoprofen, erythromycin, penicillin, and penicillin plus methylprednisolone significantly reduced the severity of pain within the first 48 hours after treatment. The second part of the study then evaluated the incidence of posttreatment pain after obturation of the same teeth in the first phase of the study.[331] Although only 411 of the

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original 588 patients participated in this phase, they were randomly given the same medications or placebo after completion of the obturation appointment. The incidence of posttreatment pain was lower after obturation (5.83%) than after cleaning and shaping (21.76%) of the root canal system, and no significant difference was found in the effectiveness of the various medications and placebo in controlling posttreatment pain after obturation.

Walton and Chiappinelli[345] were concerned that previous studies were uncontrolled, retrospective, or carried out on different patient groups at different times and with different treatment modalities. They conducted a randomized prospective double-blind clinical trial to test the hypothesis that an antibiotic (e.g., penicillin) can prevent a posttreatment endodontic flare-up.[345] Eighty patients with teeth that had necrotic pulp and chronic periradicular periodontitis were randomly divided into three groups. In the first two groups, either penicillin or a placebo was administered 1 hour before and 6 hours after the individual appointments on a double-blind basis. Upon completion of the individual appointments—which included debridement, shaping, and possibly obturation of the root canal system—the patients completed questionnaires at 4, 8, 12, 24, and 48 hours. No significant difference was found among the three groups in the incidence of flare-ups, pain, or swelling. The authors concluded that the use of prophylactic penicillin offers no benefit for postoperative pain or flare-up, and prophylactic administration of penicillin should not be used routinely in patients undergoing endodontic treatment of necrotic teeth and chronic periradicular periodontitis.

In another randomized prospective placebo-controlled clinical study, a group of investigators examined whether supplemental penicillin reduced symptoms or shortened the course of recovery of emergency patients diagnosed with necrotic pulp and acute apical abscess.[103] Patients were randomly given penicillin, a placebo, or no medication. Using a visual analog scale, the subjects then evaluated their postoperative pain and swelling for up to 72 hours. No significant difference was found among the three groups. Recovery occurred as a result of endodontic treatment alone.

It is well recognized that antibiotics may be indicated for the management of infections of endodontic origin. However, a review of the available literature indicates that prophylactic use is contraindicated in

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immunocompetent patients with no systemic signs of infection and with swelling localized to the vestibule. Controlled clinical studies indicate that antibiotics offer little or no benefit for pain reduction under these circumstances, but they may be indicated for immunocompromised patients and for patients with systemic signs and symptoms of an infection or an infection that has spread into the fascial spaces of the head and neck.

Pain management strategies

When pain in an individual patient is being managed, the skilled clinician must customize the treatment plan, balancing the general principles of endodontics, mechanisms of hyperalgesia, and pain management strategies with the particular factors of the individual patient (e.g., medical history, concurrent medications).[147,184,185,291,313]

Effective management of endodontic pain starts with the "three Ds": *diagnosis*, *definitive* dental treatment, and *drugs* (Box 6.1). Comprehensive reviews on *diagnosis* and *definitive* dental treatment (e.g., incision and drainage, pulpectomy) are provided elsewhere in this text. As described earlier in this chapter, the management of endodontic pain should focus on the removal of peripheral mechanisms of hyperalgesia and allodynia (see Box 6.1). This generally requires treatment that removes and reduces causative factors (e.g., bacterial and immunologic factors). For example, both pulpotomy and pulpectomy have been associated with substantial reduction in patient reports of pain compared to pretreatment pain levels.[86,150,222,263] However, pharmacotherapy is often required to reduce continued nociceptor input (e.g., NSAIDs, local anesthetics) and suppress central hyperalgesia (e.g., NSAIDs, opioids).

Box 6.1

Considerations for Effective "Three D" Pain Control

1. Diagnosis
2. Definitive dental treatment
3. Drugs

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- Pretreatment with nonsteroidal anti-inflammatory drugs or acetaminophen when appropriate.
- Use of long-acting local anesthetics when indicated.
- Use of a flexible prescription plan.
- Prescription “by the clock” rather than as needed.

Pretreatment

Treatment with an NSAID before a procedure has been shown to have a significant benefit in many studies[80,162] but not all.[245] The rationale for pretreatment is to block the development of hyperalgesia by reducing the input from peripheral nociceptors. For patients who cannot take NSAIDs, pretreatment with acetaminophen has also been shown to reduce postoperative pain.[236] Patients can be pretreated 30 minutes before the procedure with either an NSAID (e.g., ibuprofen 400 mg or flurbiprofen 100 mg) or acetaminophen 1000 mg.[86,162,236]

Long-acting local anesthetics

A second pharmacologic approach for pain management is the use of long-acting local anesthetics, such as bupivacaine and ropivacaine. Clinical trials indicate that long-acting local anesthetics not only provide anesthesia during the procedure but also significantly delay the onset of posttreatment pain compared with local anesthetics that contain lidocaine.[80,130,131,166] Administration of long-acting local anesthetics by block injection has been shown to reduce posttreatment pain for 2 to 7 days after the oral procedure[130,131,166] because an afferent barrage of nociceptors can induce central hyperalgesia.[360–362] The analgesic benefit of long-acting local anesthetics is more prominent with block injections than infiltration injections, but the clinician should also be aware of adverse effects attributed to these agents.[18,235]

Flexible plan

A third pharmacologic approach is the use of a flexible plan for prescribing analgesics (Fig. 6.21).[13,66,142,144,147,184,186,334] A flexible prescription plan serves to minimize both postoperative pain and side effects. Given these

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goals, the clinician's strategy is twofold: (1) to achieve a maximally effective dose of the nonnarcotic analgesic (either an NSAID or acetaminophen for patients who cannot take NSAIDs) and (2) in the rare cases in which the patient still has moderate to severe pain, to consider adding drugs that increase the NSAID's analgesia. Because of its predictive value, the presence of preoperative pain or mechanical allodynia may be an indicator that such NSAID combinations should be considered.

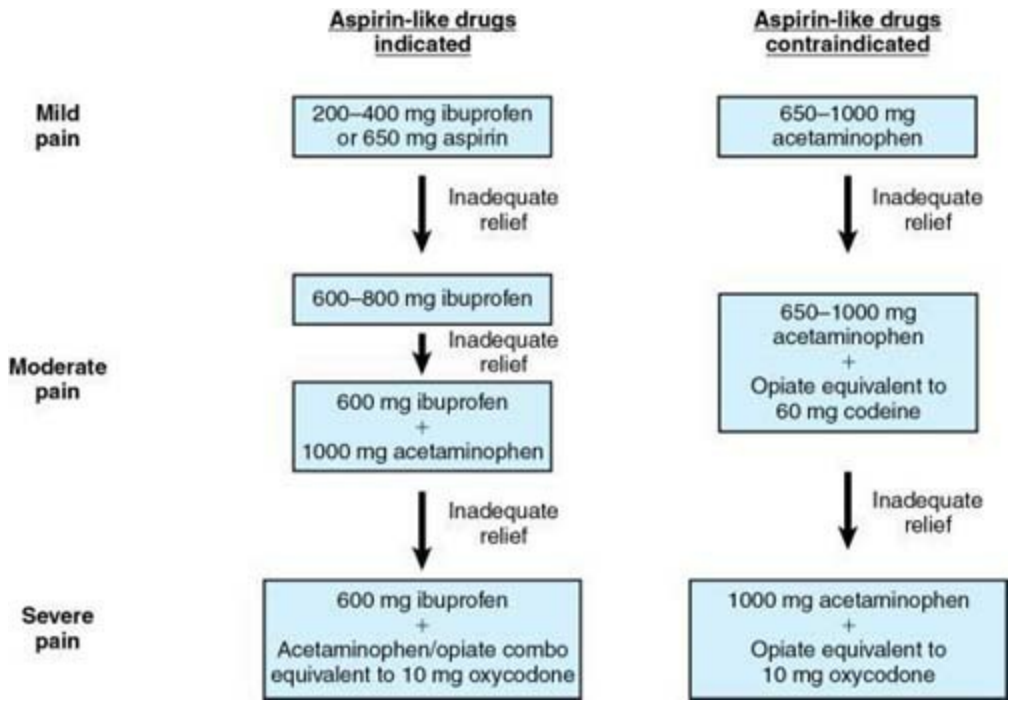

FIG. 6.21 A flexible analgesic strategy.

Schematic of analgesic strategy is as follows: Aspirin-like drugs indicated: mild pain: 200–400 mg ibuprofen or 650 mg aspirin leads to inadequate relief, 600–800 mg ibuprofen again leads to inadequate relief. Moderate pain: 600 mg ibuprofen plus 1000 mg acetaminophen leads to inadequate relief. Severe pain: 600 mg ibuprofen plus Acetaminophen/opiate combo equivalent to 10 mg oxycodone.

Aspirin-like drugs contraindicated: Mild pain: 650–1000 mg 650–1000 mg acetaminophen leads to inadequate relief. Moderate pain: 650 to 1000 mg acetaminophen plus Opiate equivalent to 60 mg codeine. Severe pain: 1000 mg acetaminophen plus opiate equivalent to 10 mg oxycodone.

Most studies report that combining an NSAID with acetaminophen 1000

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mg alone (i.e., no opioid) produces nearly twice the analgesic response as just the NSAID.[37,68,230,350] Administration of ibuprofen 600 mg with acetaminophen 1000 mg produced significant relief of posttreatment endodontic pain compared with ibuprofen alone or placebo (Fig. 6.22). However, in a recent study, the analgesic effect of a combination of ibuprofen 600 mg with acetaminophen 1000 mg did not differ from that of acetaminophen alone.[350] Studies have also shown that concurrent administration of an NSAID and an acetaminophen-opioid combination drug provided significantly greater analgesia than an NSAID alone.[37,323] The concurrent administration of acetaminophen and NSAID appears to be well tolerated, with no detectable increase in side effects or alterations in pharmacokinetics.[37,200,323,363]

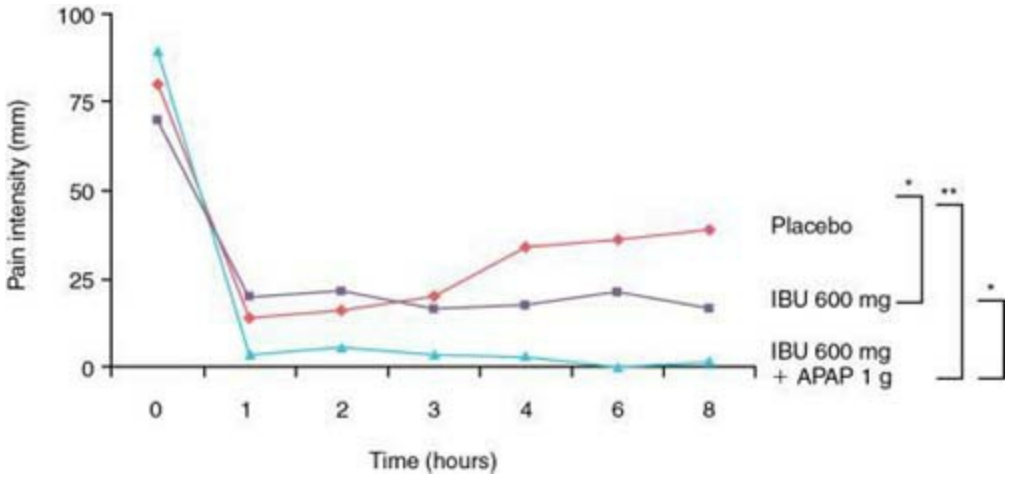

FIG. 6.22 Comparison of ibuprofen *(IBU)* 600 mg plus acetaminophen *(APAP)* 1000 mg with IBU alone or to placebo treatment in postendodontic pain patients.

Graph shows pain intensity (VAS) against time in minutes. The vertical axis ranges from 0 to 100. The horizontal axis ranges from 0 (away from the origin) to 8. The data points for placebo, IBU 600 mg, IBU 600 mg plus APAP 1 g are represented by diamond, square, and triangular blocks, respectively. Three lines connect the data points. The first line representing placebo originates from (0, 80), slopes down, and reaches at (1, 15). The line further moves up, reaches (4, 30), and terminates at (8, 31). The second line of IBU 600 mg originates from (0, 73), intersects the first line at (0. 8, 44), slopes down, fluctuates horizontally, and terminates at (8, 22). The third line of IBU 600 mg plus APAP 1 g originates from (0.3, 88), intersects the first two lines, reaches (1, 2), and terminates at (8, 1). All data are approximate.

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Source: (From Menhinick KA, Gutmann JL, Regan JD, et al: The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study, *Int Endod J* 37:531, 2004.)

In rare cases of severe pain, an NSAID may need to be administered with an opioid. Two general methods are used to combine NSAIDs and opioids to achieve the analgesic benefits of both. The first involves an alternating regimen of an NSAID followed by an acetaminophen-opioid combination.[13,66] Aspirin and opioid combinations are not used in this alternating schedule because of the potential for NSAID/aspirin interactions. The second method involves administration of a single drug consisting of an NSAID-opioid combination. For example, postoperative pain studies have shown that the combination of 200 mg of ibuprofen and 7.5 mg of hydrocodone (Vicoprofen) was about 80% more effective for analgesia than the ibuprofen alone, with about the same incidence of side effects.[353] Other opioids can be added to an NSAID for increased analgesia. A study on posttreatment endodontic pain demonstrated short-term benefits of the combination of flurbiprofen and tramadol.[86] Other NSAID and opioid combinations have also been evaluated.[83] However, the results of clinical trials on the use of NSAIDs alone and NSAIDs combined with acetaminophen (see Fig. 6.22) suggest that opioid combinations may be required only rarely.

Not all patients require concurrent use of NSAIDs with acetaminophen-opioid combinations or combinations of an NSAID and opioid. The basic premise of a flexible prescription plan is that the analgesic prescribed is matched to the patient's need. The major advantage of a flexible plan is that the clinician is prepared for those rare cases when additional pharmacotherapy is indicated to increase the efficacy of pain control. As mentioned, preoperative hyperalgesia may be an indicator for more comprehensive pharmacotherapy. When the clinician is considering the combination of various analgesics, she or he must make sure to use dosing regimens that do not exceed any of the drugs' maximal daily dosage.

Future directions

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COX-inhibiting nitric oxide (NO) donators (CINODs), a new class of NSAIDs, have a NO-donating moiety. These drugs, also known as NO-NSAIDs, were developed with the idea that the released NO would lead to improved vascular tone and mucosal blood flow, thus attenuating the adverse effects of NSAIDs on GI mucosa and blood pressure. Although none of the CINODS have yet been approved by the FDA, they may be available in the near future.

Current use of analgesics has been driven by clinical trials to arrive at doses that obtain the desired level of pain relief with acceptable side-effect profiles. After administration, drugs are absorbed and distributed to their site of action, where they interact with functional targets. They then undergo metabolism and eventual excretion. All of the steps along the way are influenced by a variety of environmental and genetic factors. The ability to predict how a patient's genome may affect the efficacy of a given analgesic drug is being discovered in the field of pain pharmacogenomics (for a review, see Rollason et al.[288]).

One example relevant to pain control in dentistry is the variable efficacy of codeine in a specific population. Many analgesic drugs are metabolized by the cytochrome P450 (CYP) family of hepatic enzymes, and the genes that encode for their biosynthesis have been identified. Codeine is a prodrug that is demethylated to produce morphine, which is responsible for its analgesic action. This demethylation is catalyzed by the enzyme cytochrome P402D6. It has been estimated that 6% to 7% of the Caucasian population has a nonfunctional CYP2D6 mutant allele, making them poor metabolizers of codeine to morphine.[79] These patients may be aware from past experience that codeine is ineffective in their systems and may request a different (usually more potent) form of narcotic. The clinician may suspect drug-seeking behavior when in fact there is a biochemical reason for such a request. Given the current state of clinical DNA analysis, it is not incomprehensible to envision a time when rapid (chairside) genomic evaluation will lead to specific recommendations for analgesic prescriptions.

Summary

The information and recommendations provided in this chapter were selected

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to aid the clinician in the management of acute endodontic pain. Clinical judgment must also take into account other sources of information—the patient history, concurrent medications, the nature of the pain, and the overall treatment plan—when the best means of alleviating patient pain are being determined. An effective approach to managing endodontic pain demands integration of the general principles of pain mechanisms and management with thorough, individualized clinical assessment.

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References

1. Abbott A, et al: *A prospective randomized trial on efficacy of antibiotic prophylaxis in asymptomatic teeth with pulpal necrosis and associated periapical pathosis*, Oral Surg 1988;66: 722.
2. Aelig W, Laurence D, O'Neil R, et al: *Cardiac effects of adrenaline and felypressin as vasoconstrictors in local anaesthesia for oral surgery under diazepam sedation*, Br J Anaesth 1970;42: 174.
3. Aggarwal V, Jain A, Kabi D. *Anesthetic efficacy of supplemental buccal and lingual infiltrations of articaine and lidocaine after an inferior alveolar nerve block in patients with irreversible pulpitis*, J Endod 2009;35: 925.
4. Aggarwal V, Singla M, Kabi D. *Comparative evaluation of anesthetic efficacy of Gow-Gates mandibular conduction anesthesia, Vazirani-Akinosi technique, buccal-plus-lingual infiltrations, and conventional inferior alveolar nerve anesthesia in patients with irreversible pulpitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: 303.
5. Aggarwal V, Singla M, Miglani S, et al: *Efficacy of articaine versus lidocaine administered as supplementary intraligamentary injection after a failed inferior alveolar nerve block: a randomized double-blind study*, J Endod 2019;45: 1.
6. Aggarwal V, Singla M, Miglani S, et al: *A prospective, randomized single-blind evaluation of effect of injection speed on anesthetic efficacy of inferior alveolar nerve block in patients with symptomatic irreversible pulpitis*, J Endod 2012;38: 1578.

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7. Aggarwal V, Singla M, Rizvi A, et al: *Comparative evaluation of local infiltration of articaine, articaine plus ketorolac, and dexamethasone on anesthetic efficacy of inferior alveolar nerve block with lidocaine in patients with irreversible pulpitis*, J Endod 2011;37: 445.
8. Agren E, Danielsson K. *Conduction block analgesia in the mandible: a comparative investigation of the techniques of Fischer and Gow-Gates*, Swed Dent J 1981;5: 91.
9. Akinosi J. *A new approach to the mandibular nerve block*, Br J Oral Surg 1977;15: 83.
10. Aldous J. *Needle deflection: a factor in the administration of local anesthetics*, J Am Dent Assoc 1968;77: 602.
11. Al-Sultan AF. *Effectiveness of pH adjusted lidocaine versus commercial lidocaine for maxillary infiltration anesthesia*, Al-Rafidain Dent J 2004;4: 34.
12. Al-Sultan AF, Fathie WK, Hamid RS. *A clinical evaluation on the alkalization of local anesthetic solution in periapical surgery*, Al-Rafidain Dent J 2006;6: 71.
13. American Association of Endodontists. Post-endodontic pain control 1995; The Association Chicago.
14. Anderson B. *Paracetamol (acetaminophen): mechanisms of action*, Paediatr Anaesth 2008;18: 915.
15. Arnold J, Salom I, Berger A. *Comparison of gastrointestinal microbleeding associated with use of etodolac, ibuprofen, indomethacin, and naproxen in normal subjects*, Curr Ther Res 1985;37: 730.
16. Asarch T, Allen K, Petersen B, et al: *Efficacy of a computerized local anesthesia device in pediatric dentistry*, Pediatr Dent 1999;21: 421.
17. Ashraf H, Kazem M, Dianat O, et al: *Efficacy of articaine versus lidocaine in block and infiltration anesthesia*

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www.konkur.in

administered in teeth with irreversible pulpitis: a prospective, randomized, double-blind study, J Endod 2013;39: 6.

18. Bacsik C, Swift J, Hargreaves K. *Toxic systemic reactions of bupivacaine and etidocaine*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 18.
19. Balasco M, Drum M, Reader A, et al: *Buffered lidocaine for incision and drainage: a prospective, randomized double-blind study*, J Endod 2013;39: 1329.
20. Batinac Sotosek Tokmadzic T, V, Peharda V, et al: *Adverse reactions and alleged allergy to local anesthetics: analysis of 331 patients*, J Dermatol 2013;40: 522.
21. Batista da Silva C, Berto LA, Volpato MC, et al: *Anesthetic efficacy of articaine and lidocaine for incisive/mental nerve block*, J Endod 2010;36: 438.
22. Beaver W. *Mild analgesics: a review of their clinical pharmacology*, Am J Med Sci 1966;251: 576.
23. Benkwitz C, Garrison JC, Linden J, et al: *Lidocaine enhances G alpha I protein function*, Anesthesiology 2003;99: 1093.
24. Berberich G, Reader A, Drum M, et al: *A prospective, randomized, double-blind comparison of the anesthetic efficacy of 2% lidocaine with 1: 100,000 and 1: 50,000 epinephrine and 3% mepivacaine in the intraoral, infraorbital nerve block*, J Endod 2009;35: 1498.
25. Berlin J, Nusstein J, Reader A, et al: *Efficacy of articaine and lidocaine in a primary intraligamentary injection administered with a computer-controlled local anesthetic delivery system*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 361.
26. Berns J, Sadove M. *Mandibular block injection: a method of study using an injected radiopaque material*, J Am Dent

Telegram: @dental_k

www.konkur.in

Assoc 1962;65: 736.
27. Bigby J, Reader A, Nusstein J, et al: *Articaine for supplemental intraosseous anesthesia in patients with irreversible pulpitis*, J Endod 2006;32: 1044.
28. Biocanin V, Brkovic B, Milicic B, et al: *Efficacy and safety of intraseptal and periodontal ligament anesthesia achieved by computer-controlled articaine 1 epinephrine delivery: a dose-finding study*, Clin Oral Investig 2013;17: 525.
29. Birchfield J, Rosenberg PA. *Role of the anesthetic solution in intrapulpal anesthesia*, J Endod 1975;1: 26.
30. Black JA, Liu S, Tanaka M, et al: *Changes in the expression of tetrodotoxin-sensitive sodium channels within dorsal root ganglia neurons in inflammatory pain*, Pain 2004;108: 237.
31. Bonar T, Nusstein J, Reader A, et al: *Anesthetic efficacy of articaine and lidocaine in a primary intraseptal injection: a prospective, randomized double-blind study*, Anesth Prog 2017;64: 203.
32. Bosco DA, Haas DA, Young ER, et al: *An anaphylactoid reaction following local anesthesia: a case report*, Anesth Pain Control Dent 1993;2: 87.
33. Bowles WR, Flores CM, Jackson DL, et al: *Beta 2-adrenoceptor regulation of CGRP release from capsaicin-sensitive neurons*, J Dent Res 2003;82: 308.
34. Bowles WH, Frysh H, Emmons R. *Clinical evaluation of buffered local anesthetic*, Gen Dent 1995;43: 182.
35. Brandt RG, Anderson PF, McDonald NJ, et al: *The pulpal anesthetic efficacy of articaine versus lidocaine in dentistry: a meta-analysis*, J Am Dent Assoc 2011;142: 493.
36. Brannstrom M, Lindskog S, Nordenvall K. *Enamel*

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www.konkur.in

hypoplasia in permanent teeth induced by periodontal ligament anesthesia of primary teeth, J Am Dent Assoc 1984;109: 735.

37. Breivik E, Barkvoll P, Skovlund E. *Combining diclofenac with acetaminophen or acetaminophen-codeine after oral surgery: a randomized, double-blind, single oral dose study*, Clin Pharmacol Ther 1999;66: 625.
38. Brkovic BM, Savic M, Andric M, et al: *Intraseptal vs. periodontal ligament anaesthesia for maxillary tooth extraction: quality of local anaesthesia and haemodynamic response*, Clin Oral Investig 2010;14: 675.
39. Broering R, Reader A, Drum M, et al: *A prospective, randomized comparison of the anesthetic efficacy of the greater palatine and high tuberosity second division nerve blocks*, J Endod 2009;35: 1337.
40. Brunetto PC, Ranali J, Ambrosano GMB, et al: *Anesthetic efficacy of 3 volumes of lidocaine with epinephrine in maxillary infiltration anesthesia*, Anesth Prog 2008;55: 29.
41. Bultema K, Fowler S, Drum M, et al: *Pain reduction in untreated symptomatic irreversible pulpitis using liposomal bupivacaine (Exparel): a prospective, randomized, double-blind trial*, J Endod 2016;42: 1707.
42. Bunczak-Reeh M, Hargreaves K. *Effect of inflammation on delivery of drugs to dental pulp*, J Endod 1998;24: 822.
43. Burns Y, Reader A, Nusstein J, et al: *Anesthetic efficacy of the palatal-anterior superior alveolar injection*, J Am Dent Assoc 2004;135: 1269.
44. Byers MR, Taylor PE, Khayat BG, et al: *Effects of injury and inflammation on pulpal and periapical nerves*, J Endod 1990;16: 78.
45. Byrne B. *Drug interactions: a review and update*, Endod Topics 2004;4: 9.

Telegram: @dental_k

www.konkur.in

46. Cannell H, Kerwala C, Webster K, et al: *Are intraligamentary injections intravascular?*, Br Dent J 1993;175: 281.
47. Cairns BE, Dong XD, Wong H, et al: *Intramuscular ketorolac inhibits activation of rat peripheral NMDA receptors*, J Neurophysiol 2012;107: 3308.
48. Cepeda MS, Tzortzopoulou A, Thackrey M, et al: *Adjusting the pH of lidocaine for reducing pain on injection (review)*, Cochrane Database Syst Rev 2010;8: 12.
49. Certosimo A, Archer R. *A clinical evaluation of the electric pulp tester as an indicator of local anesthesia*, Oper Dent 1996;21: 25.
50. Chamberlain T, Davis R, Murchison D, et al: *Systemic effects of an intraosseous injection of 2% lidocaine with 1:100,000 epinephrine*, Gen Dent 2000;48: 299.
51. Chance K, Lin L, Shovlin F, et al: *Clinical trial of intracanal corticosteroid in root canal therapy*, J Endod 1987;13: 466.
52. Chandrasekharan NV, Dai H, Roos KL, et al: *COX-3, a cyclooxygenase-1 variant inhibited by acetaminophen and other analgesic/antipyretic drugs: cloning, structure, and expression [see comment]*, Proc Natl Acad Sci U S A 2002;99: 13926.
53. Chaney MA, Kerby R, Reader A, et al: *An evaluation of lidocaine hydrocarbonate compared with lidocaine hydrochloride for inferior alveolar nerve block*, Anesth Prog 1991;38: 212.
54. Childers M, Reader A, Nist R, et al: *Anesthetic efficacy of the periodontal ligament injection after an inferior alveolar nerve block*, J Endod 1996;22: 317.
55. Chiu CY, Lin TY, Hsia SH, et al: *Systemic anaphylaxis*

Telegram: @dental_k

www.konkur.in

following local lidocaine administration during a dental procedure, Pediatr Emerg Care 2004;20: 178.

56. Ciancio SG, Hutcheson MC, Ayoub F, et al: *Safety and efficacy of a novel nasal spray for maxillary dental anesthesia*, J Dent Res 2013;92: 43S.
57. Claffey E, Reader A, Nusstein J, et al: *Anesthetic efficacy of articaine for inferior alveolar nerve blocks in patients with irreversible pulpitis*, J Endod 2004;30: 568.
58. Clark S, Reader A, Beck M, et al: *Anesthetic efficacy of the mylohyoid nerve block and combination inferior alveolar nerve block/mylohyoid nerve block*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 557.
59. Clause DW, Zach GW. *Reaction to diphenhydramine hydrochloride (Benadryl) used as a local anesthetic*, Gen Dent 1989;37: 426.
60. Click V, Drum M, Reader A, et al: *Evaluation of the Gow-Gates and Vazirani-Akinosi techniques in patients with symptomatic irreversible pulpitis: a prospective randomized study*, J Endod 2015;41: 16.
61. Coggins R, Reader A, Nist R, et al: *Anesthetic efficacy of the intraosseous injection in maxillary and mandibular teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 634.
62. Cohen H, Cha B, Spangberg L. *Endodontic anesthesia in mandibular molars: a clinical study*, J Endod 1993;19: 370.
63. *Drug facts and comparisons*, St. Louis, 2000, Facts and Comparisons, Inc.
64. Cooley R, Robison S. *Comparative evaluation of the 30-gauge dental needle*, Oral Surg Oral Med Oral Pathol 1979;48: 400.
65. Cooper S. *New peripherally acting oral analgesics*, Annu

Telegram: @dental_k

www.konkur.in

Rev Pharmacol Toxicol 1983;23: 617.
66. Cooper S. *Treating acute dental pain*, Postgrad Dent 1995;2: 7.
67. Cooper S, Berrie R, Cohn P. *The analgesic efficacy of ketoprofen compared to ibuprofen and placebo*, Adv Ther 1988;5: 43.
68. Cooper SA. *The relative efficacy of ibuprofen in dental pain*, Compend Contin Educ Dent 1986;7: 580.
69. Corbett IP, Kanaa MD, Whitworth JM, et al: *Articaine infiltration for anesthesia of mandibular first molars*, J Endod 2008;34: 514.
70. Cromley N, Adams D. *The effect of intraligamentary injections on diseased periodontiums in dogs*, Gen Dent 1991;39: 33.
71. Crout R, Koraido G, Moore P. *A clinical trial of long-acting local anesthetics for periodontal surgery*, Anesth Prog 1990;37: 194.
72. D'Souza J, Walton R, Peterson L. *Periodontal ligament injection: an evaluation of extent of anesthesia and postinjection discomfort*, J Am Dent Assoc 1987;114: 341.
73. Dagher FB, Yared GM, Machtou P. *An evaluation of 2% lidocaine with different concentrations of epinephrine for inferior alveolar nerve block*, J Endod 1997;23: 178.
74. Danielsson K, Evers H, Nordenram A. *Long-acting local anesthetics in oral surgery: an experimental evaluation of bupivacaine and etidocaine for oral infiltration anesthesia*, Anesth Prog 1985;32: 65.
75. Davidson M. *Bevel-oriented mandibular injections: needle deflection can be beneficial*, Gen Dent 1989;37: 410.
76. Davis W, Oakley J, Smith E. *Comparison of the effectiveness of etidocaine and lidocaine as local*

Telegram: @dental_k

www.konkur.in

anesthetic agents during oral surgery, Anesth Prog 1984;31: 159.

77. DeJong R. *Neural blockade by local anesthetics*, J Am Dent Assoc 1997;238: 1383.
78. Dernedde M, Furlan D, Verbesselt R, et al: *Grand mal convulsion after an accidental intravenous injection of ropivacaine*, Anesth Analg 2004;98: 521.
79. Diogenes A, Akopian AN, Hargreaves KM. *NGF upregulates TRPA1: implications for orofacial pain*, J Dent Res 2007;86: 550.
80. Dionne R. *Suppression of dental pain by the preoperative administration of flurbiprofen*, Am J Med Sci 1986;80: 41.
81. Dionne R. *COX-2 inhibitors: better than ibuprofen for dental pain?*, Compendium 1999;20: 518.
82. Dionne RA. *Additive analgesic effects of oxycodone and ibuprofen in the oral surgery model*, J Oral Maxillofac Surg 1999;57: 673.
83. Dionne RA, Berthold C. *Therapeutic uses of non-steroidal anti-inflammatory drugs in dentistry*, Crit Rev Oral Biol Med 2000;12: 315.
84. Dionne RA, Lepinski AM, Gordon SM, et al: *Analgesic effects of peripherally administered opioids in clinical models of acute and chronic inflammation*, Clin Pharmacol Ther 2001;70: 66.
85. Dong XD, Svensson P, Cairns BE. *The analgesic action of topical diclofenac may be mediated through peripheral NMDA receptor antagonism*, Pain 2009;15: 36.
86. Doroshak A, Bowles W, Hargreaves K. *Evaluation of the combination of flurbiprofen and tramadol for management of endodontic pain*, J Endod 1999;25: 660.
87. Dou L, Luo J, Yang D. *Anaesthetic efficacy of supplemental lingual infiltration of mandibular molars*

Telegram: @dental_k

www.konkur.in

after inferior alveolar nerve block plus buccal infiltration in patients with irreversible pulpitis, Int Endod J 2012;120: 42.

88. Dreven LJ, Reader A, Beck M, et al: *An evaluation of an electric pulp tester as a measure of analgesia in human vital teeth*, J Endod 1987;13: 233.
89. Dreyer de V Joubert WP, van Heerden JD, JJ. *The route of periodontal ligament injection of local anesthetic solution*, J Endod 1983;9: 471.
90. Dunbar D, Reader A, Nist R, et al: *Anesthetic efficacy of the intraosseous injection after an inferior alveolar nerve block*, J Endod 1996;22: 481.
91. Dunsky JL, Moore PA. *Long-acting local anesthetics: a comparison of bupivacaine and etidocaine in endodontics*, J Endod 1984;10: 457.
92. Edwards R, Head T. *A clinical trial of intraligamentary anesthesia*, J Dent Res 1989;68: 1210.
93. Ehrmann EH, Messer HH, Adams GG. *The relationship of intracanal medicaments to postoperative pain in endodontics*, Int Endod J 2003;36: 868.
94. Evans G, Nusstein J, Drum M, et al: *A prospective, randomized, double-blind comparison of articaine and lidocaine for maxillary infiltrations*, J Endod 2008;34: 389.
95. Fan S, Chen WL, Pan CB, et al: *Anesthetic efficacy of inferior alveolar nerve block plus buccal infiltration or periodontal ligament injections with articaine in patients with irreversible pulpitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: 89.
96. Farhad A, Razavian H, Shafiee M. *Effect of intraosseous injection versus inferior alveolar nerve block as primary pulpal anaesthesia of mandibular posterior teeth with*

Telegram: @dental_k

www.konkur.in

symptomatic irreversible pulpitis: a prospective randomized clinical trial, Acta Odontol Scand 2018;76: 442.

97. Fehrenbacher J, Sun XX, Locke E, et al: *Capsaicin-evoked iCGRP release from human dental pulp: a model system for the study of peripheral neuropeptide secretion in normal healthy tissue*, Pain 2009;144: 253.
98. Fernandez C, Reader A, Beck M, et al: *A prospective, randomized, double-blind comparison of bupivacaine and lidocaine for inferior alveolar nerve blocks*, J Endod 2005;31: 499.
99. Fernieini EM, Bennett JD, Silverman DG, et al: *Hemodynamic assessment of local anesthetic administration by laser Doppler flowmetry*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 526.
100. Finder R, Moore PA. *Adverse drug reactions to local anesthesia*, Dent Clin North Am 2002;46: 747.
101. Forloine A, Drum M, Reader A, et al: *A prospective, randomized, double-blind comparison of the anesthetic efficacy of two percent lidocaine with 1:100,000 epinephrine and three percent mepivacaine in the maxillary high tuberosity second division nerve block*, J Endod 2010;36: 1770.
102. Fouad A. *Are antibiotics effective for endodontic pain? An evidence-based review*, Endod Topics 2002;3: 52.
103. Fouad A, Rivera E, Walton R. *Penicillin as a supplement in resolving the localized acute apical abscess*, Oral Surg Oral Med Oral Pathol 1996;81: 590.
104. Fowler S, Drum M, Reader A, et al: *Anesthetic success of an inferior alveolar nerve block and supplemental articaine buccal infiltration for molars and premolars in patients with symptomatic irreversible pulpitis*, J Endod

Telegram: @dental_k

www.konkur.in

2016;42: 390.
105. Fowler S, Reader A. *Is the volume of 3.6 mL better than 1.8 mL for inferior alveolar nerve blocks in patients with symptomatic irreversible pulpitis?*, J Endod 2013;39: 970.
106. Fowler S, Reader A, Beck M. *Incidence of missed inferior alveolar nerve blocks in vital asymptomatic subjects and in patients with symptomatic irreversible pulpitis*, J Endod 2015;41: 637.
107. Friedman M, Hochman M. *A 21st century computerized injection system for local pain control*, Compendium 1997;18: 995.
108. Friedman M, Hochman M. *The AMSA injection: a new concept for local anesthesia of maxillary teeth using a computer-controlled injection system*, Quintessence Int 1998;29: 297.
109. Friedman M, Hochman M. *P-ASA block injection: a new palatal technique to anesthetize maxillary anterior teeth*, J Esthet Dent 1999;11: 63.
110. Friedman M, Hochman M. *Using AMSA and P-ASA nerve blocks for esthetic restorative dentistry*, Gen Dent 2001;5: 506.
111. Frommer J, Mele F, Monroe C. *The possible role of the mylohyoid nerve in mandibular posterior tooth sensation*, J Am Dent Assoc 1972;85: 113.
112. Froum SJ, Tarnow D, Caiazzo A, et al: *Histologic response to intraligament injections using a computerized local anesthetic delivery system: a pilot study in mini-swine*, J Periodontol 2000;71: 1453.
113. Fuhs QM, Walker WA III, Gough RW, et al: *The periodontal ligament injection: histological effects on the periodontium in dogs*, J Endod 1983;9: 411.
114. Gaffen AS, Haas DA. *Retrospective review of voluntary*

Telegram: @dental_k

www.konkur.in

reports of nonsurgical paresthesia in dentistry, J Can Dent Assoc 2009;75: 579.

115. Gage T, Pickett F. Mosby's dental drug reference ed 4 2000; Mosby St. Louis.
116. Galbreath J. *Tracing the course of the mandibular block injection*, Oral Surg Oral Med Oral Pathol 1970;30: 571.
117. Galili D, Kaufman E, Garfunkel AA, et al: *Intraligamental anesthesia: a histological study*, Int J Oral Surg 1984;13: 511.
118. Gallatin E, Stabile P, Reader A, et al: *Anesthetic efficacy and heart rate effects of the intraosseous injection of 3% mepivacaine after an inferior alveolar nerve block*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 83.
119. Gallatin J, Nusstein J, Reader A, et al: *A comparison of injection pain and postoperative pain of two intraosseous anesthetic techniques*, Anesth Prog 2003;50: 111.
120. Gallatin J, Reader A, Nusstein J, et al: *A comparison of two intraosseous anesthetic techniques in mandibular posterior teeth*, J Am Dent Assoc 2003;134: 1476.
121. Garisto GA, Gaffen AS, Lawrence HP, et al: *Occurrence of paresthesia after dental local anesthetic administration in the United States*, J Am Dent Assoc 2010;141: 836.
122. Gear RW, Miaskowski C, Gordon NC, et al: *Kappa-opioids produce significantly greater analgesia in women than in men*, Nat Med 1996;2: 1248.
123. Geborek P, Mansson B, Wollheim FA, et al: *Intraarticular corticosteroid injection into rheumatoid arthritis knees improves extensor muscles strength*, Rheumatol Int 1990;9: 265.
124. Giannakopoulos H, Levin LM, Chou JC, et al: *The cardiovascular effects and pharmacokinetics of intranasal*

Telegram: @dental_k

www.konkur.in

tetracaine plus oxymetazoline: preliminary findings, J Am Dent Assoc 2012;143: 872.

125. Glassman G, Krasner P, Morse DR, et al: *A prospective randomized double-blind trial on efficacy of dexamethasone for endodontic interappointment pain in teeth with asymptomatic inflamed pulps*, Oral Surg Oral Med Oral Pathol 1989;67: 96.
126. Glenn B, Drum M, Reader A, et al: *Does liposomal bupivacaine (Exparel) significantly reduce postoperative pain/numbness in symptomatic teeth with a diagnosis of necrosis? A prospective, randomized, double-blind trial*, J Endod 2016;42: 1301.
127. Gold M, Reichling D, Shuster M, et al: *Hyperalgesic agents increase a tetrodotoxin-resistant Na 1 -current in nociceptors*, Proc Natl Acad Sci U S A 1996;93: 1108.
128. Goldberg S, Reader A, Drum M, et al: *A comparison of the anesthetic efficacy of the conventional inferior alveolar, Gow-Gates and Vazirani-Akinosi techniques*, J Endod 2008;34: 1306.
129. Goldstein DS, Dionne R, Sweet J, et al: *Circulatory, plasma catecholamine, cortisol, lipid, and psychological responses to a real-life stress (third molar extractions): effects of diazepam sedation and of inclusion of epinephrine with the local anesthetic*, Psychosom Med 1982;44: 259.
130. Gordon SM. *Blockade of peripheral neuronal barrage reduces postoperative pain*, Pain 1997;306: 264.
131. Gordon SM, Brahim JS, Dubner R, et al: *Attenuation of pain in a randomized trial by suppression of peripheral nociceptive activity in the immediate postoperative period*, Anesth Analg 2002;95: 1351.
132. Gow-Gates G. *Mandibular conduction anesthesia: a new*

Telegram: @dental_k

www.konkur.in

technique using extra-oral landmarks, Oral Surg Oral Med Oral Pathol 1973;36: 321.

133. Gray R, Lomax A, Rood J. *Periodontal ligament injection: with or without a vasoconstrictor?*, Br Dent J 1987;162: 263.
134. Gross R, McCartney M, Reader A, et al: *A prospective, randomized, double-blind comparison of bupivacaine and lidocaine for maxillary infiltrations*, J Endod 2007;33: 1021.
135. Guglielmo A, Reader A, Nist R, et al: *Anesthetic efficacy and heart rate effects of the supplemental intraosseous injection of 2% mepivacaine with 1:20,000 levonordefrin*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 284.
136. Haas DA, Lennon D. *A 21 year retrospective study of reports of paresthesia following local anesthetic administration*, J Can Dent Assoc 1995;61: 319.
137. Haas DA, Pynn BR, Sands TD. *Drug use for the pregnant or lactating patient*, Gen Dent 2000;48: 54.
138. Haase A, Reader A, Nusstein J, et al: *Comparing anesthetic efficacy of articaine versus lidocaine as a supplemental buccal infiltration of the mandibular first molar after an inferior alveolar nerve block*, J Am Dent Assoc 2008;139: 1228.
139. Hanna MN, Elhassan A, Veloso PM, et al: *Efficacy of bicarbonate in decreasing pain on intradermal injection of local anesthetics: a meta-analysis*, Reg Anesth Pain Med 2009;34: 122.
140. Hannan L, Reader A, Nist R, et al: *The use of ultrasound for guiding needle placement for inferior alveolar nerve blocks*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 658.

Telegram: @dental_k

www.konkur.in

141. Harboe T, Guttormsen AB, Aarebrot S, et al: *Suspected allergy to local anaesthetics: follow-up in 135 cases*, Acta Anaesthesiol Scand 2010;54: 536.
142. Hargreaves K. *Neurochemical factors in injury and inflammation in orofacial tissues*, Lund JP Lavigne GJ Dubner R Orofacial pain: basic sciences to clinical management 2000; Quintessence Chicago.
143. Hargreaves K, Joris J. *The peripheral analgesic effects of opioids*, APS J 1993;2: 51.
144. Hargreaves K, Troullos E, Dionne R. *Pharmacologic rationale for the treatment of acute pain*, Dent Clin North Am 1987;31: 675.
145. Hargreaves KM, Jackson DL, Bowles WR. *Adrenergic regulation of capsaicin-sensitive neurons in dental pulp*, J Endod 2003;29: 397.
146. Hargreaves KM, Keiser K. *Local anesthetic failure in endodontics: mechanisms and management*, Endod Topics 2003;1: 26.
147. Hargreaves KM, Keiser K. *New advances in the management of endodontic pain emergencies*, J Calif Dent Assoc 2004;32: 469.
148. Harreld TK, Fowler S, Drum M, et al: *Efficacy of a buffered 4% lidocaine formulation for incision and drainage: a prospective, randomized, double-blind study*, J Endod 2015;41: 1583.
149. Hasse AL, Heng MK, Garrett NR. *Blood pressure and electrocardiographic response to dental treatment with use of local anesthesia*, J Am Dent Assoc 1986;113: 639.
150. Hasselgren G, Reit C. *Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings*, J Endod 1989;15: 254.
151. Hersh EV, Lally ET, Moore PA. *Update on*

Telegram: @dental_k

www.konkur.in

cyclooxygenase inhibitors: Has a third COX isoform entered the fray?, Curr Med Res Opin 2005;21: 1217.

152. Hersh EV, Pinto A, Saraghi M, et al: *Double-masked, randomized, placebo-controlled study to evaluate the efficacy and tolerability of intranasal K305 (3% tetracaine plus 0.05% oxymetazoline) in anesthetizing maxillary teeth*, J Am Dent Assoc 2016;147: 278.
153. Hersh EV, Saraghi M, Moore PA. *Intranasal tetracaine and oxymetazoline: a newly approved drug formulation that provides maxillary dental anesthesia without needles*, Curr Med Res Opin 2016;3: 1.
154. Hinkley SA, Reader A, Beck M, et al: *An evaluation of 4% prilocaine with 1:200,000 epinephrine and 2% mepivacaine with 1:20,000 levonordefrin compared with 2% lidocaine with 1:100,000 epinephrine for inferior alveolar nerve block*, Anesth Prog 1991;38: 84.
155. Hobeich P, Simon S, Schneiderman E, et al: *A prospective, randomized, double-blind comparison of the injection pain and anesthetic onset of 2% lidocaine with 1:100,000 epinephrine buffered with 5% and 10% sodium bicarbonate in maxillary infiltrations*, J Endod 2013;39: 597.
156. Hochman M, Friedman M. *In vitro study of needle deflection: a linear insertion technique versus a bidirectional rotation insertion technique*, Quintessence Int 2000;31: 33.
157. Hollmann MW, Herroeder S, Kurz KS, et al: *Time-dependent inhibition of G protein-coupled receptor signaling by local anesthetics*, Anesthesiology 2004;100: 852.
158. Holstein A, Hargreaves KM, Niederman R. *Evaluation of NSAIDs for treating post-endodontic pain*, Endod Topics

Telegram: @dental_k

www.konkur.in

2002;3: 3.
159. Hull T, Rothwell B. *Intraosseous anesthesia comparing lidocaine and etidocaine (abstract)*, J Dent Res 1998;77: 197.
160. Ingle J, Bakland L. Endodontics (vol 6) 2008; Decker Hamilton, Ontario.
161. Jaber A, Whitworth JM, Corbett IP, et al: *The efficacy of infiltration anaesthesia for adult mandibular incisors: a randomized double-blind cross-over trial comparing articaine and lidocaine buccal and buccal plus lingual infiltrations*, Br Dent J 2010;209: E16.
162. Jackson D, Moore P, Hargreaves K. *Preoperative nonsteroidal anti-inflammatory medication for the prevention of postoperative dental pain*, J Am Dent Assoc 1989;119: 641.
163. Jage J. *Circulatory effects of vasoconstrictors combined with local anesthetics*, Anesth Pain Control Dent 1993;2: 81.
164. Jalalzadeh SM, Mamavi A, Shahriari S, et al: *Effect of pretreatment prednisolone on postendodontic pain: a double-blind parallel-randomized clinical trial*, J Endod 2010;36: 978.
165. Jastak J, Yagiela J. Local anesthesia of the oral cavity 1995; Elsevier Health Science New York.
166. Jebeles JA, Reilly JS, Gutierrez JF, et al: *Tonsillectomy and adenoidectomy pain reduction by local bupivacaine infiltration in children*, Int J Pediatr Otorhinolaryngol 1993;25: 149.
167. Jensen J, Nusstein J, Drum M, et al: *Anesthetic efficacy of a repeated intraosseous injection following a primary intraosseous injection*, J Endod 2008;34: 126.
168. Jeske A, Boschart B. *Deflection of conventional versus*

Telegram: @dental_k

www.konkur.in

nondeflecting dental needles in vitro, Anesth Prog 1985;32: 62.

169. Johnson G, Hlava G, Kalkwarf K. *A comparison of periodontal intraligamental anesthesia using etidocaine HCL and lidocaine HCL*, Anesth Prog 1985;32: 202.
170. Jones VR, Rivera EM, Walton RE. *Comparison of carbon dioxide versus refrigerant spray to determine pulpal responsiveness*, J Endod 2002;28: 531.
171. Jung IY, Kim JH, Kim ES, et al: *An evaluation of buccal infiltrations and inferior alveolar nerve blocks in pulpal anesthesia for mandibular first molars*, J Endod 2008;34: 11.
172. Kanaa MD, Meechan JG, Corbett IP, et al: *Speed of injection influences efficacy of inferior alveolar nerve blocks: a double-blind randomized controlled trial in volunteers*, J Endod 2006;32: 919.
173. Kanaa MD, Whitworth JM, Corbett IP, et al: *Articaine and lidocaine mandibular buccal infiltration anesthesia; a prospective randomized double-blind cross-over study*, J Endod 2006;32: 296.
174. Kanaa MD, Whitworth JM, Corbett IP, et al: *Articaine buccal infiltration enhances the effectiveness of lidocaine inferior alveolar nerve block*, Int Endod J 2009;42: 238.
175. Kanaa MD, Whitworth JM, Meechan JG. *A prospective trial of different supplementary local anesthetic techniques after failure of inferior alveolar nerve block in patients with irreversible pulpitis in mandibular teeth*, J Endod 2012;38: 421.
176. Karkut B, Reader A, Drum M, et al: *A comparison of the local anesthetic efficacy of the extraoral versus the intraoral infraorbital nerve block*, J Am Dent Assoc 2010;141: 185.

Telegram: @dental_k

www.konkur.in

177. Kashyap VM, Desai R, Reddy PB, et al: *Effect of alkalinisation of lignocaine for intraoral nerve block on pain during injection, and speed of onset of anaesthesia*, Br J Oral Maxillofac Surg 2011;49: e72.
178. Katz S, Drum M, Reader A, et al: *A prospective, randomized, double-blind comparison of 2% lidocaine with 1:100,000 epinephrine, 4% prilocaine with 1:200,000 epinephrine and 4% prilocaine for maxillary infiltrations*, Anesth Prog 2010;57: 45.
179. Kaufman E, Solomon V, Rozen L, et al: *Pulpal efficacy of four lidocaine solutions injected with an intraligamentary syringe*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1994;78: 17.
180. Kaufman E, Weinstein P, Milgrom P. *Difficulties in achieving local anesthesia*, J Am Dent Assoc 1984;108: 205.
181. Kaufman E, Heling I, Rotstein I, et al: *Intraligamentary injection of slow-release methylprednisolone for the prevention of pain after endodontic treatment*, Oral Surg Oral Med Oral Pathol 1994;77: 651.
182. Kearney PM, Baigent C, Godwin J, et al: *Do selective cyclo-oxygenase-2 inhibitors and traditional non-steroidal anti-inflammatory drugs increase the risk of atherothrombosis? Meta-analysis of randomised trials [see comment]*, Br Med J 2006;332: 1302.
183. Keenan JV, Farman A, Fedorowicz Z, et al: *A Cochrane systematic review finds no evidence to support the use of antibiotics for pain relief in irreversible pulpitis*, J Endod 2006;32: 87.
184. Keiser K. *Strategies for managing the endodontic pain patient*, Tex Dent J 2003;120: 250.
185. Keiser K, Hargreaves K. *Building effective strategies for*

Telegram: @dental_k

www.konkur.in

the management of endodontic pain, Endod Topics 2002;3: 93.

186. Keiser K, Hargreaves KM. *Strategies for managing the endodontic pain patient*, J Tenn Dent Assoc 2003;83: 24.
187. Kennedy M, Reader A, Beck M, et al: *Anesthetic efficacy of ropivacaine in maxillary anterior infiltration*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 406.
188. Kerckhove N, Mallet C, François A, et al: *Cav3.2 calcium channels: the key protagonist in the supraspinal effect of paracetamol*, Pain 2014;155: 764.
189. Khan AA, Dionne RA. *The COX-2 inhibitors: new analgesic and anti-inflammatory drugs*, Dent Clin North Am 2002;46: 679.
190. Kim S. *Ligamental injection: a physiological explanation of its efficacy*, J Endod 1986;12: 486.
191. Kindler CH, Paul M, Zou H, et al: *Amide local anesthetics potently inhibit the human tandem pore domain background K1 channel TASK-2 (KCNK5)*, J Pharmacol Exp Ther 2003;306: 84.
192. Kis B, Snipes A, Bari F, et al: *Regional distribution of cyclooxygenase-3 mRNA in the rat central nervous system*, Brain Res Mol Brain Res 2004;126: 78.
193. Klein SM, Pierce T, Rubin Y, et al: *Successful resuscitation after ropivacaine-induced ventricular fibrillation*, Anesth Analg 2003;97: 901- [erratum appears in Anesth Analg 98:200, 2004].
194. Knoll-Kohler E, Frie A, Becker J, et al: *Changes in plasma epinephrine concentration after dental infiltration anesthesia with different doses of epinephrine*, J Dent Res 1989;68: 1098.
195. Knoll-Kohler E, Knoller M, Brandt K, et al: *Cardiohemodynamic and serum catecholamine response*

Telegram: @dental_k

www.konkur.in

to surgical removal of impacted mandibular third molars under local anesthesia: a randomized double-blind parallel group and crossover study, J Oral Maxillofac Surg 1991;49: 957.

196. Krasner P, Jackson E. *Management of posttreatment endodontic pain with oral dexamethasone: a double-blind study*, Oral Surg Oral Med Oral Pathol 1986;62: 187.
197. Kung J, McDonagh M, Sedgley CM. *Does articaine provide an advantage over lidocaine in patients with symptomatic irreversible pulpitis? A systematic review and meta-analysis*, J Endod 2015;41: 1784.
198. Lai J, Porreca J, Hunter J, et al: *Voltage-gated sodium channels and hyperalgesia*, Annu Rev Pharmacol 2004;44: 371.
199. Laine L, Bombardier C, Hawkey CJ, et al: *Stratifying the risk of NSAID-related upper gastrointestinal clinical events: results of a double-blind outcomes study in patients with rheumatoid arthritis*, Gastroenterology 2002;123: 1006.
200. Lanza FL, Royer GL, Nelson RS, et al: *Effect of acetaminophen on human gastric mucosal injury caused by ibuprofen*, Gut 1986;27: 440.
201. Larson AM, Polson J, Fontana RJ, et al: *Acute liver failure study G: Acetaminophen-induced acute liver failure: results of a United States multicenter, prospective study [see comment]*, Hepatology 2005;42: 1364.
202. Lawaty I, Drum M, Reader A, et al: *A prospective, randomized, double-blind comparison of 2% mepivacaine with 1:20,000 levonordefrin versus 2% lidocaine with 1:100,000 epinephrine for maxillary infiltrations*, Anesth Prog 2010;57: 139.
203. Lee S, Reader A, Nusstein J, et al: *Anesthetic efficacy of*

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www.konkur.in

the anterior middle superior alveolar (AMSA) injection, Anesth Prog 2004;51: 80.

204. Liesinger A, Marshall F, Marshall J. *Effect of variable doses of dexamethasone on posttreatment endodontic pain*, J Endod 1993;19: 35.
205. Lin L, Lapeyrolerie M, Skribner J, et al: *Periodontal ligament injection: effects on pulp tissue*, J Endod 1985;11: 529.
206. Lin S, Levin L, Emodi O, et al: *Etodolac versus dexamethasone effect in reduction of postoperative symptoms following surgical endodontic treatment: a double-blind study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 814.
207. Linden E, Abrams H, Matheny J, et al: *A comparison of postoperative pain experience following periodontal surgery using two local anesthetic agents*, J Periodontol 1986;57: 637.
208. List G, Meinster F Jr, Nery EB, et al: *Gingival crevicular fluid response to various solutions using the intraligamentary injection*, Quintessence Int 1988;19: 559.
209. Littner MM, Tamse A, Kaffe I. *A new technique of selective anesthesia for diagnosing acute pulpitis in the mandible*, J Endod 1983;9: 116.
210. Loetscher C, Melton D, Walton R. *Injection regimen for anesthesia of the maxillary first molar*, J Am Dent Assoc 1988;117: 337.
211. Malamed S. *The Gow-Gates mandibular block: evaluation after 4,275 cases*, Oral Surg Oral Med Oral Pathol 1981;51: 463.
212. Malamed S. *The periodontal ligament (PDL) injection: an alternative to inferior alveolar nerve block*, Oral Surg Oral Med Oral Pathol 1982;53: 117.

Telegram: @dental_k

www.konkur.in

213. Malamed S. Handbook of local anesthesia ed 6 2013; Mosby/Elsevier St. Louis.
214. Malamed SF, Gagnon S, Leblanc D. *Articaine hydrochloride: a study of the safety of a new amide local anesthetic*, J Am Dent Assoc 2001;132: 177.
215. Malav K, Singariya G, Mohammed S, et al: *Comparison of 0.5% ropivacaine and 0.5% levobupivacaine for sciatic nerve block using Labat approach in foot and ankle surgery*, Turk J Anaesthesiol Reanim 2018;46: 15.
216. Malmberg A, Yaksh T. *Antinociceptive actions of spinal nonsteroidal anti-inflammatory agents on the formalin test in rats*, J Pharmacol Exp Ther 1992;263: 136.
217. Marshall G. *Consideration of steroids for endodontic pain*, Endod Topics 2002;3: 41.
218. Marshall J, Walton R. *The effect of intramuscular injection of steroid on posttreatment endodontic pain*, J Endod 1984;10: 584.
219. Martinez G, Benito P, Fernandez C, et al: *A comparative study of direct mandibular nerve block and the Akinosi technique*, Med Oral 2003;8: 143.
220. Mason R, Drum M, Reader A, et al: *A prospective, randomized, double-blind comparison of 2% lidocaine with 1:100,000 and 1:50,000 epinephrine and 3% mepivacaine for maxillary infiltrations*, J Endod 2009;35: 1173.
221. Matthews R, Drum M, Reader A, et al: *Articaine for supplemental, buccal mandibular infiltration anesthesia in patients with irreversible pulpitis when the inferior alveolar nerve block fails*, J Endod 2009;35: 343.
222. McDougal RA, Delano EO, Caplan D, et al: *Success of an alternative for interim management of irreversible pulpitis*, J Am Dent Assoc 2004;135: 1707.

Telegram: @dental_k

www.konkur.in

223. McEntire M, Nusstein J, Drum M, et al: *Anesthetic efficacy of 4% articaine with 1:100,000 epinephrine versus 4% articaine with 1:200,000 epinephrine as a primary buccal infiltration in the mandibular first molar*, J Endod 2011;37: 450.
224. McGettigan P, Henry D. *Cardiovascular risk and inhibition of cyclooxygenase: a systematic review of the observational studies of selective and nonselective inhibitors of cyclooxygenase 2 [see comment]*, JAMA 2006;296: 1633.
225. McLean C, Reader A, Beck M, et al: *An evaluation of 4% prilocaine and 3% mepivacaine compared with 2% lidocaine (1:100,000 epinephrine) for inferior alveolar nerve block*, J Endod 1993;19: 146.
226. Meechan J. *A comparison of ropivacaine and lidocaine with epinephrine for intraligamentary anesthesia*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 469.
227. Meechan J, Ledvinka J. *Pulpal anesthesia for mandibular central incisor teeth: a comparison of infiltration and intraligamentary injections*, Int Endod J 2002;35: 629.
228. Meechan JG, Rawlins MD. *The effects of two different dental local anesthetic solutions on plasma potassium levels during third molar surgery*, Oral Surg Oral Med Oral Pathol 1988;66: 650.
229. Mehrvarzfar P, Shababi B, Sayyad R, et al: *Effect of supraperiosteal injection of dexamethasone on postoperative pain*, Aust Endod J 2008;34: 25.
230. Menhinick K, Gutmann J, Regan J, et al: *The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-*

Telegram: @dental_k

www.konkur.in

controlled study, Int Endod J 2003;37: 531.

231. Mikesell A, Drum M, Reader A, et al: *Anesthetic efficacy of 1.8 mL and 3.6 mL of 2% lidocaine with 1:100,000 epinephrine for maxillary infiltrations*, J Endod 2008;34: 121.
232. Modaresi J, Dianat O, Soluti A. *Effect of pulp inflammation on nerve impulse quality with or without anesthesia*, J Endod 2008;34: 438.
233. Montagnese TA, Reader A, Melfi R. *A comparative study of the Gow-Gates technique and a standard technique for mandibular anesthesia* J Endod 1984;10: 158.
234. Moore KD, Reader A, Meyers WJ, et al: *A comparison of the periodontal ligament injection using 2% lidocaine with 1:100,000 epinephrine and saline in human mandibular premolars*, Anesth Prog 1987;34: 181.
235. Moore P. *Long-acting local anesthetics: a review of clinical efficacy in dentistry*, Compendium 1990;11: 24.
236. Moore P, Werther JR, Seldin EB, et al: *Analgesic regimens for third molar surgery: pharmacologic and behavioral considerations*, J Am Dent Assoc 1986;113: 739.
237. Moore PA, Dunsky JL. *Bupivacaine anesthesia: a clinical trial for endodontic therapy*, Oral Surg Oral Med Oral Pathol 1983;55: 176.
238. Moore PA, Hersh EV. *Combining ibuprofen and acetaminophen for acute pain management after third-molar extractions: Translating clinical research to dental practice*, J Am Dent Assoc 2013;144: 898.
239. Moore TJ, Walsh CS, Cohen MR. *Reported adverse event cases of methemoglobinemia associated with benzocaine products*, Arch Intern Med 2004;164: 1192.
240. Morais-Almeida M, Gaspar A, Marinho S, et al: *Allergy*

Telegram: @dental_k

www.konkur.in

to local anesthetics of the amide group with tolerance to procaine, Allergy 2003;58: 827.

241. Morse D, Furst ML, Belott RM, et al: *Infectious flare-ups and serious sequelae following endodontic treatment: a prospective randomized trial on efficacy of antibiotic prophylaxis in cases of asymptomatic pulpal-periapical lesions*, Oral Surg Oral Med Oral Pathol 1987;64: 96.
242. Morse D, Furst ML, Belott RM, et al: *Prophylactic penicillin versus erythromycin taken at the first sign of swelling in cases of asymptomatic pulpal-periapical lesions: a comparative analysis*, Oral Surg Oral Med Oral Pathol 1988;65: 228.
243. Moskow A, Morse DR, Krasner P, et al: *Intracanal use of a corticosteroid solution as an endodontic anodyne*, Oral Surg Oral Med Oral Pathol 1984;58: 600.
244. Naftalin L, Yagiela J. *Vasoconstrictors: indications and precautions*, Dent Clin North Am 2002;46: 733.
245. Niv D. *Intraoperative treatment of postoperative pain*, Campbell JN Pain 1996: an updated review 1996; IASP Press Seattle.
246. Nobuhara WK, Carnes DL, Gilles JA. *Anti-inflammatory effects of dexamethasone on periapical tissues following endodontic overinstrumentation*, J Endod 1993;19: 501.
247. Nusstein J, Berlin J, Reader A, et al: *Comparison of injection pain, heart rate increase and post-injection pain of articaine and lidocaine in a primary intraligamentary injection administered with a computer-controlled local anesthetic delivery system*, Anesth Prog 2004;51: 126.
248. Nusstein J, Burns Y, Reader A, et al: *Injection pain and postinjection pain of the palatal-anterior superior alveolar injection, administered with the Wand Plus system, comparing 2% lidocaine with 1:100,000 epinephrine to*

Telegram: @dental_k

www.konkur.in

3% mepivacaine, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 164.

249. Nusstein J, Claffey E, Reader A, et al: *Anesthetic effectiveness of the supplemental intraligamentary injection, administered with a computer-controlled local anesthetic delivery system, in patients with irreversible pulpitis*, J Endod 2005;31: 354.
250. Nusstein J, Kennedy S, Reader A, et al: *Anesthetic efficacy of the supplemental X-tip intraosseous injection in patients with irreversible pulpitis*, J Endod 2003;29: 724.
251. Nusstein J, Lee S, Reader A, et al: *Injection pain and postinjection pain of the anterior middle superior alveolar injection administered with the Wand or conventional syringe*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 124.
252. Nusstein J, Reader A, Beck FM. *Anesthetic efficacy of different volumes of lidocaine with epinephrine for inferior alveolar nerve blocks*, Gen Dent 2002;50: 372- quiz, 376.
253. Nusstein J, Reader A, Nist R, et al: *Anesthetic efficacy of the supplemental intraosseous injection of 2% lidocaine with 1:100,000 epinephrine in irreversible pulpitis*, J Endod 1998;24: 487.
254. Nusstein J, Wood M, Reader A, et al: *Comparison of the degree of pulpal anesthesia achieved with the intraosseous injection and infiltration injection using 2% lidocaine with 1:100,000 epinephrine*, Gen Dent 2005;53: 50.
255. Nusstein JM, Beck M. *Effectiveness of 20% benzocaine as a topical anesthetic for intraoral injections*, Anesth Prog 2003;50: 159.
256. Nuzum FM, Drum M, Nusstein J, et al: *Anesthetic efficacy of articaine for combination labial plus lingual infiltrations versus labial infiltration in the mandibular*

Telegram: @dental_k

www.konkur.in

lateral incisor, J Endod 2010;36: 952.

257. Oleson M, Drum M, Reader A, et al: *Effect of preoperative ibuprofen on the success of the inferior alveolar nerve block in patients with irreversible pulpitis*, J Endod 2010;36: 379.
258. Pacira Pharmaceuticals, Inc. *How DepoFoam works*. http://www.pacira.com/depofoam-platform/how-it-works.php. Accessed June 23, 2015.
259. Papagiannopoulou P, Argiriadou H, Georgiou M, et al: *Preincisional local infiltration of levobupivacaine vs ropivacaine for pain control after laparoscopic cholecystectomy*, Surg Endosc 2003;17: 1961.
260. Parente SA, Anderson RW, Herman WW, et al: *Anesthetic efficacy of the supplemental intraosseous injection for teeth with irreversible pulpitis*, J Endod 1998;24: 826.
261. Parirokh M, Sadeghi A, Nakhaee N, et al: *Effect of topical anesthesia on pain during infiltration injection and success of anesthesia for maxillary central incisors*, J Endod 2012;38: 1553.
262. Parirokh M, Yosefi MH, Nakhaee N, et al: *The success rate of bupivacaine and lidocaine as anesthetic agents in inferior alveolar nerve block in teeth with irreversible pulpitis without spontaneous pain*, Restor Dent Endod 2015;40: 155.
263. Penniston S, Hargreaves K. *Evaluation of periapical injection of ketorolac for management of endodontic pain*, J Endod 1996;22: 55.
264. Pereira LA, Groppo FC, Bergamaschi CD, et al: *Articaine (4%) with epinephrine (1:100,000 or 1:200,000) in intraosseous injections in symptomatic irreversible pulpitis of mandibular molars: anesthetic efficacy and*

Telegram: @dental_k

www.konkur.in

cardiovascular effects, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2013;116: e85.

265. Pertot W, Dejou J. *Bone and root resorption: effects of the force developed during periodontal ligament injections in dogs*, Oral Surg Oral Med Oral Pathol 1992;74: 357.
266. Peterson J, Matsson L, Nation W. *Cementum and epithelial attachment response to the sulcular and periodontal ligament injection techniques*, Pediatr Dent 1983;5: 257.
267. Peurach J. *Pulpal response to intraligamentary injection in cynomolgus monkey*, Anesth Prog 1985;32: 73.
268. Pfeil L, Drum M, Reader A, et al: *Anesthetic efficacy of 1.8 milliliters and 3.6 milliliters of 2% lidocaine with 1:100,000 epinephrine for posterior superior alveolar nerve blocks*, J Endod 2010;36: 598.
269. Piccinni C, Gissi DB, Gabusi A, et al: *Paraesthesia after local anaesthetics: an analysis of reports to the FDA Adverse Event Reporting System*, Basic Clin Pharmacol Toxicol 2015;117: 52.
270. Plamondon T, Walton R, Graham G, et al: *Pulp response to the combined effects of cavity preparation and periodontal ligament injection*, Oper Dent 1990;15: 86.
271. Pogrel M. *Permanent nerve damage from inferior alveolar nerve blocks: an update to include articaine*, J Calif Dent Assoc 2007;35: 217.
272. Pogrel M. *Permanent nerve damage from inferior alveolar nerve blocks: a current update*, J Calif Dent Assoc 2012;40: 795.
273. Poorni S, Veniashok B, Senthilkumar AD, et al: *Anesthetic efficacy of four percent articaine for pulpal anesthesia by using inferior alveolar nerve block and buccal infiltration techniques in patients with irreversible*

Telegram: @dental_k

www.konkur.in

pulpitis: a prospective randomized double-blind clinical trial, J Endod 2011;37: 1603.

274. Primosch RE, Robinson L. *Pain elicited during intraoral infiltration with buffered lidocaine*, Am J Dent 1996;9: 5.
275. Quercia R, Voleman CI. *Liposomal bupivacaine: a long-acting local anesthetic for postsurgical analgesia*, Clin Pharmacol 2012;47: 212.
276. Rawson R, Orr D. *Vascular penetration following intraligamental injection*, J Oral Maxillofac Surg 1985;43: 600.
277. Reader A, Nusstein J, Drum M. Successful local anesthesia for restorative dentistry and endodontics ed 2. Hanover Park 2017; Quintessence IL.
278. Reemers T, Glickman G, Spears R, et al: *The efficacy of the IntraFlow intraosseous injection as a primary anesthesia technique*, J Endod 2008;34: 280.
279. Reisman D, Reader A, Nist R, et al: *Anesthetic efficacy of the supplemental intraosseous injection of 3% mepivacaine in irreversible pulpitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84: 676.
280. Reitz J, Reader A, Nist R, et al: *Anesthetic efficacy of a repeated intraosseous injection given 30 minutes following an inferior alveolar nerve block/intraosseous injection*, Anesth Prog 1998;45: 143.
281. Reitz J, Reader A, Nist R, et al: *Anesthetic efficacy of the intraosseous injection of 0.9 mL of 2% lidocaine (1:100,000 epinephrine) to augment an inferior alveolar nerve block*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86: 516.
282. Replogle K, Reader A, Nist R, et al: *Anesthetic efficacy of the intraosseous injection of 2% lidocaine (1:100,000 epinephrine) and 3% mepivacaine in mandibular first*

Telegram: @dental_k

www.konkur.in

molars, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 30.

283. Replogle K, Reader A, Nist R, et al: *Cardiovascular effects of intraosseous injections of 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine*, J Am Dent Assoc 1999;130: 649.
284. Roahen JO, Marshall FJ. *The effects of periodontal ligament injection on pulpal and periodontal tissues*, J Endod 1990;16: 28.
285. Robertson D, Nusstein J, Reader A, et al: *Anesthetic efficacy of articaine and lidocaine in buccal infiltration injections of the mandibular first molar*, J Am Dent Assoc 2007;138: 1104.
286. Robison SF, Mayhew RB, Cowan RD, et al: *Comparative study of deflection characteristics and fragility of 25-, 27-, and 30-gauge short dental needles*, J Am Dent Assoc 1984;109: 920.
287. Rogers MJ, Johnson BR, Remeikis NA, et al: *Comparison of effect of intracanal use of ketorolac tromethamine and dexamethasone with oral ibuprofen on post treatment endodontic pain*, J Endod 1999;25: 381.
288. Rollason V, Samer C, Piguet V, et al: *Pharmacogenetics of analgesics: toward the individualization of prescription*, Pharmacogenomics 2008;9: 905.
289. Rood J. *The nerve supply of the mandibular incisor region*, Br Dent J 1977;143: 227.
290. Rood JP. *Adverse reaction to dental local anesthetic injection: "allergy" is not the cause*, Br Dent J 2000;189: 380.
291. Rosenberg P. *Clinical strategies for managing endodontic pain*, Endod Topics 2002;3: 78.
292. Rosenquist J, Rosenquist K, Lee P. *Comparison between*

Telegram: @dental_k

www.konkur.in

lidocaine and bupivacaine as local anesthetics with diflunisal for postoperative pain control after lower third molar surgery, Anesth Prog 1988;35: 1.

293. Roy M, Nakanishi T. *Differential properties of tetrodotoxin-sensitive and tetrodotoxin-resistant sodium channels in rat dorsal root ganglion neurons*, J Neurosci 1992;12: 2104.
294. Ryan JF, Jureidini B, Hodges JS, et al: *Gender differences in analgesia for endodontic pain*, J Endod 2008;34: 552.
295. Saadoun AP, Malamed S. *Intraseptal anesthesia in periodontal surgery*, J Am Dent Assoc 1985;111: 249.
296. Saatchi M, Khademi A, Baghaei B, et al: *Effect of sodium bicarbonate-buffered lidocaine on the success of inferior alveolar nerve block for teeth with symptomatic irreversible pulpitis: a prospective, randomized double-blind study*, J Endod 2015;41: 33.
297. Saatchi M, Shafiee M, Khademi A, et al: *Anesthetic efficacy of Gow-Gates nerve block, inferior alveolar nerve block, and their combination in mandibular molars with symptomatic irreversible pulpitis: a prospective, randomized clinical trial*, J Endod 2018;44: 384.
298. Salomen M, Forsell H, Sceinin M. *Local dental anesthesia with lidocaine and adrenalin: effects on plasma catecholamines*, heart rate, and blood pressure, Int J Oral Maxillofac Surg 1988;17: 392.
299. Sampaoi RM, Carnaval TG, Lanfredi CB, et al: *Comparison of the anesthetic efficacy between bupivacaine and lidocaine in patients with irreversible pulpitis of mandibular molar*, J Endod 2012;38: 594.
300. Schleder JR, Reader A, Beck M, et al: *The periodontal ligament injection: a comparison of 2% lidocaine, 3%*

Telegram: @dental_k

www.konkur.in

mepivacaine, and 1:100,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine in human mandibular premolars, J Endod 1988;14: 397.

301. Schwab JM, Schluesener HJ, Meyermann R, et al: *COX-3 the enzyme and the concept: steps towards highly specialized pathways and precision therapeutics?*, Prostaglandins Leukot Essent Fatty Acids 2003;69: 339.
302. Scott J, Drum M, Reader A, et al: *Efficacy of a repeated infiltration to prolong duration of pulpal anesthesia in maxillary lateral incisors*, J Am Dent Assoc 2009;140: 318.
303. Seng G, Kraus K, Cartridge G. *Confirmed allergic reactions to amide local anesthetics*, Gen Den 1996;44: 52.
304. Shahi S, Rahimi S, Yavari HR, et al: *Success rate of 3 injection methods with articaine for mandibular first molars with symptomatic irreversible pulpitis: a CONSORT randomized double-blind clinical trial*, J Endod 2018;44: 1462.
305. Shapiro MR, McDonald NJ, Gardner RJ, et al: *Efficacy of articaine versus lidocaine in supplemental infiltration for mandibular first versus second molars with irreversible pulpitis: a prospective, randomized, double-blind clinical trial*, J Endod 2018;44: 523.
306. Sherman MG, Flax M, Namerow K, et al: *Anesthetic efficacy of the Gow-Gates injection and maxillary infiltration with articaine and lidocaine for irreversible pulpitis*, J Endod 2008;34: 656.
307. Shojaei A, Haas D. *Local anesthetic cartridges and latex allergy: a literature review*, J Can Dent Assoc 2002;68: 622.
308. Shoji N, Enod Y, Iikubo M, et al: *Dentin hypersensitivity-*

Telegram: @dental_k

www.konkur.in

like tooth pain seen in patients receiving steroid therapy: An exploratory study, Pharm Sci 2016 2016;132: 187.

309. Simon F, Reader A, Drum M, et al: *A prospective, randomized single-blind study of the anesthetic efficacy of the inferior alveolar nerve block administered with a peripheral nerve stimulator*, J Endod 2010;36: 429.
310. Simpson M, Drum M, Reader A, et al: *Effect of preoperative ibuprofen/acetaminophen on the success of the inferior alveolar nerve block in patients with symptomatic irreversible pulpitis*, J Endod 2011;37: 593.
311. Singla M, Subbiya A, Aggarwal V, et al: *Comparison of the anesthetic efficacy of different volumes of 4% articaine (1.8 and 3.6 mL) as supplemental buccal infiltration after failed inferior alveolar nerve block*, Int Endod J 2015;48: 103.
312. Sinnott CJ, Strichartz GR. *Levobupivacaine versus ropivacaine for sciatic nerve block in the rat*, Reg Anesth Pain Med 2003;28: 294.
313. Siqueira J, Barnett F. *Interappointment pain: mechanisms, diagnosis, and treatment*, Endod Topics 2004;3: 93.
314. Sisk A. *Evaluation of the Akinosi mandibular block technique in oral surgery*, Oral Maxillofac Surg 1986;44: 113.
315. Smith G, Pashley D. *Periodontal ligament injection: evaluation of systemic effects*, Oral Surg Oral Med Oral Pathol 1983;56: 571.
316. Smith G, Walton R. *Periodontal ligament injections: distribution of injected solutions*, Oral Surg Oral Med Oral Pathol 1983;55: 232.
317. Smith G, Walton R, Abbott B. *Clinical evaluation of periodontal ligament anesthesia using a pressure syringe*,

Telegram: @dental_k

www.konkur.in

J Am Dent Assoc 1983;107: 953.

318. Smolinske SC. *Review of parenteral sulfite reactions*, J Toxicol Clin Toxicol 1992;30: 597.
319. Sood R, Hans MK, Shetty S. *Comparison of anesthetic efficacy of 4% articaine with 1:100,000 epinephrine and 2% lidocaine with 1:80,000 epinephrine for inferior alveolar nerve block in patients with irreversible pulpitis*, J Clin Exp Dent 2014;6: 520.
320. Sorensen H, Skidmore L, Rzasa R, et al: *Comparison of pulpal sodium channel density in normal teeth to diseased teeth with severe spontaneous pain (abstract)*, J Endod 2004;30: 287.
321. Spiegel BM, Chiou CF, Ofman JJ. *Minimizing complications from nonsteroidal antiinflammatory drugs: cost-effectiveness of competing strategies in varying risk groups*, Arthritis Rheum 2005;53: 185.
322. Stabile P, Reader A, Gallatin E, et al: *Anesthetic efficacy and heart rate effects of the intraosseous injection of 1.5% etidocaine (1:200,000 epinephrine) after an inferior alveolar nerve block*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 407.
323. Stambaugh J, Drew J. *The combination of ibuprofen and oxycodone/acetaminophen in the management of chronic cancer pain*, Clin Pharmacol Ther 1988;44: 665.
324. Steinkruger G, Nusstein J, Reader A, et al: *The significance of needle bevel orientation in success of the inferior alveolar nerve block*, J Am Dent Assoc 2006;137: 1685.
325. Strichartz G. *Molecular mechanisms of nerve block by local anesthetics*, Anesthesiology 1967;45: 421.
326. Svensson CI, Yaksh TL. *The spinal phospholipase-cyclooxygenase-prostanoid cascade in nociceptive*

Telegram: @dental_k

www.konkur.in

processing, Annu Rev Pharmacol Toxicol 2002;42: 553.
327. Taggar TJ, Wu D, Khan AA. *A randomized clinical trial comparing two ibuprofen formulations in patients with acute odontogenic pain*, J Endod 2017;43: 674.
328. Teplitsky P, Hablichek C, Kushneriuk J. *A comparison of bupivacaine to lidocaine with respect to duration in the maxilla and mandible*, J Can Dent Assoc 1987;53: 475.
329. Todorovic L, Stajcic Z, Petrovic V. *Mandibular versus inferior alveolar dental anaesthesia: clinical assessment of 3 different techniques*, Int J Oral Maxillofac Surg 1986;15: 733.
330. Tolas AG, Pflug AE, Halter JB. *Arterial plasma epinephrine concentrations and hemodynamic responses after dental injection of local anesthetic with epinephrine*, J Am Dent Assoc 1982;104: 41.
331. Torabinejad M, Dorn SO, Eleazer PD, et al: *Effectiveness of various medications on postoperative pain following root canal obturation*, J Endod 1994;20: 427.
332. Tortamano IP, Siviero M, Costa CG, et al: *A comparison of the anesthetic efficacy of articaine and lidocaine in patients with irreversible pulpitis*, J Endod 2009;35: 165.
333. Trope M. *Relationship of intracanal medicaments to endodontic flare-ups*, Endod Dent Traumatol 1990;6: 226.
334. Troullos E, Freeman R, Dionne R. *The scientific basis for analgesic use in dentistry*, Anesth Prog 1986;33: 123.
335. Troullos ES, Goldstein DS, Hargreaves KM, et al: *Plasma epinephrine levels and cardiovascular response to high administered doses of epinephrine contained in local anesthesia*, Anesth Prog 1987;34: 10.
336. Troullos ES, Hargreaves KM, Goldstein DS, et al: *Epinephrine suppresses stress-induced increases in plasma immunoreactive beta-endorphin in humans*, J Clin

Telegram: @dental_k

www.konkur.in

Endocrinol Metab 1989;69: 546.

337. Turner CL, Eggleston GW, Lunos S, et al: *Sniffing out endodontic pain: use of an intranasal analgesic in a randomized clinical trial*, J Endod 2011;37: 439.

338. Vanderheyden PJ, Williams RA, Sims TN. *Assessment of ST segment depression in patients with cardiac disease after local anesthesia*, J Am Dent Assoc 1989;119: 407.

339. VanGheluwe J, Walton R. *Intrapulpal injection: factors related to effectiveness*, Oral Surg Oral Med Oral Pathol 1997;19: 38.

340. Vreeland DL, Reader A, Beck M, et al: *An evaluation of volumes and concentrations of lidocaine in human inferior alveolar nerve block*, J Endod 1989;15: 6.

341. Wali M, Drum M, Reader A, Nusstein J. *Prospective, randomized, single-blind study of the anesthetic efficacy of 1.8 and 3.6 milliliters of 2% lidocaine with 1:50,000 epinephrine for the inferior alveolar nerve block*, J Endod 2010;36: 1459.

342. Wallace J. *Selective COX-2 inhibitors: Is the water becoming muddy?*, Trends Pharmacol Sci 1999;20: 4.

343. Wallace JA, Michanowicz AE, Mundell RD, et al: *A pilot study of the clinical problem of regionally anesthetizing the pulp of an acutely inflamed mandibular molar*, Oral Surg Oral Med Oral Pathol 1985;59: 517.

344. Walton R, Abbott B. *Periodontal ligament injection: a clinical evaluation*, J Am Dent Assoc 1981;103: 571.

345. Walton R, Chiappinelli J. *Prophylactic penicillin: effect on posttreatment symptoms following root canal treatment of asymptomatic periapical pathosis*, J Endod 1993;19: 466.

346. Walton RE. *Distribution of solutions with the periodontal ligament injection: clinical, anatomical, and histological*

Telegram: @dental_k

www.konkur.in

evidence, J Endod 1986;12: 492.

347. Warren CA, Mok L, Gordon S, et al: *Quantification of neural protein in extirpated tooth pulp*, J Endod 2008;34: 7.
348. Webster S Jr, Drum M, Reader A, et al: *How effective is supplemental intraseptal anesthesia in patients with symptomatic irreversible pulpitis?*, J Endod 2016;42: 1453.
349. Wells JE, Bingham V, Rowland KC, et al: *Expression of Nav1.9 channels in human dental pulp and trigeminal ganglion*, J Endod 2007;33: 1172.
350. Wells LK, Drum M, Nusstein J, et al: *Efficacy of Ibuprofen and ibuprofen/acetaminophen on postoperative pain in symptomatic patients with a pulpal diagnosis of necrosis*, J Endod 2011;37: 1608.
351. Whitcomb M, Drum M, Reader A, et al: *A prospective, randomized double-blind study of the anesthetic efficacy of sodium bicarbonate buffered 2% lidocaine with 1:100,000 epinephrine in inferior alveolar nerve blocks*, Anesth Prog 2010;57: 59.
352. White JJ, Reader A, Beck M, et al: *The periodontal ligament injection: a comparison of the efficacy in human maxillary and mandibular teeth*, J Endod 1988;14: 508.
353. Wideman G, Keffer M, Morris E, et al: *Analgesic efficacy of a combination of hydrocodone with ibuprofen in postoperative pain*, Clin Pharmacol Ther 1999;65: 66.
354. Wilburn-Goo D, Lloyd L. *When patients become cyanotic: acquired methemoglobinemia*, J Am Dent Assoc 1999;130: 826.
355. Willett J, Reader A, Drum M, et al: *The anesthetic efficacy of diphenhydramine and the combination of diphenhydramine/lidocaine for the inferior alveolar nerve*

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block, J Endod 2009;34: 1446.

356. Wilson S, Johns P, Fuller P. *The inferior alveolar and mylohyoid nerves: an anatomic study and relationship to local anesthesia of the anterior mandibular teeth*, J Am Dent Assoc 1984;108: 350.
357. Wolf M, Lichtenstein D, Singh G. *Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs*, N Engl J Med 1999;340: 1888.
358. Wood M, Reader A, Nusstein J, et al: *Comparison of intraosseous and infiltration injections for venous lidocaine blood concentrations and heart rate changes after injection of 2% lidocaine with 1:100,000 epinephrine*, J Endod 2005;31: 435.
359. Woodmansey K. *Intraseptal anesthesia: a review of a relevant injection technique*, Gen Dent 2005;53: 418.
360. Woolf C. *Evidence for a central component of post-injury pain hypersensitivity*, Nature 1983;306: 686.
361. Woolf C. *Windup and central sensitization are not equivalent*, Pain 1996;66: 105.
362. Woolf C. *Transcriptional and posttranslational plasticity and the generation of inflammatory pain*, Proc Natl Acad Sci U S A 1999;96: 7723.
363. Wright C, Antal EJ, Gillespie WR, et al: *Ibuprofen and acetaminophen kinetics when taken concurrently*, Clin Pharmacol Ther 1983;34: 707.
364. Wynn R, Meiller T, Crossley H. Drug information handbook for dentistry 2000; Lexi-Comp Hudson, Ohio.
365. Wynn RL, Bergman SA, Meiller TF. *Paresthesia associated with local anesthetics: a perspective on articaine*, Gen Dent 2003;51: 498.
366. Yared GM, Dagher FB. *Evaluation of lidocaine in human inferior alveolar nerve block*, J Endod 1997;23: 575.

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367. Yingling NM, Byrne BE, Hartwell GR. *Antibiotic use by members of the American Association of Endodontists in the year 2000: report of a national survey*, J Endod 2002;28: 396.
368. Yonchak T, Reader A, Beck M, et al: *Anesthetic efficacy of infiltrations in mandibular anterior teeth*, Anesth Prog 2001;48: 55.
369. Yucel E, Hutchison I. *A comparative evaluation of the conventional and closed mouth technique for inferior alveolar nerve block*, Aust Dent J 1995;40: 15.
370. Zarei M, Ghoddusi J, Sharifi E, et al: *Comparison of the anesthetic efficacy of and heart rate changes after periodontal ligament or intraosseous X-tip injection in mandibular molars: a randomized controlled clinical trial*, Int Endod J 2012;45: 921.
371. Zorian EV, Sharagin NV. *Comparative evaluation of the topical action of anesthetics on the dental tissues in experimental conditions*, Stomatologiia 1974;53: 1.

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*Any discussion of local anesthesia over the last 40 years would inevitably lead to conversations surrounding the work of Dr. Al Reader. Although this chapter no longer bears his name, it is based on his tireless efforts, astute research mind, and never-ending quest to provide positive patient experiences in dentistry through clinical research. The authors would like to thank Dr. Reader for all of his contributions to local anesthesia and for being the guiding force that has inspired countless others to continue in the search for better local anesthesia and pain control.

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7: Tooth morphology and pulpal access cavities

James L. Gutmann, Bing Fan

CHAPTER OUTLINE

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The dental pulp presents with a variety of configurations and shapes throughout the dentition. Knowledge of the complexity of the root canal system is essential to understand the principles and problems encountered in access preparation from a traditional and contemporary standpoint. This chapter describes and illustrates current perspectives on tooth morphology and explains the techniques necessary to achieve direct access to the root canal system.

Initial anatomical evaluation of the pulpal anatomy is achieved with careful evaluation of two or more periapical radiographs, exposed at different horizontal angulations of the x-ray cone, and is essential, along with cone-beam computed tomography (CBCT) scans in some clinical situations (see Chapter 2). The detailed reading and interpreting of each radiograph before

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and during root canal procedures is necessary because many teeth present with unusual canal morphology. Unfortunately, the interpretation of traditional radiographs may not always result in the correct morphologic assessment, particularly when only a buccolingual view is taken. Furthermore, the recent development of microcomputed tomography (μCT) scanning of teeth has greatly increased clinical assessment of these complexities and three-dimensional (3D) relationships found in root canal systems. Therefore the use of a vast array of tools, such as magnification and illumination, is essential. This approach is particularly helpful in posterior teeth, where a vast array of anatomical complexities can be observed (Fig. 7.1).

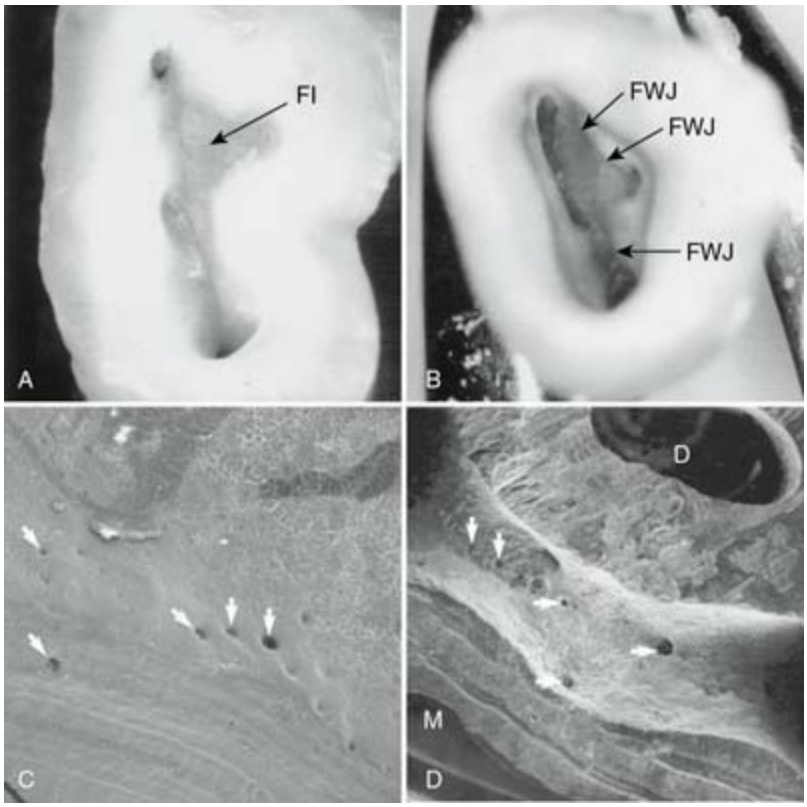

FIG. 7.1 **A,** The color of the pulp chamber floor *(FI)* is always darker than the walls. The orifices are usually located at the junction of the walls and the floor *(FWJ)*. **B,** The same is true for the maxillary first premolar. **C,** Electron photomicrograph of the pulp chamber floor of a mandibular first molar (×20). **D,** Electron photomicrograph of the furcation surface of a mandibular first molar (×30). *D,* Distal canal; *M,* mesial canals; *arrows*, accessory foramina. (**A** and **B**, From Krasner P, Rankow HJ: Anatomy of the pulp chamber floor, *J Endod* 30:5–16,

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2004.)

A) Cut specimen of a tooth shows dark chamber, marked FI.

B) Cut specimen of the first premolar shows the junction between light periphery tissues and a dark chamber inside, marked FWJ.

C) Electron photomicrograph of first molar shows holes in the pulp.

D) Electron photomicrograph of first molar shows well-defined dark patches in mesial and distal canals. The central diagonal strip has perforated tissue with holes.

The use of the dental operating microscope (DOM) that provides superior magnification, increased lighting, and enhanced visibility is recommended to determine the location of root canal orifices in the properly prepared coronal access (Fig. 7.2),[92,105] and has been shown to be superior to the use of the naked eye and magnifying loupes for this assessment.[11,96] As with all advances in technology, however, there are varying points of concurrence and disagreement. For example, one group of investigators determined that dental loupes and the DOM were equally effective for locating mesiopalatal canals in maxillary molars,[13] whereas other studies determined that the DOM did not significantly enhance the ability to locate canals.[34]

FIG. 7.2 The dental operating microscope (DOM) has vastly improved locating the position of the coronal canal anatomy.

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A dentist with illuminated probe in gloved hands examines the teeth of a patient under DOM, with an assistant standing opposite to him.

Components of the pulp cavity

The pulp cavity (Fig. 7.3) is divided into two main portions: the pulp chamber, located in (or extending to just below) the anatomic crown of the tooth, and the root canal (or canals), found in the anatomic root. Other notable features are the pulp horns; accessory, lateral, and furcation canals; canal orifices; isthmus; fins; apical deltas; and apical foramina. The outline of the pulp cavity generally corresponds to the external contour of the tooth. However, factors such as physiologic aging, pathosis, trauma, and occlusion all can modify its dimensions through the production of dentin or reparative (irregular secondary, irritational, and tertiary) dentin (Fig. 7.4).[100]

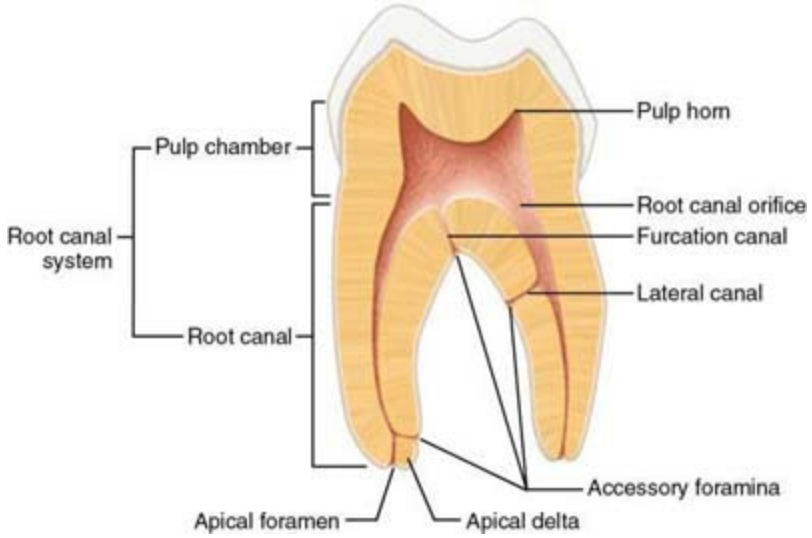

FIG. 7.3 Major anatomic components of the pulp cavity.

Cross-section diagram of a tooth shows labels for pulp horn, root canal orifice, furcation canal, lateral canal, accessory foramina, apical delta, apical foramen, Root canal system: pulp chamber and root canal.

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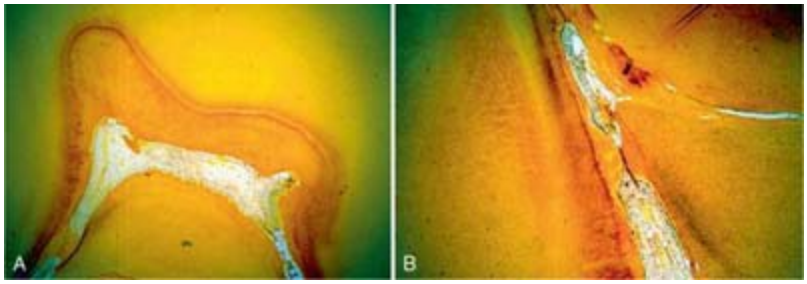

FIG. 7.4 **A,** Formation of reparative dentin and reduction of the pulp chamber space. **B,** Calcific narrowing and closing of a root canal as it leaves the pulp chamber.

A) Tooth shows a heart shaped crown with pulp below.

B) Tooth shows wedge shaped root canal closed at the center.

Source: (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics*, ed 5, St. Louis, 2011, Elsevier.)

In single-rooted teeth, pulp chamber extends to the most apical portion of the cervical margin of the crown; in double/multirooted teeth, it extends to the floor of chamber located in the coronal third of the root.[2] The pulp horns are important because the pulp in them is often exposed by caries, trauma, or mechanical invasion, which usually necessitates vital pulp or root canal procedures. Also, the pulp horns undergo rapid mineralization, along with reduction of the size and shape of the pulp chamber because of the formation of reparative/irritational dentin over time.

The root canal begins as a funnel-shaped canal orifice, generally at or just apical to the cervical line, and ends at the apical foramen (AF), which opens onto the root surface at or within 3 mm of the center of the root apex.[14,35,36,87,113,115] Nearly all root canals are curved, particularly in a faciolingual direction, thereby posing problems during enlargement and shaping procedures because they are not evident on a standard two-dimensional (2D) radiograph. In most cases, the number of root canals corresponds to the number of roots; however, an oval root may have more than one canal.

Accessory canals are minute canals that extend in a horizontal, vertical, or lateral direction from the pulp space to the periodontium. In 74% of cases they are found in the apical third of the root, in 11% in the middle third, and in 15% in the cervical third.[113] The diameter, length, shape, and undulation

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may vary among accessory canals.[122] Apical deltas are multiple accessory canals that branch out from the main canal at or near the root apex (Figs. 7.5 and 7.6) (see Videos 7.1 and 7.2 online at the Expert Consult site). Accessory canals contain connective tissue and vessels but may not supply the pulp with sufficient circulation to form a collateral source of blood flow. They are formed by the entrapment of periodontal vessels in Hertwig's epithelial root sheath during mineralization.[24] They may play a significant role in the communication of disease processes, serving as avenues for the passage of irritants, primarily from the pulp to the periodontium, although communication of inflammatory processes may occur from either tissue.

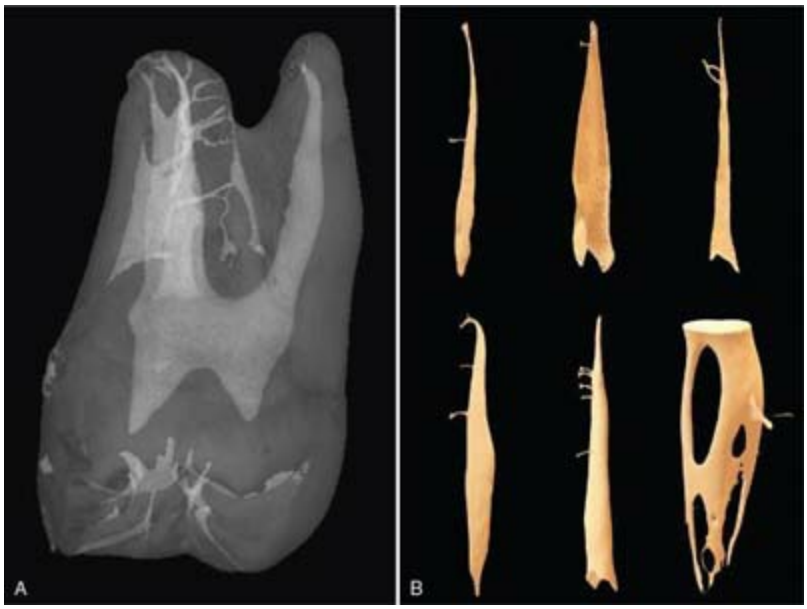

FIG. 7.5 **A,** μCT reconstruction image of a maxillary molar showing the location of accessory canal. **B,** Morphological variations of accessory canal.

A) Maxillary molar shows the additional branched canal extending along one root canal.

B) Six types of canals are shown as follows: Wedge-shaped with the tapering end and a secondary branch near the center.

• Two pulp horns with a secondary branch near the tip.

• Triangular shape with secondary branches forming a loop near the tip.

• Curved tip with two secondary branches below the center.

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• Two pulp horns with multiple branches on the left.

• Fused branches with hollow chambers.

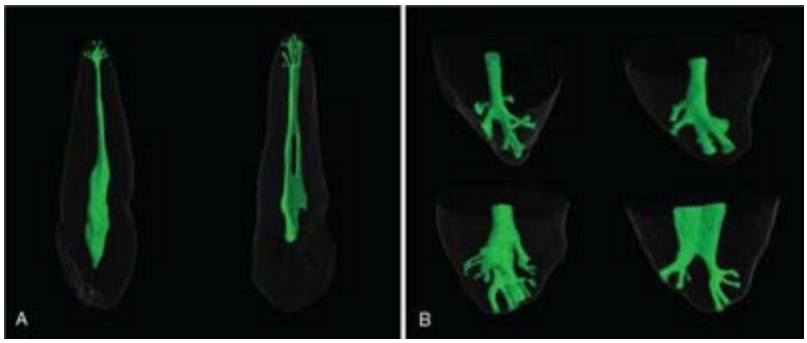

FIG. 7.6 **A,** Apical delta in maxillary canine and maxillary second premolar. **B,** Morphological variations of apical delta.

A) First tooth shows multiple branches at the root tip. Second tooth shows an oval chamber in the canal that extends from the neck.

B) Structure on top-left shows conical end with multiple branches. Structure on top-right shows a slightly diverted funnel shaped root apex with dense multiple branches. Structure on bottom-left shows a U-shaped root apex with dense fused multiple branches. Structure on bottom-right shows a U-shaped root apex that has a bifurcated canal with multiple branches.

Accessory canals that are present in the bifurcation or trifurcation of multirooted teeth are referred to as *furcation canals* (or *chamber canals*) (Fig. 7.7).[37,113,114] These channels form as a result of the entrapment of periodontal vessels during the fusion of the diaphragm, which becomes the pulp chamber floor.[24] In mandibular molars, these canals occur in three distinct patterns (Fig. 7.8) (see Expert Consult site—Tables 7.1 and 7.2). Pulpal inflammation can communicate to the periodontium via these canals and result in furcation lesions in the absence of demonstrable periodontal disease (Fig. 7.9). Likewise, the long-term presence of periodontal furcation lesions may influence the viability of the coronal or radicular pulp tissue when these aberrant channels are present.[39]

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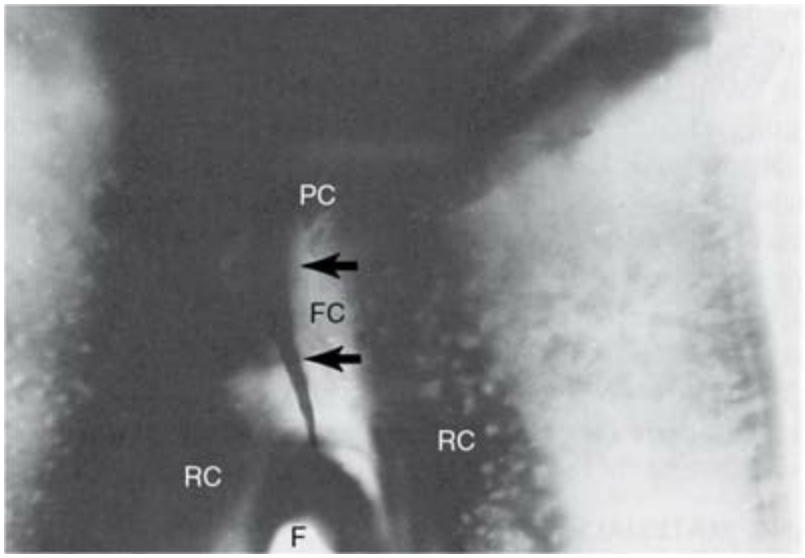

FIG. 7.7 Mandibular first molar showing a furcation canal *(FC, arrows)*. *F,* Furcation; *PC,* pulp chamber floor; *RC,* root canal.

Radiograph of first molar shows dark regions at the center, marked PC, and either sides of bifurcated root, marked RC. The light region along and in-between the root canals are marked FC and C, respectively.

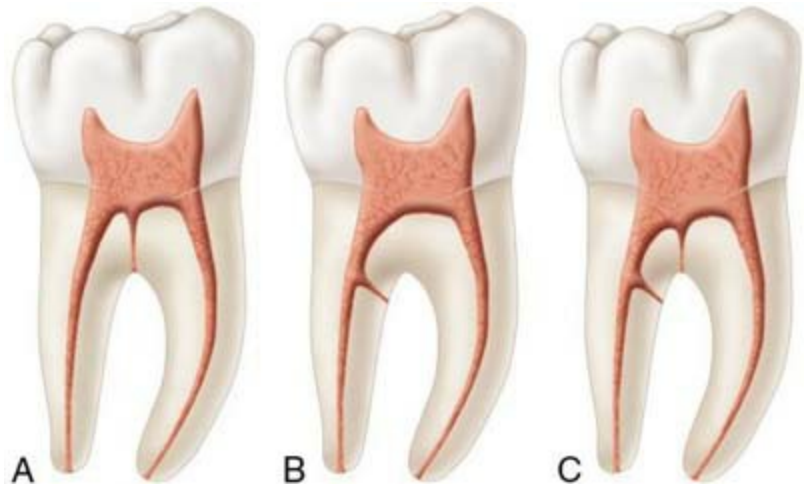

FIG. 7.8 Accessory canals occur in three distinct patterns in the mandibular first molars. **A,** In 13% a single furcation canal extends from the pulp chamber to the intraradicular region. **B,** In 23% a lateral canal extends from the coronal third of a major root canal to the furcation region (80% extend from the distal root canal). **C,** About 10% have both lateral and furcation canals.

Three models, marked A through C, show the anatomy of a double-rooted tooth with different pattern of accessory canals.

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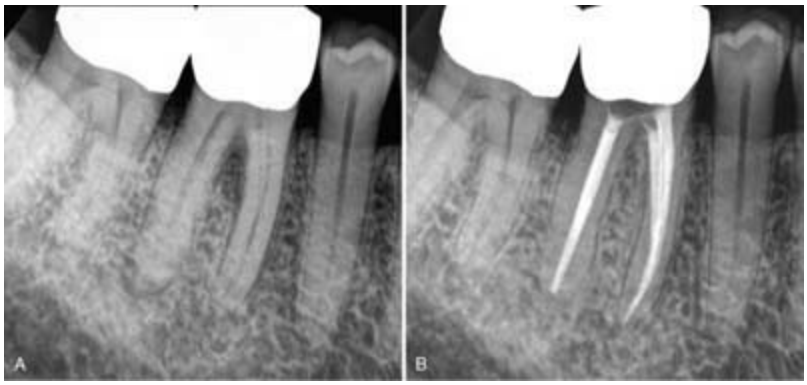

FIG. 7.9 **A,** Furcation lesion. **B,** Healing occurred after treatment.

A) Radiograph shows first molar with radiopaque crown and radiolucent root canals with thin porous tissue inside.

B) Radiograph shows filled root canals of the first molar.

Root canal anatomy

The significance of canal anatomy has been underscored by studies that demonstrated that the natural variations in canal geometry had a greater effect on the changes that occurred during enlargement and shaping than did the instrumentation techniques used to achieve these objectives.[84–86] Most recently, Versiani and co-investigators have detailed the complete pulpal anatomy of the permanent dentition. In doing so they have both verified the findings of the last 125 years and have prepared the formative knowledge base for future studies.[110]

Multiple anatomic variations present in teeth are seen in Fig. 7.10. The only tooth that showed all eight possible configurations is the maxillary second premolar (see Expert Consult site—Tables 7.3 and 7.4).

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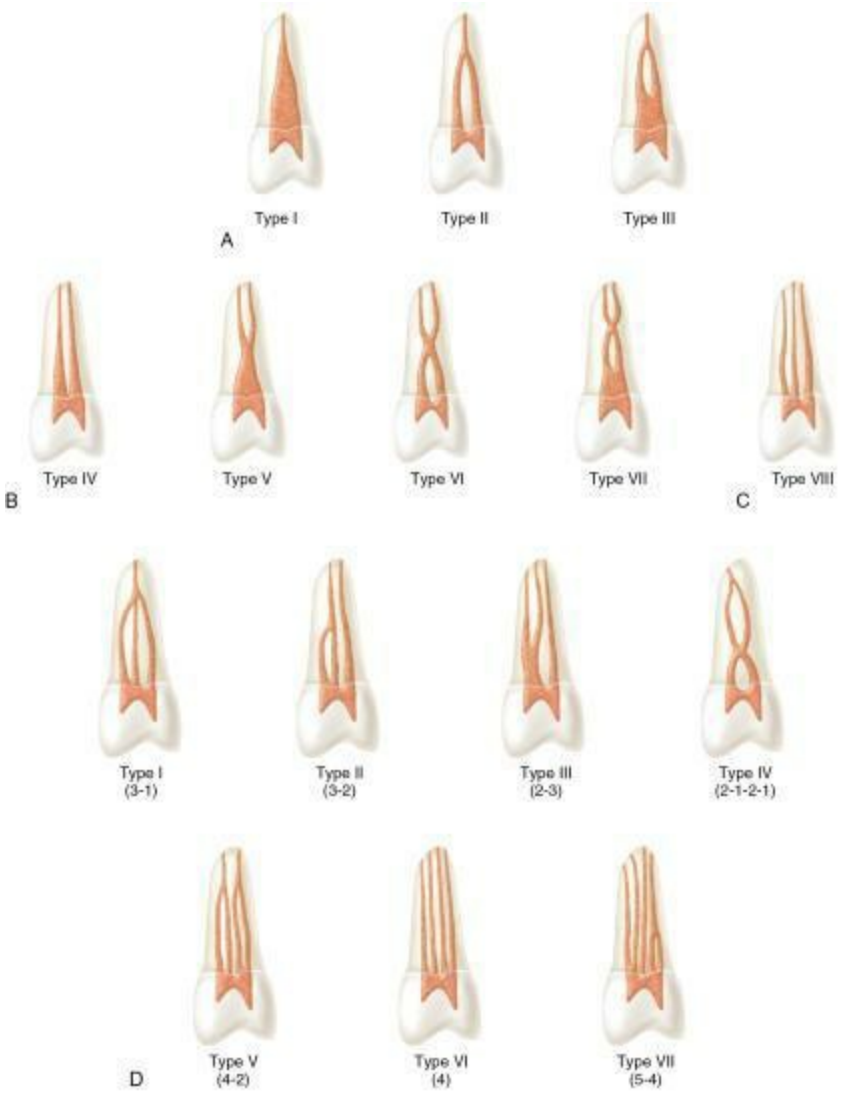

FIG. 7.10 Diagrammatic representation of canal configurations. **A,** One canal at apex based on the work of Vertucci. **B,** Two canals at apex based on the work of Vertucci. **C,** Three canals at apex based on the work of Vertucci. **D,** Supplemental canal configurations based on the work of Gulabivala and colleagues on a Burmese population.

A) Three teeth, type 1 to 3 show a large canal extending from pulp chamber to apex with two pulp horns, a large oval chamber in the single canal, and a small oval chamber near the root apex in the canal, respectively.

B) Two teeth, type 4 and type 5, show a bifurcated canal extending from the pulp chamber and the canal branched into two near the root apex, respectively.

C) Three teeth, type 6 to 8, show a large oval chamber with a bifurcation near the root apex, a small oval chamber with bifurcation toward the root apex, and trifurcation beginning from just below the pulp horns, respectively.

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D) Eight teeth, type 1 to 8, show bilocular canal (3-1), two root canals with an additional branch forming a loop (3-2), bifurcated canal with an additional branch at the center (2-3), two-chambered canal forming numeral 8 (2-1-2-1), canal forming two branches each with a hollow chamber (4-2), canal with four branches (4), and canal with multiple branches (5-4), respectively.

Source: (From Vertucci FJ, Seelig A, Gillis R: Root canal morphology of the human maxillary second premolar, *Oral Surg Oral Med Oral Pathol* 38:456–464, 1974.)

Similar observations have been described in large population studies with the exceptions that one canal was found in 23% of maxillary laterals, 55% of mesiobuccal roots of maxillary second molars, and 30% of distal roots of mandibular second molars.[17,119] Differences in these studies may be due to variations in the ethnic and racial populations studied. A well-recognized ethnic variant is the higher incidence of single-rooted and C-shaped mandibular second molars in Native American and Asians populations (Figs. 7.11 and 7.12),[26,27,64,65,118] which may also be seen in mandibular premolars and some maxillary molars. Recently, three classification system have been proposed, including (1) root and canal anomalies, (2) root and root canal configurations, and (3) accessory canal morphology that provides more detailed information on the morphological features of teeth and root canals essential for diagnosis and treatment, as well as training and research.[1–3]

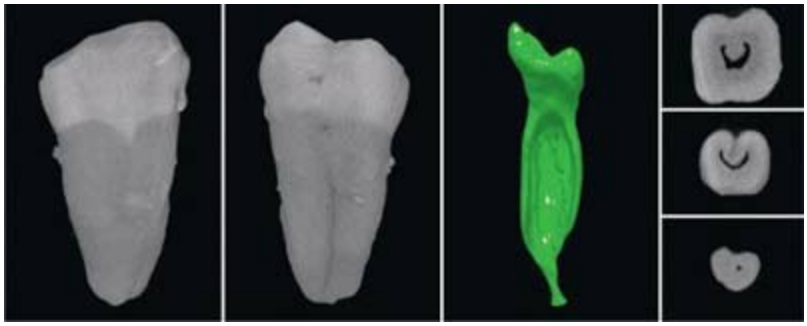

FIG. 7.11 Common variant of a C-shaped canal anatomy found in Native Americans and Asian populations.

Three models of molar are accompanied by their cross-sectional views. First molar shows a roughly U-shaped canal with two prolongations at the base. Second molar shows continuous C-shaped canal. Third molar shows a small hole at the center.

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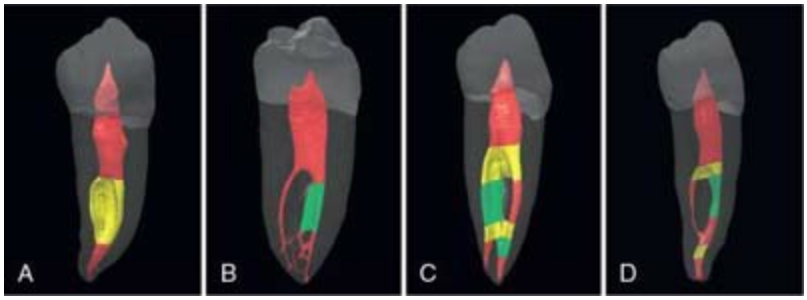

FIG. 7.12 μCT scans of mandibular premolars reflect a wide variation in canal morphology, moving from coronal to apical sections. The red color indicates a non–C-shaped canal space; green indicates a semilunar canal shape; and yellow indicates a continuous C-shaped configuration.

A) Tooth shows the canal with red color at the top, followed by yellow, and red at the base.

B) Tooth shows the canal with multiple branches toward the root apex. The canal has red region, followed by green, and again red.

C) Tooth shows the canal with an oval chamber and red region at the top, followed by yellow, green (left side)-red (right side), yellow, and red (left side)-and green followed by red (right side).

D) Tooth shows a large hollow chamber with a red region at the top, followed by yellow, red (left side)-green (right side), red, yellow, and red.

Clinical determination of the root canal configuration

Coronal considerations

Examination of the pulp chamber floor may reveal clues to the location of orifices and to the type of canal system present. If only one canal is present, it usually is located in the center of the access preparation. If only one orifice is found and it is not in the center of the root, another orifice probably exists on the opposite side (Fig. 7.13). The relationship of the two orifices to each other is also significant (Fig. 7.14). The closer they are, the greater the chance the two canals join at some point in the body of the root. As the distance between orifices in a root increases, the greater is the chance the canals will remain separate. The more separation between orifices, the less the degree of canal curvature.[19]

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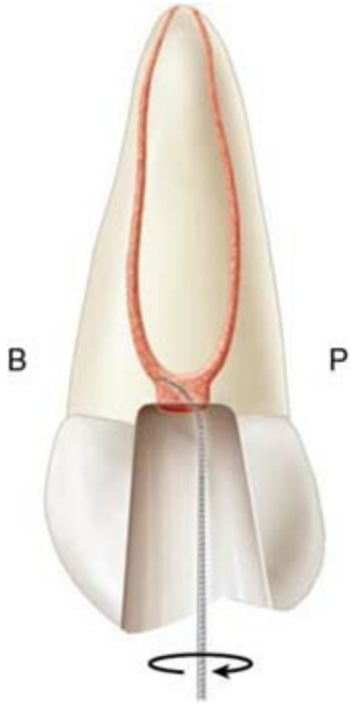

FIG. 7.13 An oval orifice must be explored with apically curved small instruments. When trying to locate the buccal canal, the clinician should place the file tip in the orifice with the tip curved to the buccal side. To explore for the palatal canal, a curved file tip is placed toward the palate. *B,* Buccal; *P,* palatal.

Tooth structure with two canals show an inverted U-shape chamber in the crown area and an instrument being inserted in the pulp. Buccal and palatal regions are marked on the left and right of the tooth, respectively.

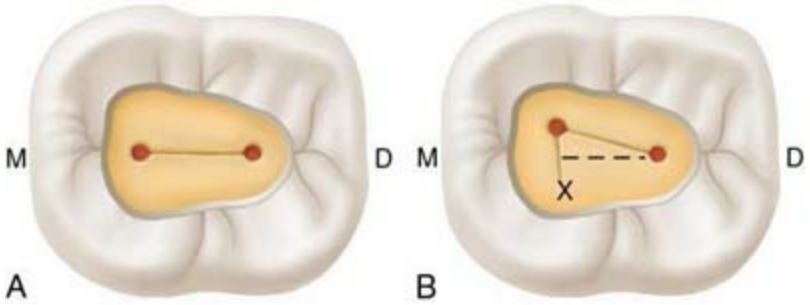

FIG. 7.14 **A,** In a mandibular second molar with two canals, both orifices are in the mesiodistal midline. **B,** If two orifices are not directly in the mesiodistal midline, a search should be made for another canal on the opposite side in the area of "X." *D,* Distal; *M,* mesial.

A) Model of molar shows two holes aligned horizontally in the pulp. The tooth

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is marked M on the left and D on the right.

B) Molar shows two holes aligned diagonally in the pulp. A vertical line from the first hole is labeled X. The tooth is marked M on the left and D on the right.

The direction a file takes when introduced into an orifice is also important. If the first file inserted into the distal canal of a mandibular molar points either in a buccal or lingual direction, a second canal is often present. If two canals are present, they will be smaller than a single canal. (See μCTs and cross sections of all tooth groups later in this chapter. In addition, rotational videos of these groups can be found online as Videos 7.3–7.19 at the Expert Consult site.)

Midroot considerations

As the canal leaves the coronal portion of the root and blends into the midroot portion, many changes can occur, including fins, webs, culs-de-sac, and isthmuses (also called *anastomoses*). These structures are narrow, ribbon-shaped communications between two root canals that contain pulp tissue, or they may midroot portion of the canal. Isthmuses are found in 15% of anterior teeth; in maxillary premolar teeth, they are found in 16% at the 1-mm level from the apex and in 52% at the 6-mm level, which puts them primarily in the middle third of the canal (Fig. 7.15). The prevalence of an isthmus increases in the mesiobuccal root of the maxillary first molar, from 30% to 50% near the junction of middle and apical thirds of the root. Eighty percent of the mesial roots of mandibular first molars have these communications at the apical to middle third junction, with the distal root display more in the apical third.

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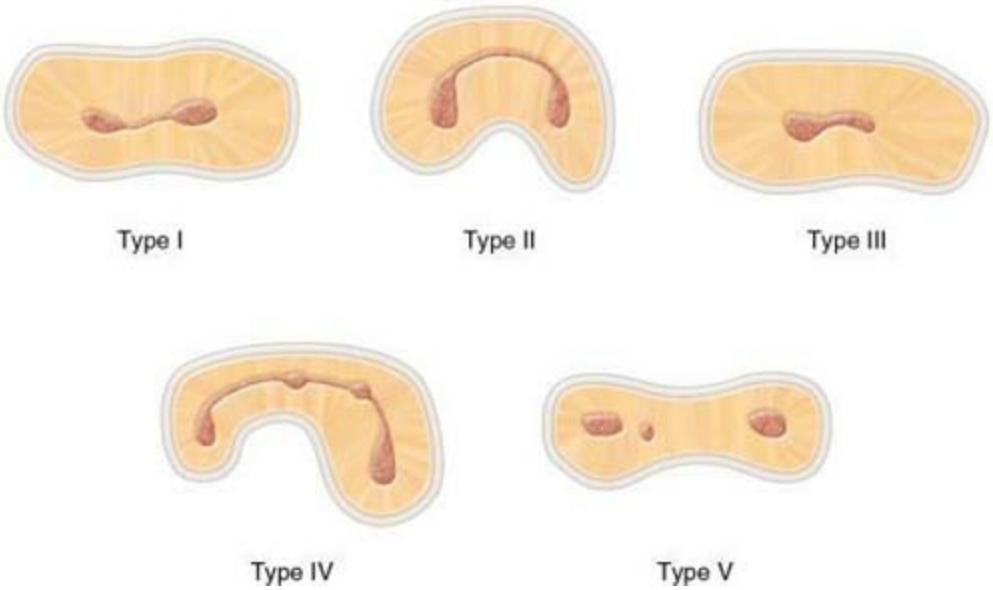

FIG. 7.15 Schematic representation of isthmus classifications described by Kim and colleagues. Type I is an incomplete isthmus; it is a faint communication between two canals. Type II is characterized by two canals with a definite connection between them (complete isthmus). Type III is a very short, complete isthmus between two canals. Type IV is a complete or incomplete isthmus between three or more canals. Type V is marked by two or three canal openings without visible connections.

Five structures, marked type 1 to 5, show a horizontal connection between two canals, inverted U-shape connection between two canals, a larger canal connected closely to a smaller canal, four connected canals, and a rectangular canal with a tiny lobe and roughly oval canal, respectively.

Source: (From Kim S, Pecora G, Rubinstein R, Dorscher-Kim J: *Color atlas of microsurgery in endodontics*, Philadelphia, 2001, Saunders.)

Another change that often occurs in the midroot portion is the splitting of a single canal into two or more canals, along with a wide variation in canal morphology. Likewise, canals often join in this area, starting coronally as two separate canals (see Fig. 7.12).

Whenever a root contains two canals that join to form one, the lingual/palatal canal generally is the one with direct access to the apex. When one canal separates into two, the division is buccal and palatal/lingual, and the lingual canal generally splits from the main canal at a sharp angle, sometimes nearly a right angle (Fig. 7.16). Although the splitting of one canal into two presents challenges, of greater concern is the very small canal that

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may split off, at times at almost a 90 degree angle, and cannot be located tactilely or even under magnification with the DOM. Although these canals may join the main canal at some point apically, some of them exit separately. This is not uncommon in mandibular first premolars.

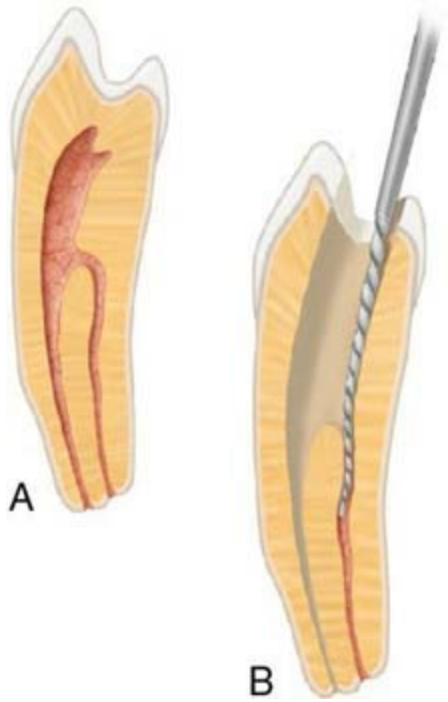

FIG. 7.16 **A,** Mesial view of a mandibular premolar with a type V canal configuration. The lingual canal separates from the main canal at nearly a right angle. **B,** This anatomy requires widening of access in a lingual direction to achieve straight-line access to the lingual canal. This should be done with a DOM.

A) Tooth shows a canal extending from pulp chamber to root apex with an additional branch.

B) Tooth shows an instrument being inserted into the additional root canal.

Apical considerations

The classic concept of apical root anatomy is based on three anatomic and histologic landmarks in the apical region of a root: the apical constriction (AC), the cementodentinal junction (CDJ), and the AF. Kuttler's description of the anatomy of the root apex has the root canal tapering from the canal orifice to the AC, which generally is 0.5 to 1.5 mm coronal to the AF (Fig.

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7.17).[51] The AC generally is considered the part of the root canal with the smallest diameter; it also is the reference point clinicians most often use as the apical termination for enlarging, shaping, cleaning, disinfecting, and filling. Violation of this area with instruments or filling materials is not recommended for long-term, successful outcomes.[38]

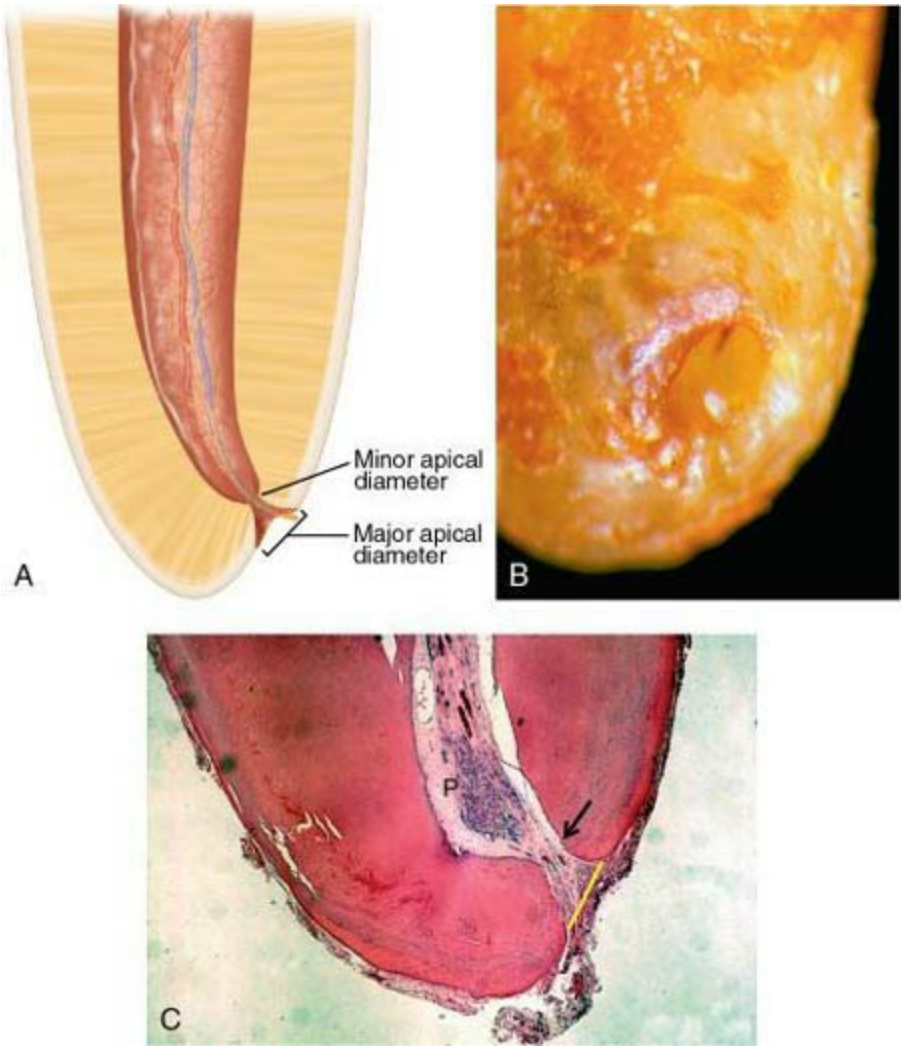

FIG. 7.17 **A**, Morphology of the root apex. From its orifice the canal tapers to the apical constriction, or minor apical diameter, which generally is considered the narrowest part of the canal. From this point the canal widens as it exits the root at the apical foramen, or major apical diameter. The space between the minor and major apical diameters is funnel shaped. **B,** Clinical view of major apical foramen. **C,** Histologic view of the apical constriction *(arrow)* and apical foramen *(yellow line)*. *P,* Pulp. (**B** and **C,** From Gutmann JL, Lovdahl PE: *Problem solving in endodontics*, ed 5, St. Louis, 2011, Elsevier.)

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A) Root of tooth shows two labels at the apex as follows: Minor apical diameter and major apical diameter.

B) Tip of the tooth root shows a large well-defined oval opening.

C) Histological view shows the shrinkage of canal slightly below the root tip.

The CDJ is the point in the canal where cementum meets dentin; it is also the point where pulp tissue ends and periodontal tissues begin (Fig. 7.18). The location of the CDJ in the root canal varies considerably. It generally is not in the same area as the AC, and estimates place it approximately 1 mm from the AF.[93,101]

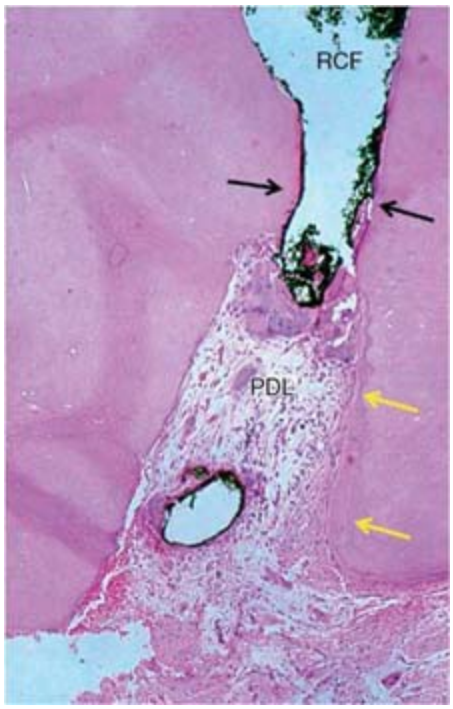

FIG. 7.18 Root apex following root canal filling *(RCF)* short of the actual root length. Histologic evidence shows that hard tissue *(black arrows)* has formed from the cells of the periodontal ligament *(PDL)* adjacent to the root filling material. Cementum formation on the internal aspect of the apical foramen is indicated by the yellow arrows. These findings accentuate the variable nature of the apical tissues.

Stained micrograph shows filled root canal of tooth, marked RDF, surrounded by a dark black boundary and two holes at the base. This is followed by slightly dense tissue, marked PDL, with an oval patch.

Source: (From Gutmann JL, Lovdahl PE: *Problem solving in*

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endodontics, ed 5, St. Louis, 2011, Elsevier.)

The AF is the "circumference or rounded edge, like a funnel or crater that differentiates the termination of the cemental canal from the exterior surface of the root."[51] The AF does not normally exit at the anatomic apex, but rather is offset 0.5 to 3 mm. This variation is more marked in older teeth through cementum apposition. Studies have shown that the AF coincides with the apical root vertex in 17% to 46% of cases.[14,35,36,87,113,115]

The location and diameter of the CDJ differ from those of the AF in maxillary anterior teeth.[88] Extension of cementum from the AF into the root canal differs considerably, even when opposite canal walls are compared (see Figs. 7.17 and 7.18). Cementum reaches the same level on all canal walls in only 5% of cases. The greatest extension generally occurs on the concave side of the canal curvature. This variability confirms that the CDJ and the AC generally are not in the same area and that the CDJ should be considered just a variable junction at which two histologic tissues meet in the root canal (see Fig. 7.18).

Scanning electron microscopy has been used to determine the number and size of main apical foramina, their distance from the anatomic apex, and the size of accessory foramina. The morphology of the apical third of the root reflects multiple anatomic variations, including numerous accessory canals; areas of resorption and repaired resorption; attached, embedded, and free pulp stones; varying amounts of reparative dentin; and varying root canal diameters (see Tables 7.5–7.7 in the Expert Consult site).[71,72] Primary dentinal tubules are found less often than in the coronal dentin and are more or less irregular in direction and density, or even absent. This variable nature of the apical structure and significant absence of dentinal tubules may lead to reduced chances of bacterial invasion into the dentinal walls; however, it also presents challenges for all root canal procedures, from cleaning and disinfection to obturation.

Clinically, considerable controversy exists over the exact termination point for root canal procedures in the apical third of the root; clinical determination of apical canal morphology is difficult at best.[39,98] Detailed perspectives, both historical and contemporary, can be found in the literature.[38] Some authors recommend that root canal procedures terminate at or within 3 mm of

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the radiographic apex, depending on the pulpal diagnosis. For vital cases, clinical and biologic evidence indicates that a favorable point at which to terminate therapy is 2 to 3 mm short of the radiographic apex.[46,99] This leaves an apical pulp stump, which prevents extrusion of irritating filling materials into the periradicular tissues in vital cases. However, what is not commonly recognized among clinicians is that this so-called pulp stump is actually not pulp tissue, but rather periodontal tissue that can ensure apical healing with cementum over the root end (see Fig. 7.18). Studies have shown that a better success rate is achieved when the procedure ends at or within 2 mm of the radiographic apex.[45,46,99] When short of the 2-mm point or extended past the radiographic apex, the success rate declined by 20%. For revision of procedural failures, apical procedures should extend to or preferably 1 to 2 mm short of the radiographic apex to prevent overextension of instruments and filling materials into the periradicular tissues.

Many investigators who have evaluated apical and periradicular tissues after root canal procedures concluded that the most favorable prognosis was obtained when procedures were terminated at the AC, and the worst prognosis was produced by treatment that extended beyond the AC.[54–57,90] Some studies support the termination of all procedures at or beyond the radiographic apex, thereby filling all apical ramifications and lateral canals.[95] This dictate is empirically based, and CBCT evaluation of procedures previously thought to be successful has allowed identification of more posttreatment disease.[18,83]

Objectives and guidelines for traditional access cavity preparation

Objectives

Access to the complex root canal system is the first important phase of any nonsurgical root canal procedure.[39,109] The objectives of access cavity preparation are to (1) remove all caries when present, (2) conserve sound tooth structure, (3) unroof the pulp chamber completely, (4) remove all coronal pulp tissue (vital or necrotic), (5) locate all root canal orifices, and (6) achieve straight- or direct-line access to the AF or to the initial curvature of

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the canal.

A properly prepared access cavity creates a smooth, straight-line path to the canal system and ultimately to the apex or position of the first curvature (Fig. 7.19, *A*). Straight-line access provides the best chance of débridement of the entire canal space; it reduces the risk of instrument breakage[73]; and it results in straight entry into the canal orifice, with the line angles forming a funnel that drops smoothly into the canal (or canals). Projection of the canal center line to the occlusal surface of the tooth indicates the location of the line angles (see Fig. 7.19, *B*).

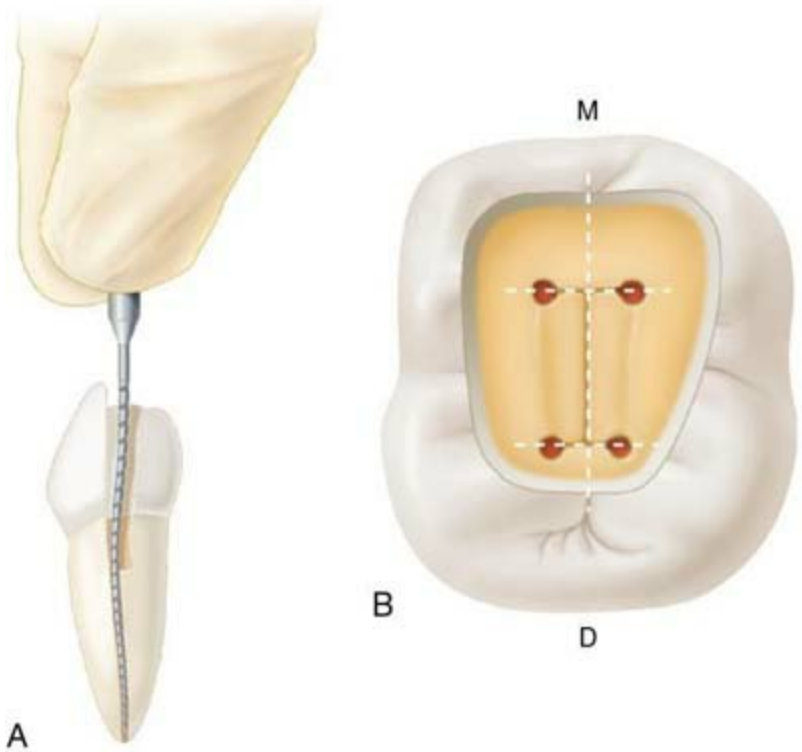

FIG. 7.19 **A,** Straight-line access to a canal. The instrument should not be deflected until it reaches the initial canal curvature. In some cases coronal tooth structure must be sacrificed to obtain direct access to the pulp chamber. **B,** Diagrammatic representation of centrality and concentricity of symmetry and location of canal orifices. *D,* Distal; *M,* mesial.

A) A gloved hand inserts the instrument to the root canal in a tooth.

B) Tooth is marked M at the top and D at the bottom. The central chamber has four holes aligned to form a rectangular shape.

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Key steps to consider in traditional access preparation

Evaluation of the cementoenamel junction and occlusal tooth anatomy

Traditionally, some access cavities have been prepared in relation to the occlusal or lingual anatomy. However, complete reliance on the anatomy is dangerous because this morphology can change as the crown is destroyed by caries and reconstructed with various restorative materials. Likewise, the root may not be perpendicular to the occlusal surface of the tooth; thus, complete dependence on the occlusal or lingual anatomy may explain the occurrence of some procedural errors, such as coronal perforations along the cervical line or into the furcation. Krasner and Rankow found that the cementoenamel junction (CEJ) was the most important anatomic landmark for determining the location of pulp chambers and root canal orifices. Their study demonstrated the existence of a specific and consistent anatomy of the pulp chamber floor.[49]

Preparation of the access cavity through the lingual and occlusal surfaces

Access cavities on anterior teeth usually are prepared through the lingual tooth surface, and those on posterior teeth are prepared through the occlusal surface. These approaches are the best for achieving straight-line access while reducing esthetic and restorative concerns. Some authors have recommended that the traditional anterior access for mandibular incisors be moved from the lingual surface to the incisal surface in selected cases[69]; this may allow for better access to the lingual canal and improve canal débridement (Fig. 7.20). In teeth that are lingually inclined or rotated, this is often the preferred choice for access and is performed before the dental dam is placed, or careful alignment of the root is determined before a bur is used.[39]

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FIG. 7.20 An incisal access cavity on mandibular anterior teeth may allow for improved straight-line access and canal débridement.

Tooth shows a wedge-shape opening extending from the crown to root apex.

Removal of all defective restorations and caries before entry into the pulp chamber

Removal of all defective restorations and caries before entering the root canal system is essential for many reasons.[39] Hidden caries or fractures are often identified, and the ability to determine the restorability of the tooth is enhanced (Fig. 7.21). In this respect, it is not uncommon, before entering the pulp chamber, to stop with the access opening preparation in favor of a crown lengthening procedure or adding a bonded buildup.[39]

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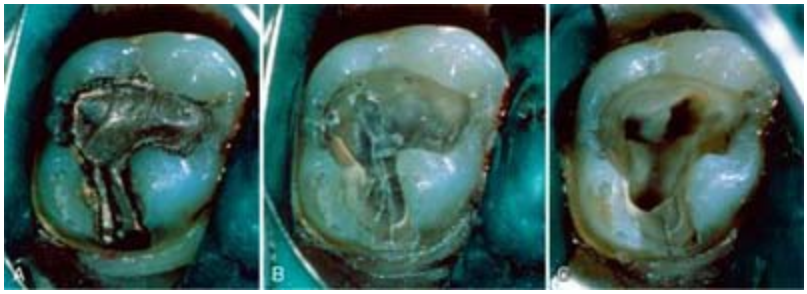

FIG. 7.21 **A,** Maxillary molar requiring root canal procedure. **B,** Removal of the amalgam reveals a vertical fracture on the palatal margin. **C,** Complete cleaning and shaping of the canals. Fracture lines are still visible, but no probings are present.

A) Close-up view of molar shows damaged root canals and pulp with multiple fractures without surrounding tissue.

B) Same tooth shows dense tissue in place of the empty spaces.

C) Tooth shows three holes at the center.

Source: (From Gutmann JL, Lovdahl PE: *Problem solving in endodontics*, ed 5, St. Louis, 2011, Elsevier.)

Preparation of access cavity walls that do not restrict straight- or direct-line passage of instruments to the apical foramen or initial canal curvature

Sufficient tooth structure must be removed to allow instruments to be placed easily into each canal orifice without interference from canal walls, particularly when a canal curves severely or leaves the chamber floor at an obtuse angle. Hence, access design depends not only on the orifice location, but also on the position and curvature of the entire canal.[106]

Objectives and guidelines for minimally invasive access cavity preparation

The objectives are to preserve as much tooth structure as possible, while managing to locate, debride, enlarge, shape, and obturate the root canal(s). Much has been written and some studies have been done to verify this approach to access preparation; however, definitive, evidence-based outcomes have yet to be seen within this empirically based approach. Access

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openings must be crafted to preserve sound tooth structure and prevent tooth fracture (referred to as the guided endodontic technique,[33,58] or microguided technique,[23] contracted cavities,[6,66,74,97] truss access openings,[8] and orifice-directed dentin conservation access[77]). The prevention of gouging, cervically, laterally or into the floor of the pulp chamber is especially important (Figs. 7.22 and 7.23). However, access that is too restricted may or may not greatly impact the remaining and important root canal procedures (Fig. 7.24).[50,77,94] While judicious orifice location and careful canal penetration are essential, efforts should be made to minimize the excess removal of cervical tooth structure.[20–22,47,97] The literature indicates that loss of tooth structure in certain teeth may weaken the tooth cervically,[124] making them susceptible to fracture,[5,41,57,60,61] although this concept has been challenged.[91,97]

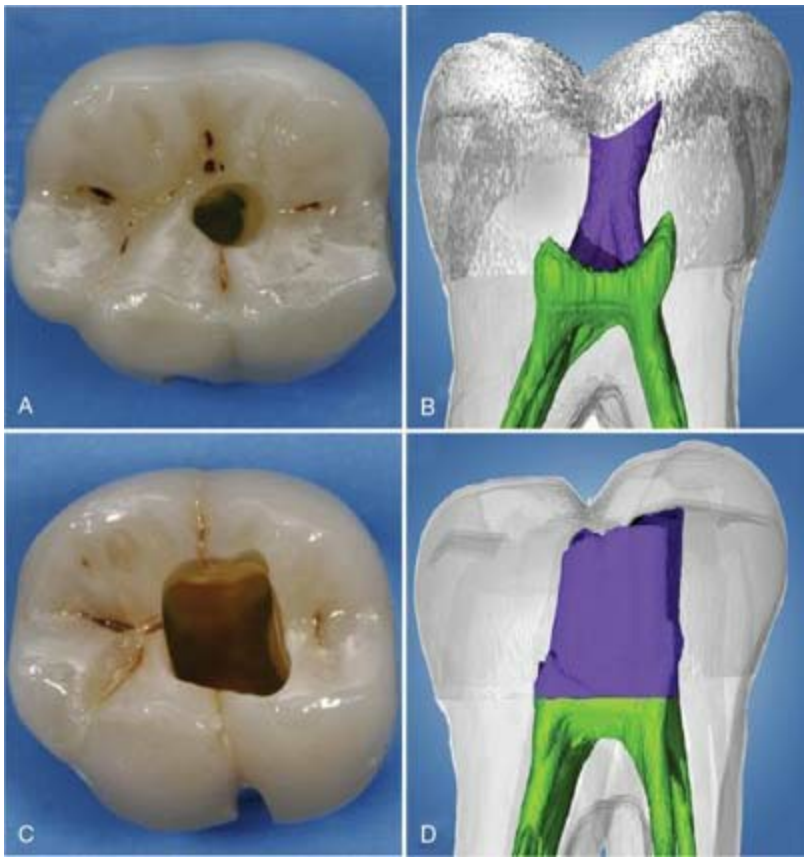

FIG. 7.22 **A,** A photograph of a mandibular molar showing minimally invasive access cavity outline. **B,** A sagittal view of a μCT reconstruction showing minimally invasive access cavity pathway. **C,** A photograph of a mandibular molar showing traditional access cavity

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outline. **D,** A sagittal view of a μCT reconstruction showing traditional access cavity pathway.

A) Tooth shows a circular cavity at the center with some dark spots in the surrounding.

B) Tooth shows a roughly concave-shaped cavity (purple) in the crown.

C) Tooth shows a rectangular cavity at the center with scars in the surrounding.

D) Tooth shows the rectangular cavity (purple) in the crown.

Source: (From Marchesan MA, Lloyd A, Clement DJ, et al: Impacts of contracted endodontic cavities on primary root canal curvature parameters in mandibular molars, *J Endod* 44:1558–1562, 2018.)

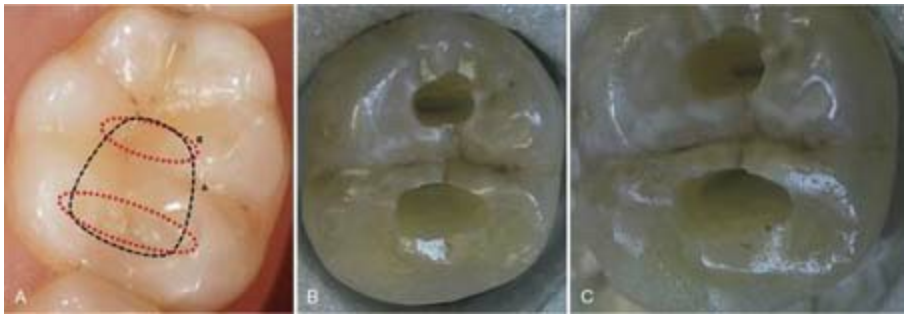

FIG. 7.23 **A,** A schematic diagram of the traditional access cavity outline *(A, black dotted line)* and minimally invasive access cavity outline *(B, red dotted line)* in a mandibular molar. **B,** A photograph of a mandibular molar showing mesial access and orifice. **C,** A photograph of a mandibular molar showing distal access and orifice.

A) Close-up view of molar tooth shows two convex-shaped cavities, marked B, within a large cavity, marked A.

B) Close-up view of tooth shows a small hole at the top and a slightly large hole at the bottom with mesial access on one side.

C) Close-up view of tooth shows similar holes as that of B with access at the distal position.

Source: (From Neelakantan P, Khan K, Pak Hei G, et al: Does the orifice-directed dentin conservation access design debride pulp chamber and mesial root canal systems of mandibular molars similar to a traditional access design? *J Endod* 44:274–279, 2018.)

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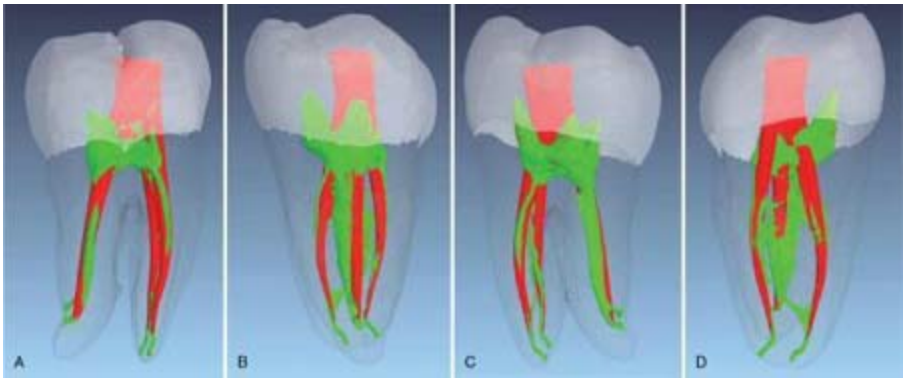

FIG. 7.24 Merged μCT images of a mandibular first molar demonstrating the root canals pretreatment *(green)* and posttreatment *(red)*. **A,** Buccal view. **B,** Distal view. **C,** Lingual view. **D,** Mesial view.

Series of four μ CT images, marked A through D, show different views of molar after the pre and post-treatment.

Source: (From Krishan R, Paqué F, Ossareh A, et al: Impacts of conservative endodontic cavity on root canal instrumentation efficacy and resistance to fracture assessed in incisors, premolars, and molars, *J Endod* 40:1160–1166, 2014.)

Mechanical phases of access cavity preparation

Magnification and illumination

For some clinicians, the access cavity is best prepared with the use of magnification and an appropriate light source.[15] In lieu of a DOM, surgical loupes with an auxiliary light source are highly recommended.

Burs

Numerous burs have been developed exclusively for access cavity preparation (Fig. 7.25). Most clinicians have their own set of preferred access burs. A significant trend in restorative dentistry is the increased use of zirconia-based crowns and onlays. Zirconia has different mechanical and thermal characteristics than metal. Carbide burs do not efficiently or safely cut zirconia. Zirconia is a brittle material and when cut can develop cracks that propagate through the framework and lead to eventual failure of the

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crown or onlay. Diamond bur manufacturers are aware of these issues, and they are currently introducing medium- and fine-grit diamond burs that efficiently cut zirconia (Komet USA, Savannah, GA; Prima Dental Gloucester, England; SS White). These diamond burs should be used with copious sprayed water to minimize heat buildup in the zirconia crowns during access preparations.[39]

FIG. 7.25 Access burs. **A,** #2, #4, and #6 round carbide burs. **B,** Safety-tip tapered diamond bur *(left)*; safety-tip tapered carbide bur *(right)*. **C,** Transmetal bur. **D,** Round-end cutting tapered diamond bur. **E,** #57 fissure carbide bur. **F,** #2 and #4 round diamond burs. **G,** Mueller bur *(left)*; LN bur or Extendo Bur *(right)*.

Seven images, marked A through G, show different dental tools used during surgeries.

Many teeth requiring access cavity preparations have metal restorations that must be penetrated. These restorations may be amalgams, all-metal cast restorations, or metal copings of porcelain fused to metal crowns. A transmetal bur (Dentsply Sirona, Ballaigues, Switzerland) is excellent for cutting through metal because of its exceptional cutting efficiency.

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Endodontic explorer and spoon

The DG-16 endodontic explorer is used to identify canal orifices and to determine canal angulation. An alternative, the JW-17 endodontic explorer (CK Dental Industries, Orange, CA) serves the same purpose, but its thinner, stiffer tip can be useful for identifying the possible location of a calcified canal. A sharp endodontic spoon, which comes in different sizes can be used to remove coronal pulp and carious dentin.

Ultrasonic unit and tips

Ultrasonic units and tips specifically designed for activities during access opening preparation are extremely valuable. Ultrasonic tips from various manufacturers can be used to trough and deepen developmental grooves, remove tissue, and explore for canals. These systems provide outstanding visibility compared with traditional handpiece heads, which typically obstruct vision.

Access cavity preparations

Anterior teeth

The following discussion outlines the steps for maxillary and mandibular anterior teeth. Tooth-specific access concerns are illustrated and discussed in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in the chapter.

External outline form

For an intact tooth, cutting commences at the center of the lingual or palatal surface of the anatomic crown. An outline form is developed that is similar in geometry to an ideal access shape for the particular anterior tooth. The bur is directed perpendicular to the lingual surface as the external outline opening is created.

Penetration of the pulp chamber roof (Fig. 7.26)

Experienced clinicians usually penetrate the pulp chamber roof using a high-speed handpiece; however, less experienced clinicians may find the increased

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tactile sensation of a slow-speed handpiece a safer option. Continuing with the same round or tapered fissure bur, the angle of the bur is rotated from perpendicular to the lingual/palatal surface to parallel to the long axis of the root (see Fig. 7.26, *C* and *D*). Penetration into the tooth continues along the root's long axis until the roof of the pulp chamber is penetrated; frequently, a drop into the chamber effect is felt when this occurs. Measuring the distance from the incisal edge to the roof of the pulp chamber on a dimensionally accurate pretreatment radiograph may serve as guide in limiting penetration and possibly preventing a perforation. If the drop-in effect is not felt at this depth, a DG-16 explorer can be used to probe the depth of the access. Often a small opening into the chamber is present, or the dentin is very thin and the explorer penetrates into the chamber. The depth and angle of penetration should be assessed for any deviation away from the long axis of the root in both the mesiodistal and buccolingual dimensions, and the penetration angle should be realigned if necessary. Angled radiographs can be used to assess progress at any time if any confusion or doubt exists.

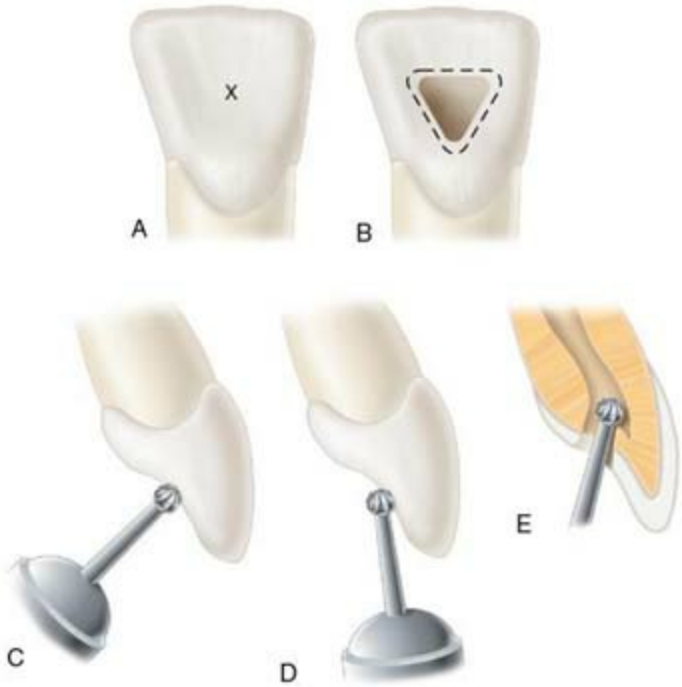

FIG. 7.26 **A,** In anterior teeth, the starting location for the access cavity is the center of the anatomic crown on the lingual surface *(X).* **B,** Preliminary outline form for anterior teeth. The shape should mimic the expected final outline form, and the size should be half to three fourths

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the size of the final outline form. **C,** The angle of penetration for the preliminary outline form is perpendicular to the lingual surface. **D,** The angle of penetration for initial entry into the pulp chamber is nearly parallel to the long axis of the root. **E,** Completion of removal of the pulp chamber roof; a round carbide bur is used to engage the pulp horn, cutting on a lingual withdrawal stroke.

A) Crown and neck region of the front tooth are shown with a label X at the center of crown.

B) Front tooth shows a triangular cavity at the center.

C) Round carbide bur is placed at 90 degrees to the center of crown.

D) Round carbide bur is placed straight and touches the center of crown.

E) Round carbide bur is penetrated into the canal from crown.

Removal of the chamber roof

Once the pulp chamber has been penetrated, the remaining roof is removed by catching the end of a round bur under the lip of the dentin roof and cutting on the bur's withdrawal stroke (see Fig. 7.26, *E*). Working in this manner enables the internal pulp anatomy to dictate the external outline form of the access opening. Copious irrigation with sodium hypochlorite helps control any hemorrhage present. Subsequently, the chamber roof, including the pulp horns, is removed and all internal walls are flared to the lingual surface of the tooth and complete roof removal is confirmed with an explorer.

Removal of the lingual shoulder and coronal flaring of the orifice

Once the orifice or orifices have been identified and confirmed, the lingual shoulder or ledge is removed. This is a shelf of dentin that extends from the cingulum to a point approximately 2 mm apical to the orifice (Fig. 7.27). Its removal improves straight-line access and allows for more intimate contact of files with the canal's walls for effective shaping and cleaning. Furthermore, its removal from mandibular anterior teeth may often expose an extra orifice and canal.

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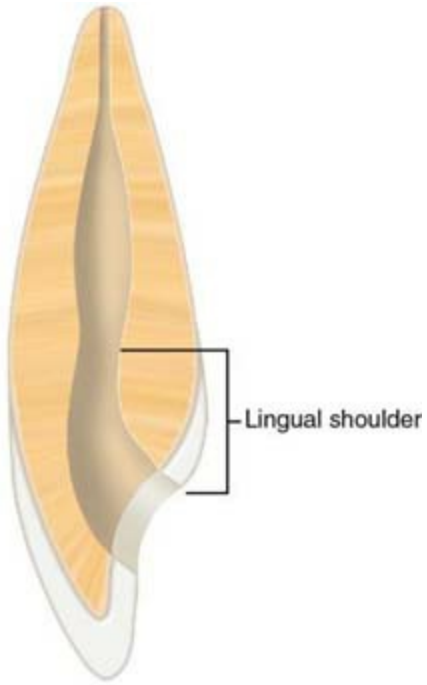

FIG. 7.27 Lingual shoulder of the anterior tooth, extending from the cingulum to 2 mm apical to the orifice.

Front tooth shows an opened canal with a lower right region, marked lingual shoulder.

The contemporary approach to flaring the orifice involves the use of rotary nickel-titanium (NiTi) orifice openers that allow rapid, safe removal of the lingual ledge, following the manufacturer's directions for use (DFUs). Some clinicians prefer the use of Gates-Glidden burs. These openers allow refinement of the orifice shape or help to enhance straight-line access to the canal with minimal removal of dentin when used properly. The bur should be placed so as to avoid putting a bevel on the incisal edge of the access preparation (Fig. 7.28). When Gates-Glidden burs are used, the largest that can be placed passively 2 mm apical to the orifice is used first with gentle pressure. During this process, the orifice is often concomitantly flared so that it is contiguous with all walls of the access preparation. If this is not achieved, use of the orifice openers is recommended.

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FIG. 7.28 Placing an incisal bevel on the lingual surface of a maxillary anterior tooth can lead to fracture of the permanent restoration during occlusal function.

Front tooth shows an opened canal with a slanted edge in the enamel, marked incisal bevel.

Straight-line access determination

Straight-line access to the canal orifice is the ideal goal. Preferably, a small intracanal file should reach the AF or the first point of canal curvature with no deflections. If the lingual shoulder has been removed and a file still binds on the incisal edge, the access cavity should be further extended incisally, until the file is not deflected (Fig. 7.29). The final position of the incisal wall of the access cavity is determined by two factors: (1) complete removal of the pulp horns and (2) straight-line access. However, the concept of straight-line access may not be totally feasible when using minimally invasive access preparations.

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FIG. 7.29 **A,** The lingual ledge of dentin remains, deflecting the file toward the labial wall. As a result, portions of the lingual canal wall will not be shaped and cleaned. **B,** Removal of the lingual ledge results in straight-line access.

A) Tooth shows the slightly curved shape of instrument inside the canal.

B) Tooth shows instrument without any bend inside the canal.

Refinement and smoothing of restorative margins

The final step in the preparation of an access cavity is to refine and smooth the cavosurface margins. Rough or irregular margins can contribute to coronal leakage through a permanent or temporary restoration.

Individual anterior teeth

See the figures in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in this chapter.

Posterior teeth

Preparing access cavities on posterior teeth is similar to the process for anterior teeth, but significant differences warrant a separate discussion.[39]

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Posterior teeth requiring root canal procedures typically have been heavily restored, which may make the process more challenging, or the carious process was extensive, which may make the process much easier.

External outline form

Removal of caries and existing restorations from a posterior tooth often results in the development of an acceptable access outline form, with pulp chamber positioned in the center of the tooth at the level of the CEJ. Therefore, in maxillary premolars, the point of entry that determines the external outline form is on the central groove between the cusp tips (Fig. 7.30). Crowns of mandibular premolars are tilted lingually relative to their roots (Fig. 7.31); therefore, the starting location must be adjusted to compensate for this tilt (Fig. 7.32). In mandibular first premolars, the starting location is halfway up the lingual incline of the buccal cusp on a line connecting the cusp tips. Mandibular second premolars require less of an adjustment because they have less lingual inclination. The starting location for this tooth is one third the way up the lingual incline of the buccal cusp on a line connecting the buccal cusp tip and the lingual groove between the lingual cusps.

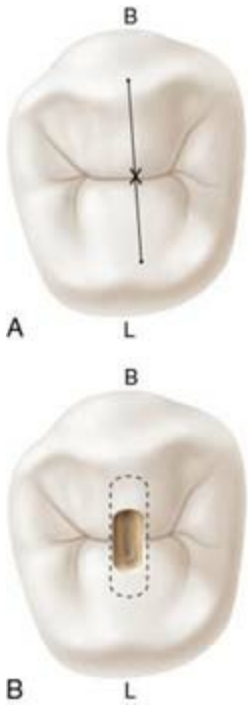

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FIG. 7.30 **A,** Starting location for access to the maxillary premolar *(X)*. **B,** Initial outline form *(dark area)* and projected final outline form *(dashed line). B,* Buccal; *L,* lingual.

A) Premolar is marked B at the top and L at the bottom. A vertical line passing the central horizontal line marked X at the center.

B) Premolar shows an oval chamber at the center.

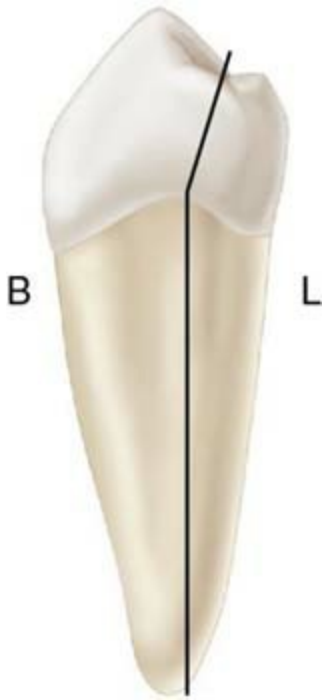

FIG. 7.31 The crown of a mandibular premolar is tilted lingually relative to the root. *B,* Buccal; *L,* lingual.

Tooth shows buccal region on the left and lingual on the right. A slant line passes through the crown and then tilts while passing through the root to its apex.

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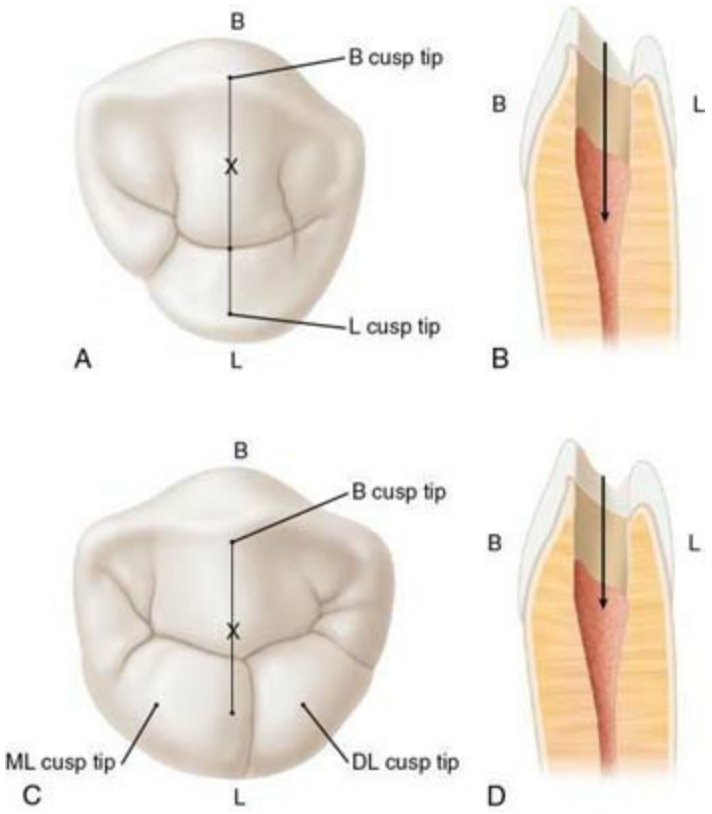

FIG. 7.32 **A,** Mandibular first premolar and access starting location *(X)* (occlusal view). **B,** Mandibular first premolar and starting location (proximal view). **C,** Mandibular second premolar and access starting location *(X)* (occlusal view). **D,** Mandibular second premolar and starting location (proximal view). *B,* Buccal; *DL,* distolingual; *L,* lingual; *ML,* mesiolingual.

A) Tooth shows labels for B cusp and L cusp tip with an X mark below B cusp tip.

B) Structure of tooth shows an arrow pointing below the pulp.

C) Tooth shows labels for B cusp tip, DL cusp tip in the lower left, and ML cusp tip in the lower right side. An X mark is located at the center.

D) Structure of tooth shows an arrow pointing just below the pulp horns.

To determine the starting location for molar access cavity preparations, the mesial-distal and apical-coronal boundary limitations for this outline must be determined (Fig. 7.33). Evaluation of bite-wing radiographs is an accurate method of assessing the extensions of the pulp chamber (Fig. 7.34). The mesial boundary for both the maxillary and mandibular molars is a line connecting the mesial cusp tips. Pulp chambers are rarely found mesial to this

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imaginary line. A good initial distal boundary for maxillary molars is the oblique ridge. For mandibular molars, the initial distal boundary is a line connecting the buccal and lingual grooves. For molars, the correct starting location is on the central groove halfway between the mesial and distal boundaries.

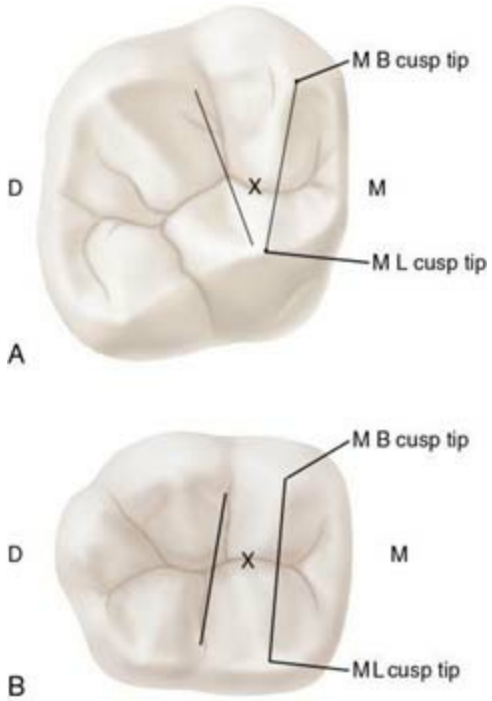

FIG. 7.33 **A,** Mesial and distal boundary of a maxillary molar with the access starting location *(X)*. **B,** Mesial and distal boundary of a mandibular molar showing the access starting location *(X)*. *D,* Distal; *M,* mesial; *MB,* mesiobuccal; *ML,* mesiolingual.

A) Maxillary molar shows a right slant line near an X mark on the mesial boundary. B cusp tip and L cusp tip are marked above and below the mesial line.

B) Mandibular molar shows a left slant line near an X mark on the mesial boundary. B cusp tip and L cusp tip are marked above and below the mesial line.

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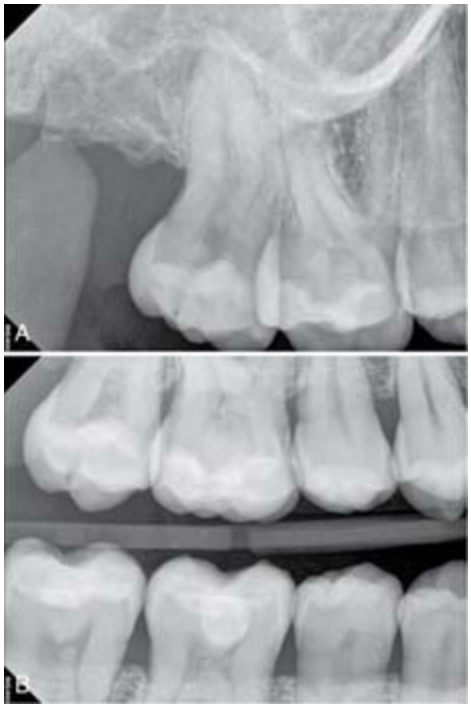

FIG. 7.34 **A,** Periapical radiograph of the teeth in the maxillary right quadrant. **B,** Bitewing radiograph of the same teeth provides a clearer delineation of the pulpal morphology.

A) Radiograph shows upper right quadrant with radiolucent patches on the teeth and porous tissue in the root of central molar.

B) Radiograph shows some teeth with radiolucent pulp chambers and some with radiolucent root canals.

Penetration through the enamel into the dentin (approximately 1 mm) is achieved with a #2 or #4 round bur for premolars and a #4 or #6 round bur for molars. A tapered fissure bur may be used instead of round burs. The bur is directed perpendicular to the occlusal table, and an initial outline shape is created at about half to three fourths its projected final size. The premolar shape is oval and widest in the buccolingual dimension. The molar shape is also oval initially; it is widest in a buccolingual dimension for maxillary molars and in a mesiodistal direction for mandibular molars. The final outline shape for molars is approximately triangular (for three canals) or rhomboid (for four canals), even when a minimal access approach is used; however, the canal orifices dictate the position of the corners of these geometric shapes. Therefore, until the orifices have been located, the initial outline form should be left as roughly oval.

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Penetration of the pulp chamber roof

As with anterior teeth, penetration through the chamber roof is limited to the distance measured on a pretreatment radiograph. If the drop-in effect is not felt at this depth, a careful evaluation of the angle of penetration is necessary before going deeper into the chamber. In multirooted posterior teeth, lateral and furcation perforations may occur rapidly without attention to 3D detail during this penetration. Aggressive probing with a DG-16 explorer at any time during the penetration is encouraged. Once the pulp chamber has been penetrated, the remaining roof is removed by catching the end of a round bur under the lip of the dentin roof and cutting with the burs on a withdrawal stroke.

Identification of all canal orifices

The canal orifices play an important role in determining the final extensions of the external access outline form. Ideally, the orifices are located at the corners of the final preparation to facilitate all of the root canal procedures (Fig. 7.35).

FIG. 7.35 An endodontic explorer is used to search for canal orifices.

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Tooth shows four holes in the center. An endodontic explorer is passing from one of the holes on the left.

Removal of the cervical dentin bulges and orifice and coronal flaring

Internal impediments to an ideal access opening are the cervical dentin ledges or bulges and the natural coronal canal constriction in posterior teeth.[39] The cervical bulges are shelves of dentin that frequently overhang orifices in posterior teeth, restricting access into root canals and accentuating existing canal curvatures (Fig. 7.36).[60]

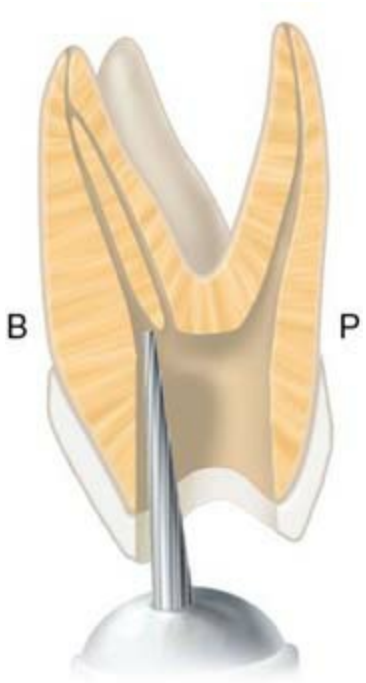

FIG. 7.36 Safety-tip carbide bur is used to shape the axial wall in one plane from the orifice to the cavosurface margin. *B,* Buccal; *P,* palatal.

Tooth shows carbide bur placed straight inside the canal toward buccal region.

Straight-line access determination

Straight-line access is paramount to successful shaping, especially given the complexity of the root canal systems in posterior teeth. However, this has not

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been verified when using a minimal access cavity. Files must have unimpeded access to the AF or the first point of canal curvature to perform properly.

Visual inspection of the pulp chamber floor

Floor and walls must be inspected, using appropriate magnification and illumination, to ensure that all canal orifices are visible and no roof overhangs are present (Fig. 7.37).

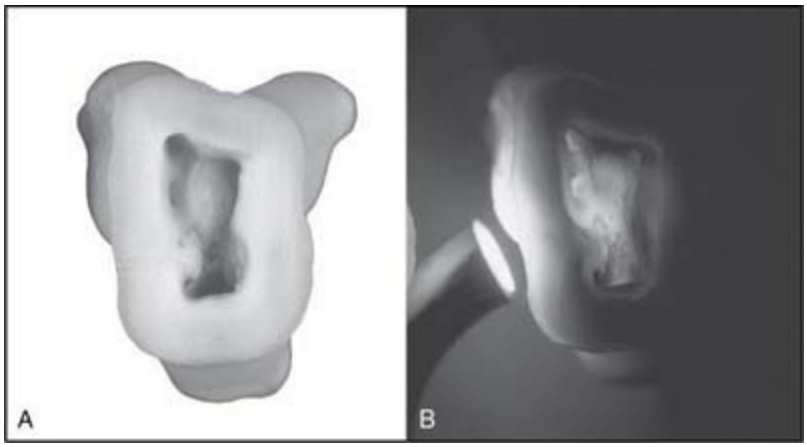

FIG. 7.37 A fiberoptic light can be applied to the cervical aspect of the crown to help obtain maximal visibility with magnification. Transillumination often reveals landmarks otherwise invisible to the unaided eye.

A) Cut specimen of tooth shows a dark chamber with orifices at the center.

B) The same specimen is observed under light that shows calcified pulp chamber with dense protuberances and dark tissues near the periphery in the center.

Individual posterior teeth.

See the figures in the section Morphology of and Access Cavity Preparations for Individual Teeth, later in the chapter.

Challenging access preparations

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Access in teeth with presumed calcified canals

Chambers and roots that demonstrate the potential for significant calcifications may present problems with locating, penetrating, and negotiating the pulpal spaces.[39] The use of magnification and transillumination, as well as careful examination of color changes and pulp chamber shapes, assists in locating the canals (Fig. 7.38). However, the search for the root canal orifices should be made only after the pulp chamber has been completely prepared and its floor has been cleaned and dried (95% denatured ethanol may be useful for drying the floor and enhancing visibility). A fiberoptic light directed from the external through the CEJ can reveal subtle landmarks and color changes that may not otherwise be visible. The chamber floor is darker in color than its walls, and developmental grooves connecting orifices are lighter in color than the chamber floor. Awareness of these color differences when searching for small orifices is essential, especially when searching for those that are located at the angles formed by the floor and walls and at the end points of developmental grooves. Additional methods to help locate calcified root canals include staining the pulp chamber floor with 1% methylene blue dye, performing the sodium hypochlorite “champagne bubble” test (Fig. 7.39), and searching for canal bleeding points. These approaches are enhanced when the area is viewed through magnification, which would be essential with a minimal access cavity.

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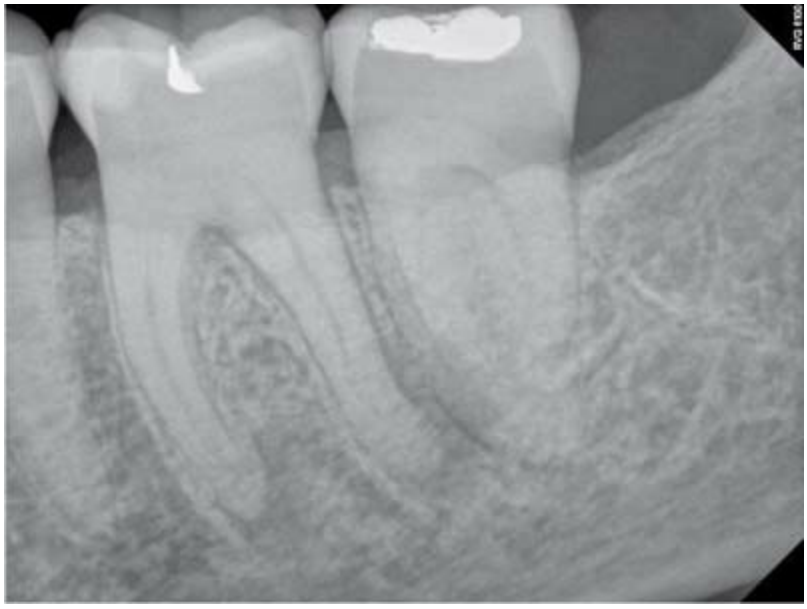

FIG. 7.38 Mandibular molar with what appears to be almost complete calcification of the pulp chamber and root canals. However, pathosis is present, which indicates the presence of bacteria and some necrotic tissue in the apical portion of the roots.

Radiograph shows two molars with radiopaque patches on the enamel. The root apices show porous tissue.

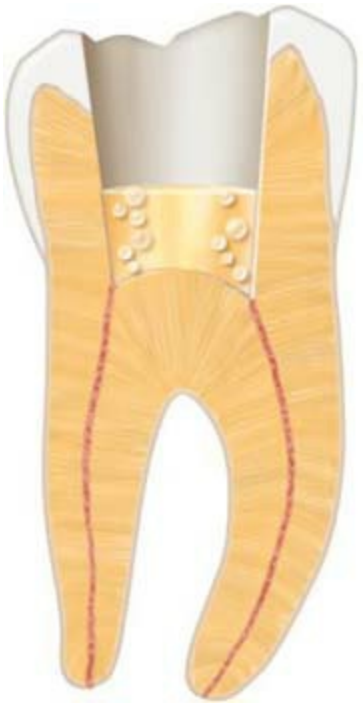

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FIG. 7.39 Allowing sodium hypochlorite (NaOCl) to remain in the pulp chamber may help locate a calcified root canal orifice. Tiny bubbles may appear in the solution, indicating the position of the orifice. This is best observed through magnification.

Two-rooted tooth shows sodium hypochlorite crystals in the pulp chamber below a U-shaped chamber.

Uncovering canals that contain calcified material is a challenge. When the canal is located, a small K-file (#6, #8, or #10 or, preferably, a C or C+ file [Dentsply Sirona—Ballaigues, Switzerland]) coated with a chelating agent should be introduced into the canal to determine patency. These instruments provide added stiffness to the shaft for better penetration. The file should not be removed until some canal enlargement has occurred. The file should be used in short up-and-down movements and in a selective circumferential filing motion, with most of the lateral pressure directed away from the furcation. This enlarges the coronal aspect of the canal safely and moves it laterally, to avoid thinning of the dentin wall adjacent to the furcation. It also creates a path of insertion for larger files and for coronal access burs. Figs. 7.40–7.45 illustrate several methods that can be used to locate calcified spaces. Furthermore, a guided access technique is advocated to perform the coronal entry into teeth with partially or completely calcified pulp chambers and/or obstructed root canals.[59]

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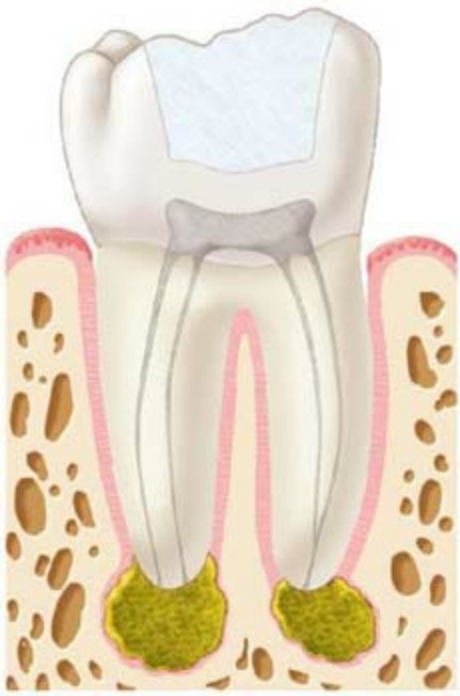

FIG. 7.40 Mandibular first molar with a class I restoration, calcified canals, and periradicular radiolucencies. Presumably a pulp exposure has occurred, resulting in calcification and ultimate necrosis of the pulp tissue.

Tooth shows green colored patches at the root tips.

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FIG. 7.41 Excavation of a restoration and base material. The cavity preparation is extended toward the assumed location of the pulp chamber, keeping in mind that pulp chambers are located in the center of the tooth at the level of the cementoenamel junction (CEJ).

Tooth shows green colored patches at the root tips. The crown has U-shaped chamber.

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FIG. 7.42 Use a long-shank #2 or #4 round bur to remove dentin and attempt to locate calcified canals.

Tooth shows green patches at the root tips and carbide bur just above the root canal on the left.

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FIG. 7.43 An endodontic explorer is used to probe the pulp floor. A straight ultrasonic tip may be used to remove dentin. Angled radiographs must be taken to monitor progress.

Tooth shows green patches at the root tips and an endodontic explorer probing the root canal on the right.

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FIG. 7.44 At the first indication of a canal space, the smallest instrument (i.e., a #.06 or #.08 C or C+ file or micro openers) should be introduced into the canal. Gentle passive movement, both apical and rotational, often produces some penetration. A slight pull, signaling resistance, usually is an indication that the canal has been located. This should be confirmed by radiographs.

Tooth shows green patches at the root tips and a small K-file entering the root canal on the right.

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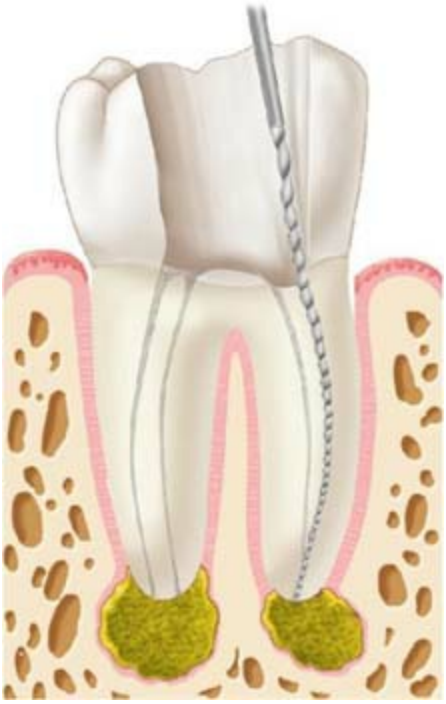

FIG. 7.45 A small K-file negotiates the canal to its terminus. An apex locator or radiograph is used to confirm the file's position.

Tooth shows green patches at the root tips and a small K-file curved slightly while passing from the root canal on the left.

Crowded or rotated teeth

Traditional or minimal access preparations may not be possible in patients with crowded or rotated teeth. The decision about an alternative approach must be based on straight-line access principles and conservation of tooth structure.[58] In certain circumstances, a buccal or incisal access preparation may be indicated, or developing the standard shape based on the degree of rotation (Fig. 7.46).[39]

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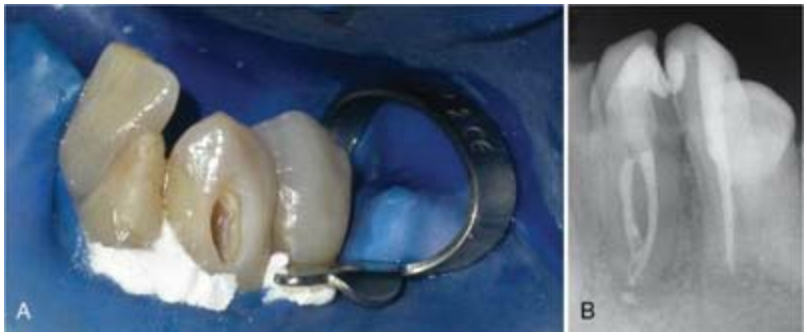

FIG. 7.46 **A,** Access cavity on crowded mandibular anterior teeth. The access preparation is cut through the buccal surface on the canine. The lateral incisor has also been accessed through the buccal surface; root canal procedures were performed, and the access cavity was permanently restored with composite. **B,** Obturation.

A) Close-up view shows a hollow chamber in the front tooth. The adjacent tooth is hold by a steel pear-shaped instrument.

B) Radiograph shows filled pulp chamber and root canals.

Other problems can occur when tooth angulations are not considered during access development, such as:

- Mistaken identification of an already located canal, resulting in a search in the wrong direction for additional canals. Whenever a difficult canal is located, a file should be placed in the canal and an angled radiograph taken.
- Failure to locate a canal or extra canals.
- Excessive gouging of coronal or radicular tooth structure.
- Instrument separation during attempts to locate an orifice.
- Failure to débride all pulp tissue from the chamber.

Morphology of and access cavity preparations for individual teeth

The anatomy shown in the following figures was obtained from human teeth through the use of recently developed 3D imaging techniques. The teeth were scanned in a high-resolution, microcomputer-assisted tomographic scanner. The data were then manipulated with proprietary computer programs to produce the 3D reconstructions and visualization. The following individuals

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and sources are recognized to have contributed greatly to this endeavor.

Maxillary central incisor

The root canal system outline of the maxillary central incisor reflects the external surface outline (Fig. 7.47) (see Table 7.8 online at the Expert Consult site). The μCT scans of this tooth are seen in Fig. 7.48. (See Video 7.3 online at the Expert Consult site for rotational views of these teeth.) The external access outline form for the maxillary central incisor is a rounded triangle with its base toward the incisal aspect (Fig. 7.49).

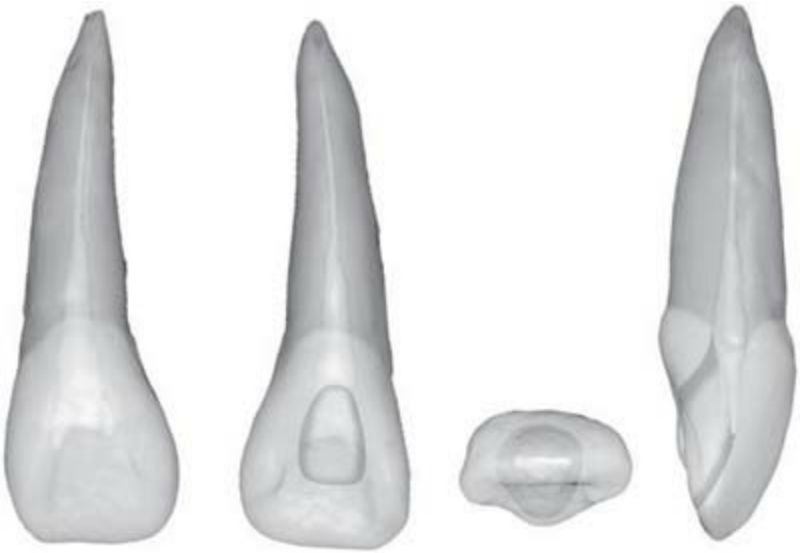

FIG. 7.47 Maxillary central incisor. Average time of eruption—7 to 8 years; average age of calcification—10 years; average length—22.5 mm. Root curvature (most common to least common): straight, labial, distal.

First model shows wedge-shaped root canal with tapering root tip. Second model shows an oval outline with broader base in the crown. Third model shows roughly rectangular structure with a depression at the center. Fourth model shows two lines extending from pulp to edge of crown.

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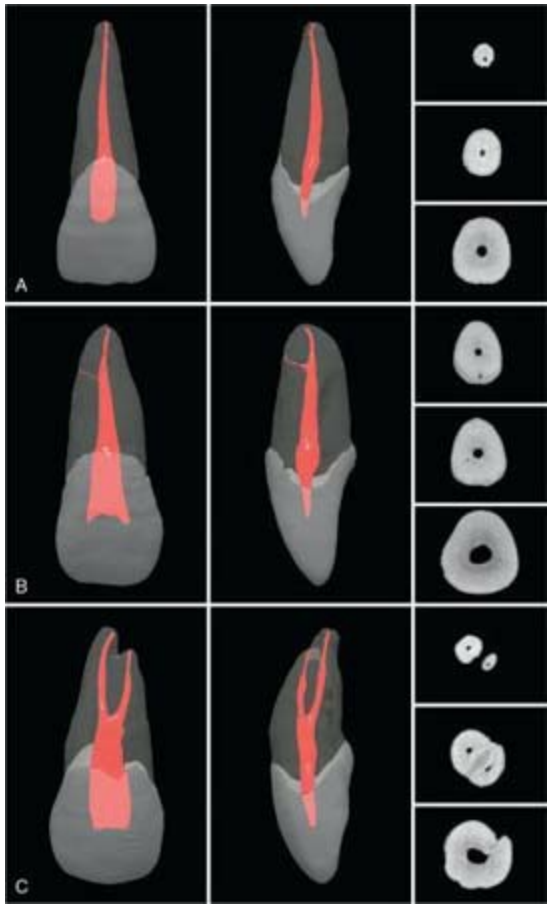

FIG. 7.48 µCT scans of maxillary central incisors. **A,** Common anatomic presentation. **B,** Central incisor with a lateral canal, which is common. **C,** Rare multiple-canal variation. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.3 online at the Expert Consult site for rotational views of these teeth.)

A) First µ CT scan shows triangular crown with a wedge-shaped root canal extending from pulp chamber to apex. Second µ CT scan shows U-shaped edge at the lower part of crown with a thin convex root canal. Their coronal, middle, and apical views show a small hole near the edge, slightly large hole at the center, and a large hole surrounded by dark patch at the center, respectively.

B) First µ CT scan shows oval-shaped crown with two pulp horns above the pulp chamber and a very thin vertical line extending near the root apex. Second µ CT scan shows U-shaped edge at the lower part of crown with a curved thin branch near the root apex. Their coronal, middle, and apical views show oval shape with a small hole at the center and another very small hole near the base, slightly large hole at the center, and very large circular hole surrounded by dark patch at the center, respectively.

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C) First μ CT scan shows a wide square canal branched into two. Second μ CT scan shows thin canal branched into two. Their coronal, middle, and apical views show two circular structures each with a tiny hole inside, two holes in the tooth, and a circular hole in the tooth, respectively.

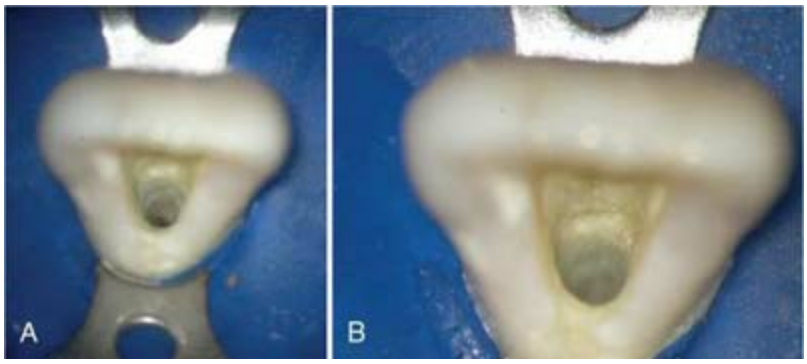

FIG. 7.49 Access cavity for a maxillary central incisor as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×8.4 magnification.

A) A triangular tooth with a small hollow chamber inside.

B) A large triangular tooth with a large hollow chamber inside.

Maxillary lateral incisor

The pulp chamber outline of the maxillary lateral incisor is similar to that of the maxillary central incisor; however, it is smaller, and two or no pulp horns may be present (Fig. 7.50) (see Table 7.9 online at the Expert Consult site). The μCT scans of this tooth are shown in Fig. 7.51. (See Video 7.4 online at the Expert Consult site for rotational views of these teeth.)

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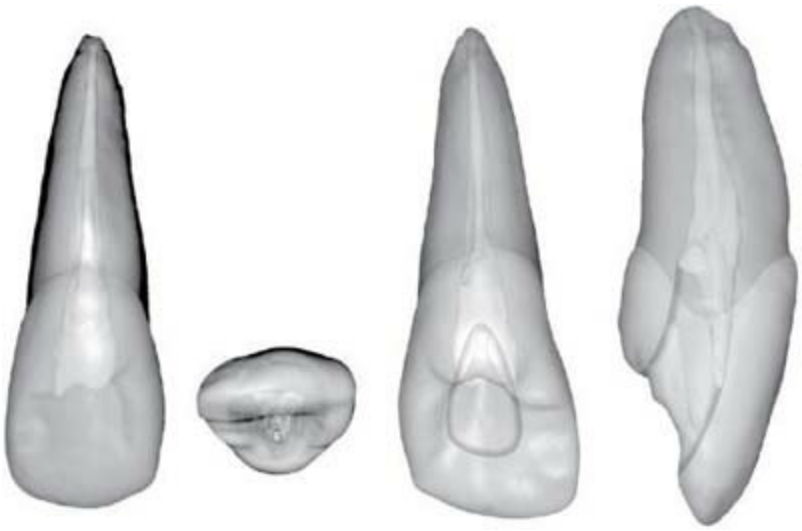

FIG. 7.50 Maxillary lateral incisor. Average time of eruption—8 to 9 years; average age of calcification—11 years; average length—22 mm. Root curvature (most common to least common): distal, straight.

First model shows normal anatomy of maxillary incisor with an oval crown and wedge-shaped root canal. Second model shows occlusal view of tooth. Third model shows tear-drop outline in the crown. Fourth model shows comparatively dense root canal.

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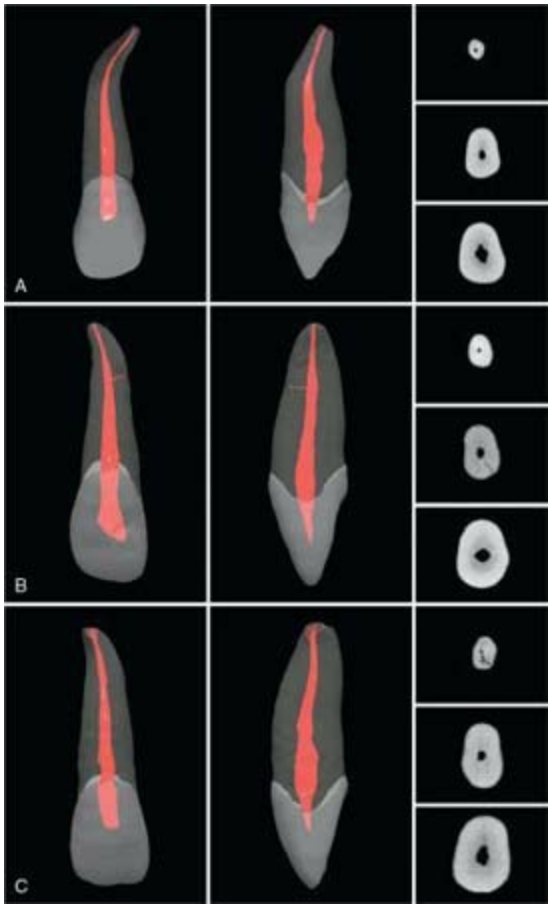

FIG. 7.51 µCT scans of maxillary lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with a large lateral canal, which is common. **C,** Lateral incisor with an apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.4 online at the Expert Consult site for rotational views of these teeth.)

A) First µ CT scan shows nearly oval crown with a canal at the center that bends near the apex. Second µ CT scan shows U-shaped edge at the lower part of crown with a thick convex root canal that narrows down toward the apex. Their coronal, middle, and apical views show circular structures each with a hole inside.

B) First µ CT scan shows the secondary branch near the root apex on the right. Second µ CT scan shows heart-shaped crown with the secondary near the root apex on the left. Their coronal, middle, and apical views show circular structures each with a hole inside and an extra line.

C) The general appearance is similar to above CT scans, except that there are multiple branches in the root tip. Their coronal view shows multiple holes, whereas middle, and apical views show oval structures with holes inside.

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Maxillary canine

The root canal system of the maxillary canine is similar in many ways to that of the maxillary incisors (Fig. 7.52). A major difference is that it is wider labiolingually than mesiodistally. Another difference is that it has no pulp horns. Usually one root canal is present, although two canals have been reported (see Table 7.10 online at the Expert Consult site). The μCT scans for the maxillary canine can be seen in Fig. 7.53. (See Video 7.5 online at the Expert Consult site for rotational views of these teeth.)

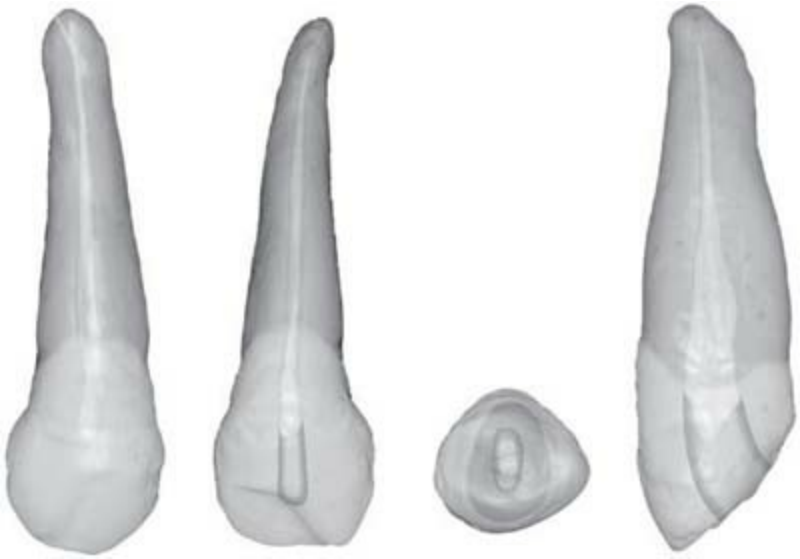

FIG. 7.52 Maxillary canine. Average time of eruption—10 to 12 years; average age of calcification—13 to 15 years; average length—26.5 mm. Root curvature (most common to least common): distal, straight, labial.

The normal external morphology of maxillary canine is followed by second model that has a U-shaped lucency in the crown, which is again followed by third model depicting the occlusal view of tooth and fourth model depicting two lucent canals in the crown.

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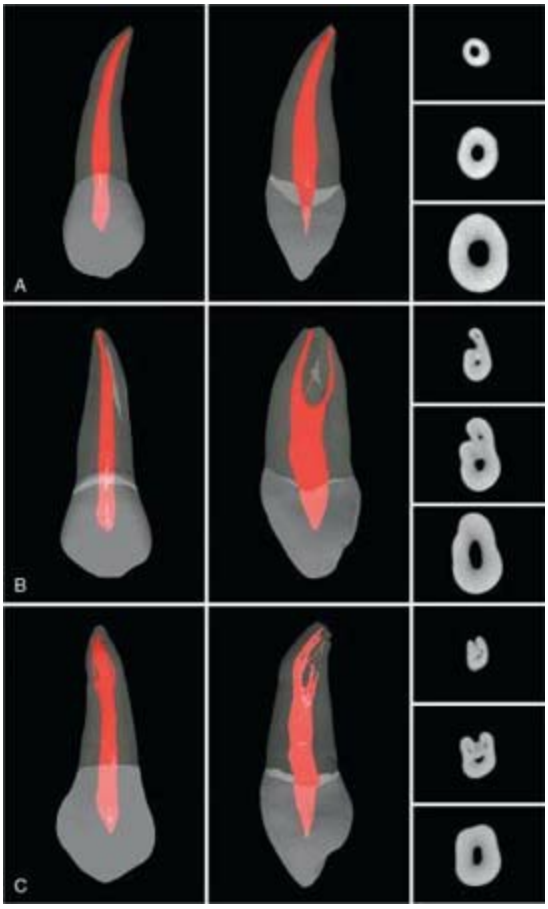

FIG. 7.53 µCT scans for the maxillary canine. **A,** Common anatomic presentation. **B,** Canine with two roots. **C,** Canine with significant deviations of the canal system in the apical third. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.5 online at the Expert Consult site for rotational views of these teeth.)

A) First µ CT scan shows nearly oval crown with a slightly deviated root canal. Second µ CT scan shows U-shaped edge at the lower part of crown with a thick convex root canal that narrows down toward the apex. Their coronal, middle, and apical views show circular structures each with a hole inside.

B) First µ CT scan shows the straight root canal. Second µ CT scan shows triangular crown with the secondary branch near the root apex on left. Their coronal and middle show 6-shaped structure with two holes. The apical view shows an oval hole at the center of structure.

C) The general appearance is similar to above CT scans, except that there are multiple branches in the root tip. Their coronal and middle views show multiple holes, whereas the apical view shows squarish structures with a hole inside.

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Maxillary first premolar

Most maxillary first premolars have two root canals, regardless of the number of roots (Fig. 7.54). Ethnicity plays a factor in that Asian people have a higher incidence of one canal than do other ethnic groups.[62,81] A furcation groove or developmental depression on the palatal aspect of the buccal root is another anatomic feature. Its prevalence has been reported as 62% to 100%.[42,53,108] At the deepest part of the invagination, the average dentin thickness was found to be 0.81 mm. The pulp chamber of the maxillary first premolar is considerably wider buccolingually than mesiodistally. In the buccolingual dimension, the chamber outline shows a buccal and a palatal pulp horn. The buccal pulp horn usually is larger. The maxillary first premolar may have one, two, or three roots and canals; it most often has two (see Table 7.11 online at the Expert Consult site). The μCT scans for the maxillary first premolar can be seen in Fig. 7.55. (See Video 7.6 available online at the Expert Consult site for rotational views of these teeth.)

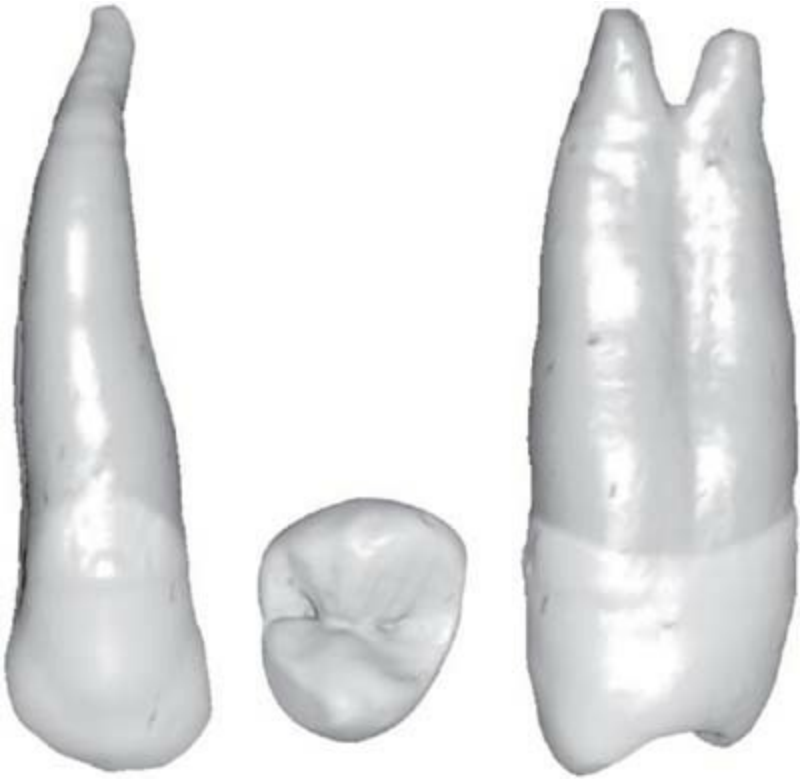

FIG. 7.54 Maxillary first premolar. Development and anatomic data:

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average time of eruption—10 to 11 years; average age of calcification —12 to 13 years; average length—20.6 mm. Root curvature (most common to least common): buccal root—lingual, straight, buccal; palatal root—straight, buccal, distal; single root—straight, distal, buccal.

The first model of normal external morphology of maxillary first premolar is followed by second model that shows occlusal view, which is again followed by third model depicting two-rooted tooth.

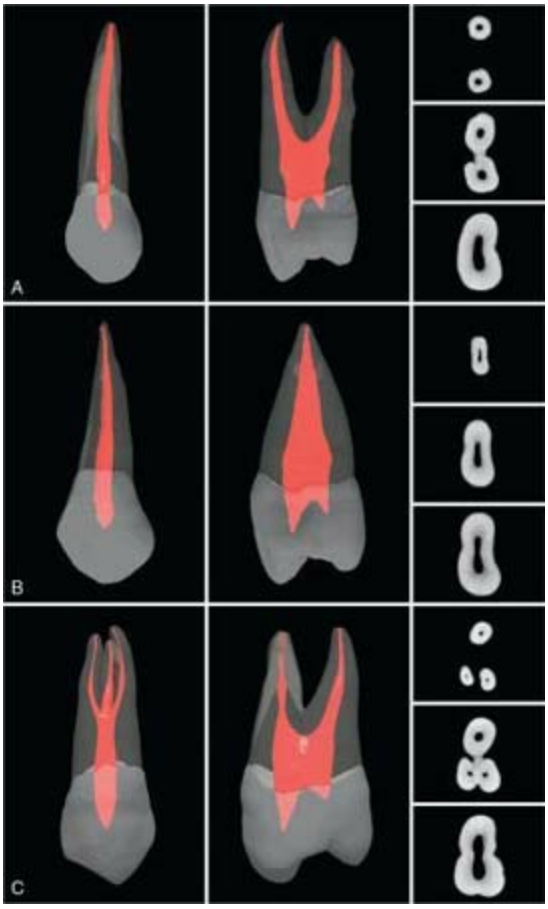

FIG. 7.55 µCT scans of maxillary first premolars. **A,** Common anatomic presentation of this tooth showing two roots. **B,** Premolar with only one canal. **C,** Premolar with three roots. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.6 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans of first premolar show a little deviated canal and two root canals with two pulp horns, respectively. Three cross-sectional views on the right show two rings, two rings forming an 8-shape, and an oval chamber,

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respectively.

B) Two μ CT scans of premolar show wedge-shaped root canal and a root canal with two pulp horns, respectively. Three different views of tooth on the right show the coronal section with a smaller hole, middle section with a medium oval hole, and apical section with large hole.

C) Two μ CT scans show a trifurcated root canal near the root apex and bifurcated root canal with a V-shaped space in-between. Three different cross-sectional views of tooth on the right show three rings, fused rings, and a hollow chamber, respectively.

Maxillary second premolar

The root canal system of the maxillary second premolar is wider buccolingually than mesiodistally (Fig. 7.56). This tooth may have one, two, or three roots and canals (see Table 7.12 online at the Expert Consult site). Two or three canals can occur in a single root.[32] The scans for the maxillary second premolar can be seen in Fig. 7.57. (See Video 7.7 online at the Expert Consult site for rotational views of these teeth.)

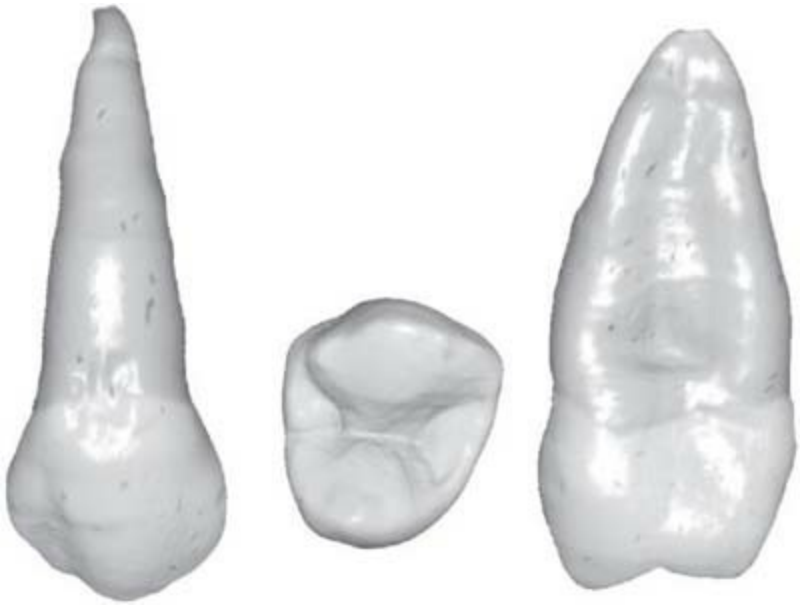

FIG. 7.56 Maxillary second premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 14 years; average length—21.5 mm. Root curvature (most common to least common): distal, bayonet, buccal, straight.

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First model shows a triangular shape of premolar, followed by second model depicting occlusal view. Third molar shows depression in the root.

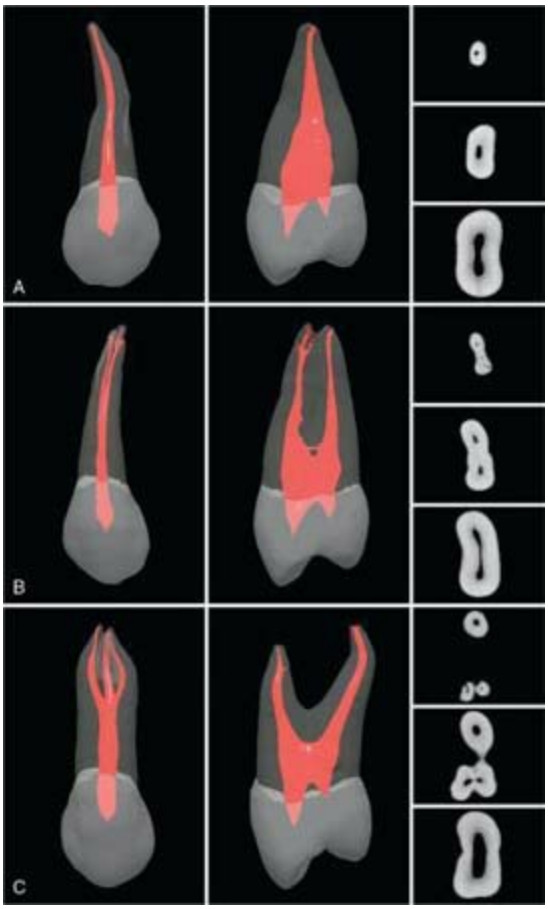

FIG. 7.57 μCT scans of maxillary second premolars. **A,** Common anatomic presentation showing one canal. **B,** Second premolar with two canals and an apical delta. **C,** Second premolar with three roots/canals that divide at the junction of the middle and apical third of the main root. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.7 online at the Expert Consult site for rotational views of these teeth.)

A) Two μ CT scans show deviated root canal, heart-shaped tooth with two pulp horns, respectively. Coronal section is with a smaller hole, middle section is with a medium oval hole, and apical section is with large vertical hole.

B) Two μ CT scans of tooth show branched root canal near the tip and two root canals where one of the root canals is branched out at the tip, respectively. Coronal section shows two very small holes. Middle section has two medium-sized oval holes. Apical section shows a vertical large slit in the cavity.

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C) Two μ CT scans of tooth show root canal with three branches and two root canals with a V-shaped cavity in-between. Coronal and middle section show three small holes and large holes, respectively. Apical view shows an oval-shaped hole.

Maxillary first molar

The maxillary first molar is the largest tooth in volume and one of the most complex in root and canal anatomy (Fig. 7.58).[25] The pulp chamber is widest in the buccolingual dimension, and four pulp horns are present (mesiobuccal, mesiopalatal, distobuccal, and distopalatal). The pulp chamber's cervical outline form has a rhomboid shape, sometimes with rounded corners. The mesiobuccal angle is an acute angle; the distobuccal angle is an obtuse angle; and the palatal angles are basically right angles. The palatal canal orifice is centered palatally; the distobuccal orifice is near the obtuse angle of the pulp chamber floor; and the main mesiobuccal canal orifice is buccal and mesial to the distobuccal orifice and is positioned within the acute angle of the pulp chamber. When there is only one canal in mesiobuccal root, the orifice can be round, oval, or flat. (See Video 7.8 online at the Expert Consult site for rotational views of these teeth.) The mesiopalatal canal orifice (also referred to as the *MB-2*) is located palatal and mesial to the mesiobuccal orifice. It can be located close to or far away from mesiobuccal orifice. (See Video 7.8 online at the Expert Consult site for rotational views of these teeth.) A line drawn to connect the three main canal orifices—the mesiobuccal orifice, distobuccal orifice, and palatal orifice—forms a triangle, known as the *molar triangle* (see Tables 7.13–7.15 online at the Expert Consult site). The palatal root often curves buccally at the apical one third, which may not be obvious on a standard periapical radiograph. The μCT scans for the maxillary first molar can be seen in Fig. 7.59. (See Video 7.9 online at the Expert Consult site for rotational views of these teeth.)

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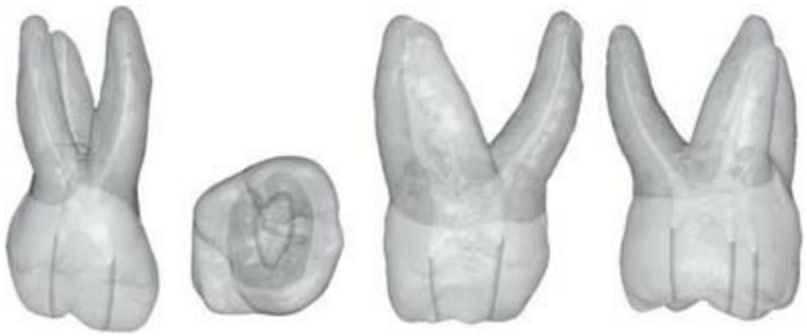

FIG. 7.58 Maxillary first molar. Average time of eruption—6 to 7 years; average age of calcification—9 to 10 years; average length—20.8 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—buccal, straight.

First model of tooth shows three root canals followed by second model depicting occlusal view with diamond shaped patch inside surrounded by thin tissue. Third molar shows V-shaped root canals toward the right. Fourth molar shows V-shaped root canals toward the left.

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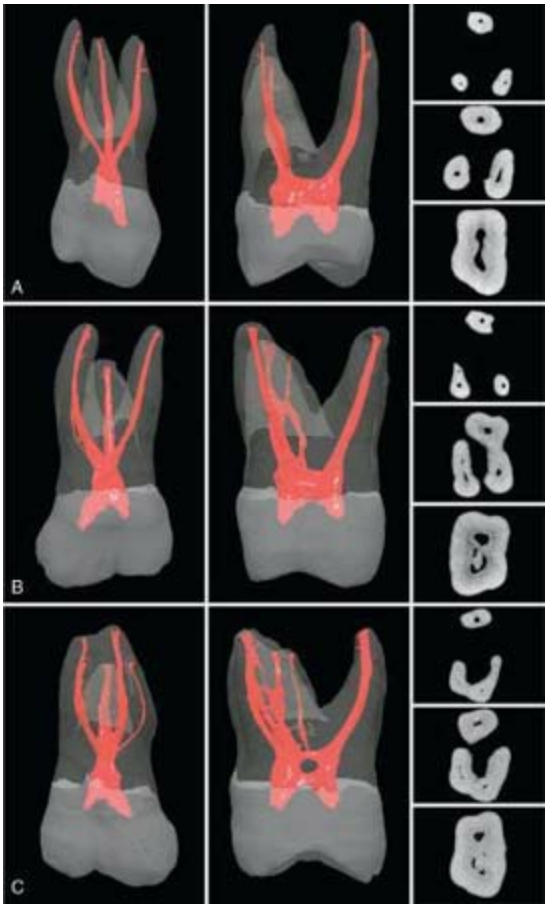

FIG. 7.59 µCT scans of maxillary first molars. **A,** Common anatomic presentation showing accessory/lateral canals. **B,** First molar with four canals, with mesiobuccal and mesiopalatal sharing an anastomosis in the midroot. **C,** Maxillary molar with four pulp horns, five canals, and significant anastomoses between the canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.9 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans of tooth show three root canals and two root canals with secondary branches, respectively. Coronal section shows three structures each with a hole inside, middle section is with slightly large holes, and apical section is with a single large hole.

B) Two µ CT scans of tooth show smaller root canal at the center and three root canals forming a connection with a separate root canal, respectively. Coronal section shows three structures each with a hole inside. Middle section shows two structures, each with two holes. Apical section shows a rectangular structure with two holes.

C) Two µ CT scans of tooth show four root canals and multiple canals with a circular cavity in the pulp chamber, respectively. Coronal and medial sections

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show a circular structure with one hole and U-shaped structure with three holes inside. Apical section shows a rectangular structure with holes inside.

Maxillary second molar

Coronally, the maxillary second molar may closely resemble the maxillary first molar (Fig. 7.60). The root and canal anatomy are similar to those of the first molar, although there are differences. The distinguishing morphologic feature of the maxillary second molar is that its three roots are generally grouped closer together and are sometimes fused (see Tables 7.16–7.18 online at the Expert Consult site). The μCT scans for the maxillary second molar can be seen in Fig. 7.61. (See Video 7.10 online at the Expert Consult site for rotational views of these teeth.)

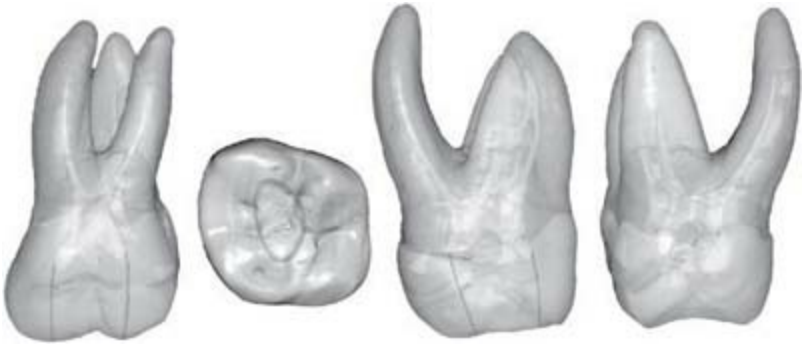

FIG. 7.60 Maxillary second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 16 years; average length—20 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—straight, buccal.

First model of second molar shows three visible root canals followed by second model depicting occlusal view with oval patch inside. Third molar shows V-shaped root canals extending toward the left. Fourth molar shows V-shaped root canals extending toward the right.

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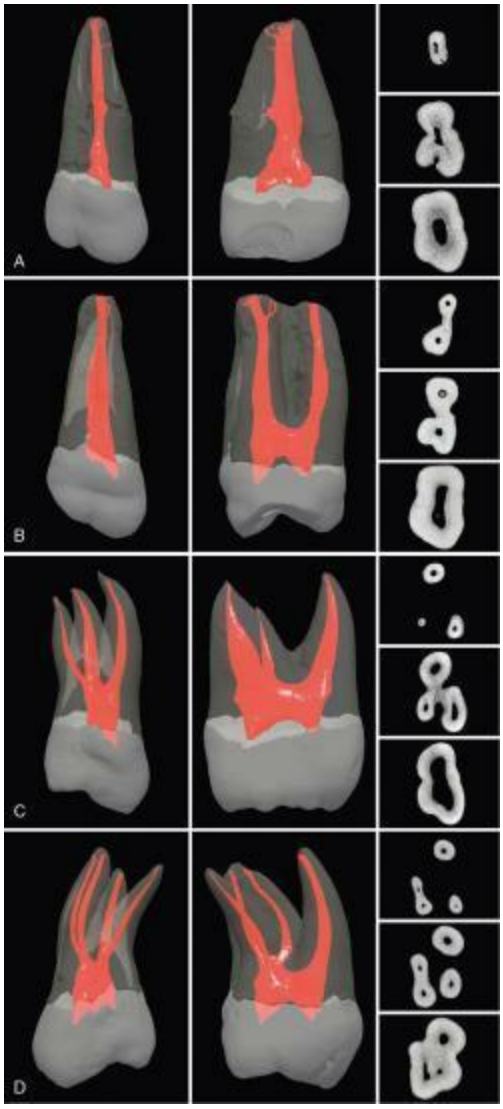

FIG. 7.61 µCT scans of maxillary second molars; four possible variations. **A,** Uncommon anatomic presentation of this tooth with one canal. **B,** Second molar with two canals. **C,** Second molar with three canals. **D,** Second molar with four distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.10 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show teeth with root canals extending from below the crown. The right side shows structures with varying size of hole inside.

B) Two µ CT scans show tooth on left one root canal and tooth on right with U-shaped root canal with branches at the root tip on left, respectively. On the right, two structures show two fused chambers each with a hole inside. The second structure has larger holes. The third structure has large cavity at the center of rectangular structure.

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C) Two μ CT scans show tooth on left with trifurcated roots and tooth on right with U-shaped canal with an additional branch extending below the pulp. The right side shows their coronal, medial, and apical views.

D) Two μ CT scans show tooth on left with four root canals diverting toward the right and tooth on right with four root canals diverting toward the left, respectively. In the right tooth, two root canals are fused. The right side shows their coronal, medial, and apical views.

Maxillary third molar

Loss of the maxillary first and second molars is often the reason the third molar must be considered a strategic abutment (Fig. 7.62). However, sometimes a third molar is intentionally transplanted to a vacated first molar position; the same being true for mandibular third molars.[48] The radicular anatomy of the third molar is completely unpredictable, and it may be advisable to explore the root canal morphology to evaluate the likelihood and degree of success. This tooth can have one to four roots and one to six canals, and C-shaped canals also can occur. The third molar usually has three roots and three root canals (see Table 7.19 online at the Expert Consult site). The tooth may be tipped significantly to the distal, the buccal, or both, which creates an even greater access problem than with the second molar. The μCT scans for the maxillary molar can be seen in Fig. 7.63. (See Video 7.11 online at the Expert Consult site for rotational views of these teeth.)

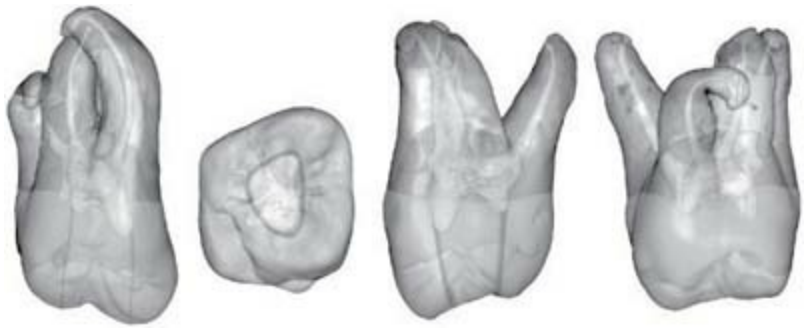

FIG. 7.62 Maxillary third molar. Average time of eruption—17 to 22 years; average age of calcification—18 to 25 years; average length—17 mm.

First model of second molar shows root canals group together. Second model shows occlusal view with triangular-shaped patch inside. Third molar shows

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V-shaped root canals aligned toward the right. Fourth molar shows posterior view of tooth with depression at the center.

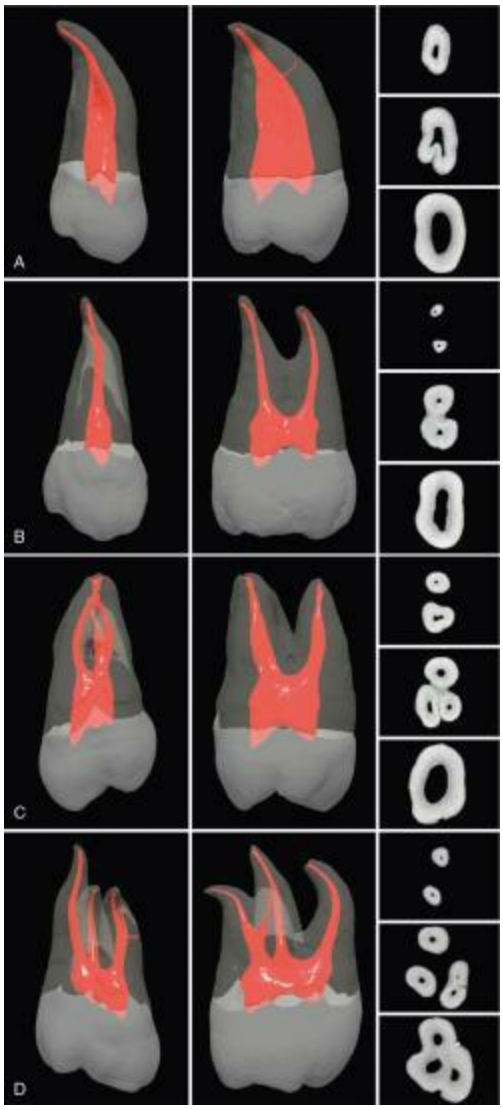

FIG. 7.63 µCT scans of maxillary third molars show a range of anatomic variations. **A,** Single-canal tooth. **B,** Two-rooted third molar. **C,** Two-rooted, three-canal third molar with significant root curvatures. **D,** Three-rooted, four-canal third molar. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.11 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scan of tooth show a deviated wedge-shaped and a heart-shaped root canal, respectively. Coronal section shows a small oval-shaped hole,

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whereas middle and apical view show a heart-shaped and large oval hole, respectively.

B) Two μ CT scans show single-rooted tooth with an accessory branch and one-rooted tooth with pulp horns at the lower part of crown. Coronal section shows two rings. Middle section and apical section show two fused rings and a rectangular structure with a large vertical hole inside, respectively.

C) Two μ CT scans show three interconnected root canals and two root canals extending from the lower part of crown, respectively. Coronal, middle, and apical section show two rings, three-fused rings, and a large hole at the center of structure, respectively.

D) Two μ CT scans show three-rooted tooth with addition branches on the left and three-rooted tooth with an additional brand on the right, respectively. Coronal, middle, and apical section show two rings, two rings with an 8-shape structure, and three fused rings, respectively.

Mandibular central and lateral incisors

The root canal systems and access cavities for the two mandibular incisors are so similar they are discussed together (Fig. 7.64). As with the maxillary incisors, a lingual shoulder must be eliminated to allow direct-line access. The shoulder conceals the orifice to a second canal that, if present, is found immediately beneath it. Most mandibular incisors have a single root with what radiographically appears to be a long, narrow canal. However, it is a broad canal labiolingually. Often a dentinal bridge is present in the pulp chamber that divides the root into two canals. The two canals usually join and exit through a single AF, but they may persist as two separate canals (Figs. 7.65 and 7.66). Occasionally, one canal branches into two canals, which subsequently rejoin into a single canal before reaching the apex (see Table 7.20 online at the Expert Consult site). (See Videos 7.12 and 7.13 online at the Expert Consult site for rotational views of these teeth.)

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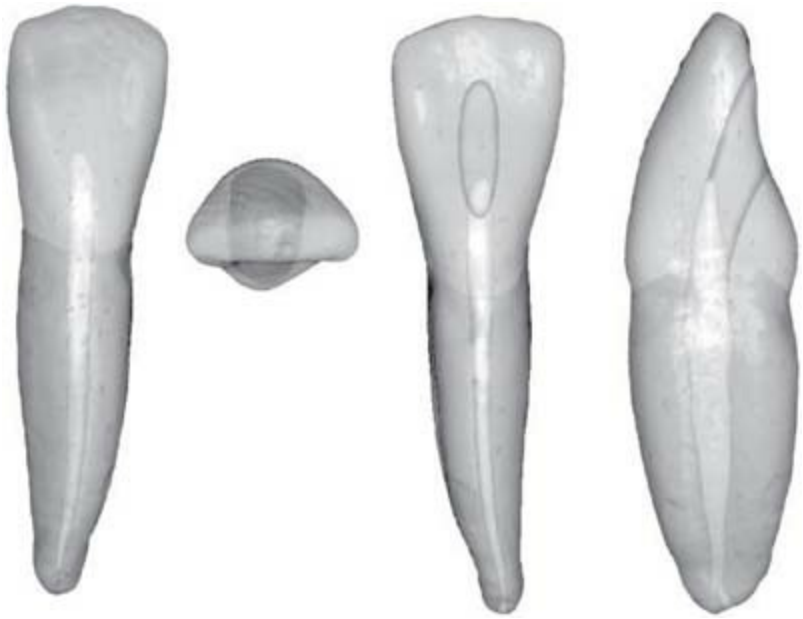

FIG. 7.64 Mandibular central/lateral incisors. Average time of eruption —6 to 8 years; average age of calcification—9 to 10 years; average length—20.7 mm. Root curvature (most common to least common): straight, distal, labial.

First model shows posterior side of incisor with filled root canal. Second model shows small mushroom-shaped structure of tooth. Third model shows tooth with an oval mark at the center. Fourth model shows curved root canal.

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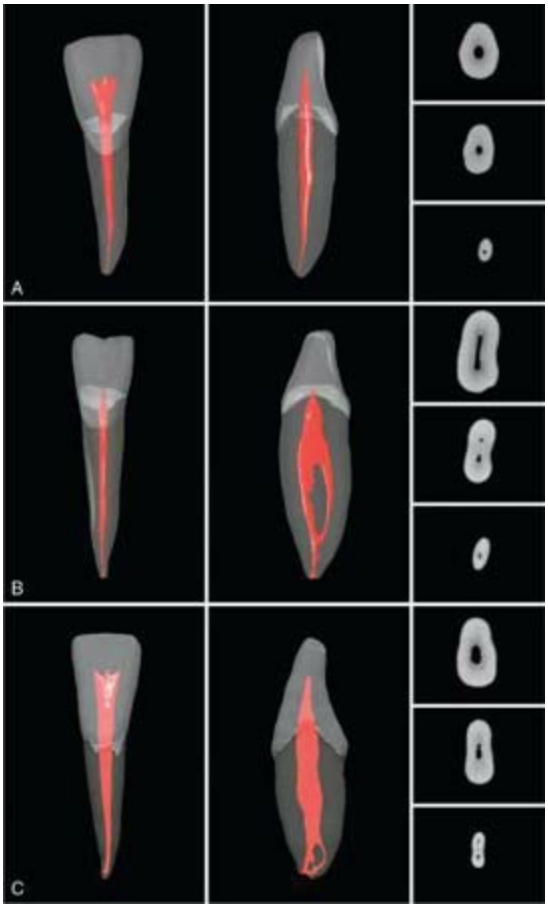

FIG. 7.65 µCT scans of mandibular central incisors. **A,** Common anatomic presentation. **B,** Central incisor with two canals. **C,** Central incisor with an apical delta. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.12 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show root canal with three pulp horns and wedge-shaped root canal, respectively. Coronal, middle, and apical views show a large hole, medium hole, and a small hole inside the rings.

B) Two µ CT scans of tooth show very thin root canal and a hollow chamber in the root canal, respectively. Coronal, middle, and apical views show a large vertical hole, two small holes in a dumb-bell shape, and an ellipsoid ring with a small hole, respectively.

C) Two µ CT scans of tooth show posterior view and proximal view with branches at the root apex. Coronal, middle, and apical views show a large oval hole, medium triangular hole, and two small holes inside the structure.

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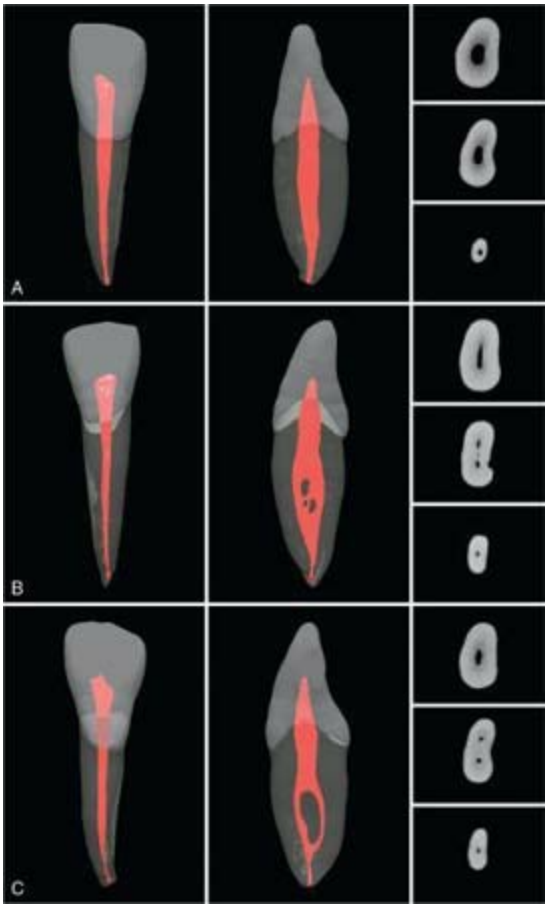

FIG. 7.66 µCT scans of mandibular lateral incisors. **A,** Common anatomic presentation. **B,** Lateral incisor with broad, thin buccolingual anatomy. **C,** Lateral incisor in which the canal splits into two but returns to form one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.13 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show a wedge-shaped root canal in tooth and tapering edges of crown with convex-shaped root canal in tooth on right, respectively. Three structures on the right show oval-shaped large hole, medium hole, and small hole, respectively.

B) Two µ CT scans show posterior view of tooth and hollow chambers in the root canal, respectively. Three structures on the right show a large vertical hole, three small holes, and a small oval-shaped hole, respectively.

C) Two µ CT scans show root canal with pulp horns tapering at the apex and a large chamber in the root canal of right tooth, respectively. Three structures on the right show an oval aperture, two holes, a single hole at the center, respectively.

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Mandibular canine

The root canal system of the mandibular canine is very similar to that of the maxillary canine, except that the dimensions are smaller, the root and root canal outlines are narrower in the mesiodistal dimension, and the mandibular canine occasionally has two roots and two root canals located labially and lingually (Fig. 7.67) (see Table 7.21 online at the Expert Consult site). The root canal of the mandibular cuspid is narrow mesiodistally but usually very broad buccolingually, and the access cavity for the mandibular canine is oval or slot-shaped.[112] The μCT scans of the mandibular canine can be seen in Fig. 7.68. (See Video 7.14 online at the Expert Consult site for rotational views of these teeth.)

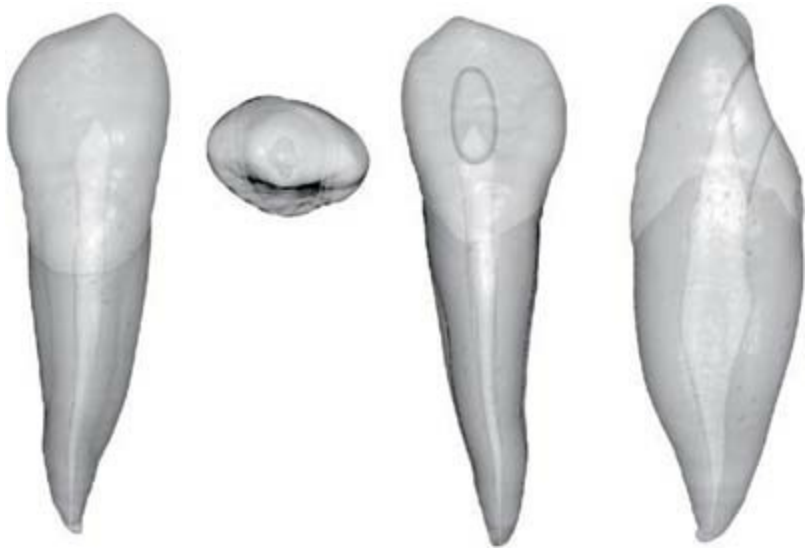

FIG. 7.67 Mandibular canine. Average time of eruption—9 to 10 years; average age of calcification—13 years; average length—25.6 mm. Root curvature (most common to least common): straight, distal, labial.

First model shows posterior side of incisor with filled root canal. Second model shows small mushroom-shaped structure of tooth. Third model shows tooth with an oval mark at the center. Fourth model shows curved root canal.

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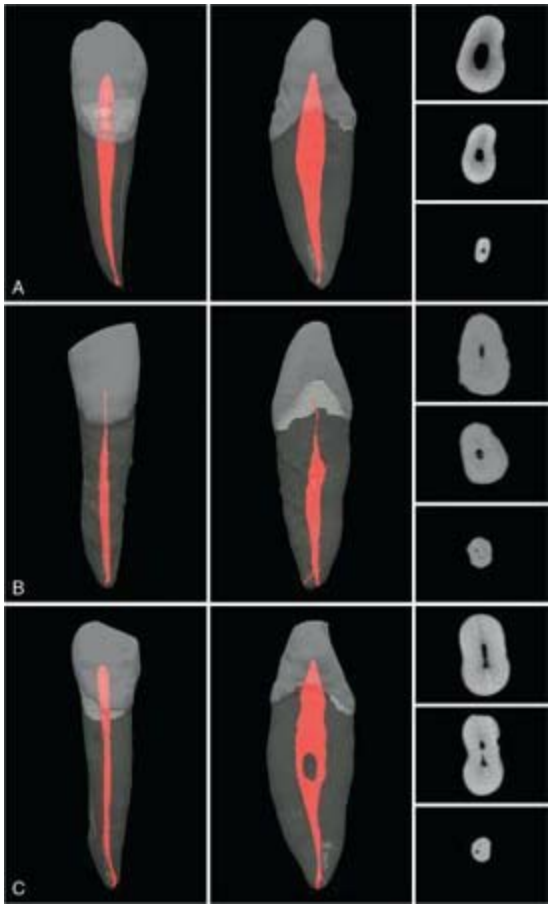

FIG. 7.68 µCT scans of mandibular canines. **A,** Common anatomic presentation. **B,** Canine with an extra apical canal. **C,** Canine that splits into two but returns to one canal apically. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.14 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show a narrow root canal with a tapering end and a wide root canal, respectively. Three structures on the right show oval-shaped large hole, medium hole, and small hole, respectively.

B) Two µ CT scans show front view of tooth with tapering root canal in the crown and root canal with a secondary branch in right tooth, respectively. Three structures on the right show an oval hole, a smaller hole, and two tiny dots, respectively.

C) Two µ CT scans show root canal with tapering end at the root apex and a large oval hole at the center of right tooth, respectively. Three structures on the right show a vertical aperture, two holes, and a tiny hole near the periphery, respectively.

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Mandibular first premolar

As a group, the mandibular premolars present significant anatomic challenges because of the extreme variations in their root canal morphology (Fig. 7.69).[63,82,121] The root canal system of the mandibular first premolar is wider buccolingually than mesiodistally.[123] Two pulp horns are present: a large, pointed buccal horn and a small, rounded lingual horn. At the cervical line, the root and canal are oval; this shape tends to become round as the canal approaches the middle of the root. If two canals are present, they tend to be round from the pulp chamber to their foramen. Direct access to the buccal canal is usually possible, whereas the lingual canal may be quite difficult to find. To address this situation, the lingual wall of the access cavity may have to be extended farther lingually; this makes the lingual canal easier to locate. The mandibular first premolar sometimes may have three roots and three canals (see Table 7.22 online at the Expert Consult site). Multiple studies have reported C-shaped canal anatomy in this tooth.[10,29,31,68,80] The μCT scans for the mandibular first premolar can be seen in Fig. 7.70. (See Video 7.15 online at the Expert Consult site for rotational views of these teeth.)

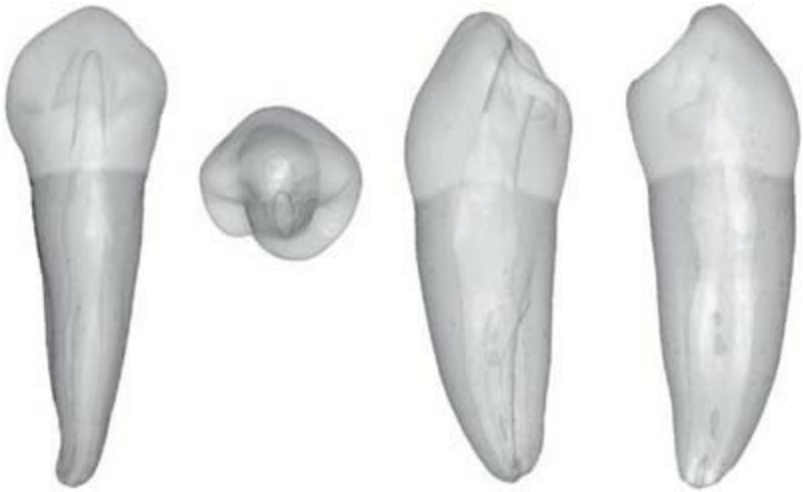

FIG. 7.69 Mandibular first premolar. Average time of eruption—10 to 12 years; average age of calcification—12 to 13 years; average length—21.6 mm. Root curvature (most common to least common): straight, distal, buccal.

First model shows single-rooted tooth with filled root canal extending from the enamel to root apex. Second model shows occlusal view of tooth with a groove inside. Third model shows diverted root canal. Fourth model shows

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crown tapering at the edge.

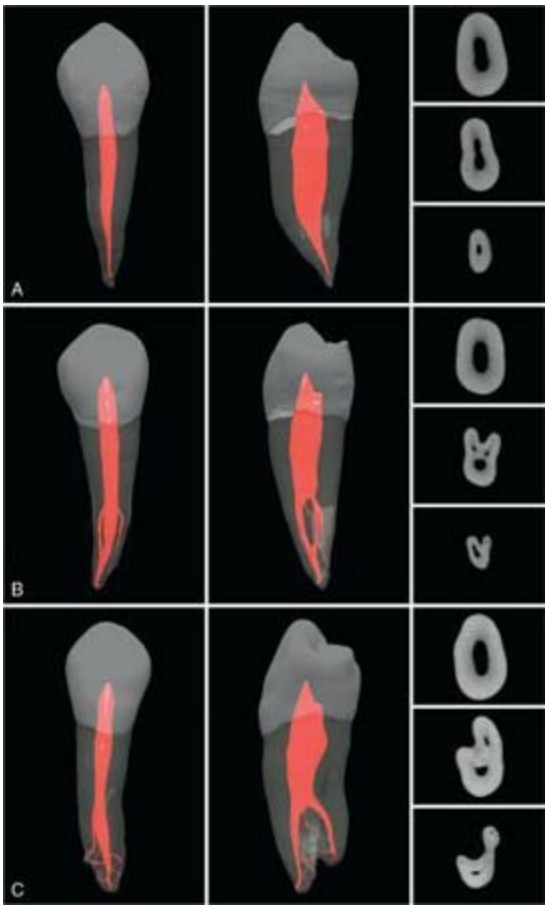

FIG. 7.70 µCT scans of mandibular first premolars. **A,** Common anatomic presentation. **B,** First premolar with significant canal deviations in the middle to apical third before returning to a single large canal apically and a small deviating canal to the proximal. **C,** First premolar with a branching main canal lingually and multiple accessory canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.15 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show a normal root canal in tooth and tapering edges of crown with a deviating root canal at apex, respectively. Three structures on the right show oval-shaped large hole, medium hole, and small hole, respectively.

B) Two µ CT scans show tooth with two branches toward the apex and a hollow chamber in the root canal with secondary branches, respectively. Three structures on the right show a medium hole, a circular cavity with two small holes, and a small oval-shaped hole, respectively.

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C) Two μ CT scans show an irregular root canal with multiple branches and two-rooted tooth with multiple branches at the apex, respectively. Three structures on the right shows an oval aperture, two holes, a C-shape with a knob at one end and a hole at the center, respectively.

Mandibular second premolar

The mandibular second premolar is similar to the first premolar, with the following differences: The lingual pulp horn usually is larger, the root and root canal are more often oval than round, the pulp chamber is wider buccolingually, and the separation of the pulp chamber and root canal is normally distinguishable compared with the more regular taper in the first premolar (Fig. 7.71). The canal morphology of the mandibular second premolar is similar to that of the first premolar with its many variations: two, three, and four canals and a lingually tipped crown. Fortunately, these variations are found less often in the second premolar (see Table 7.23 online at the Expert Consult site). The μCT scans for the mandibular second premolar can be seen in Fig. 7.72. (See Video 7.16 online at the Expert Consult site for rotational views of these teeth.)

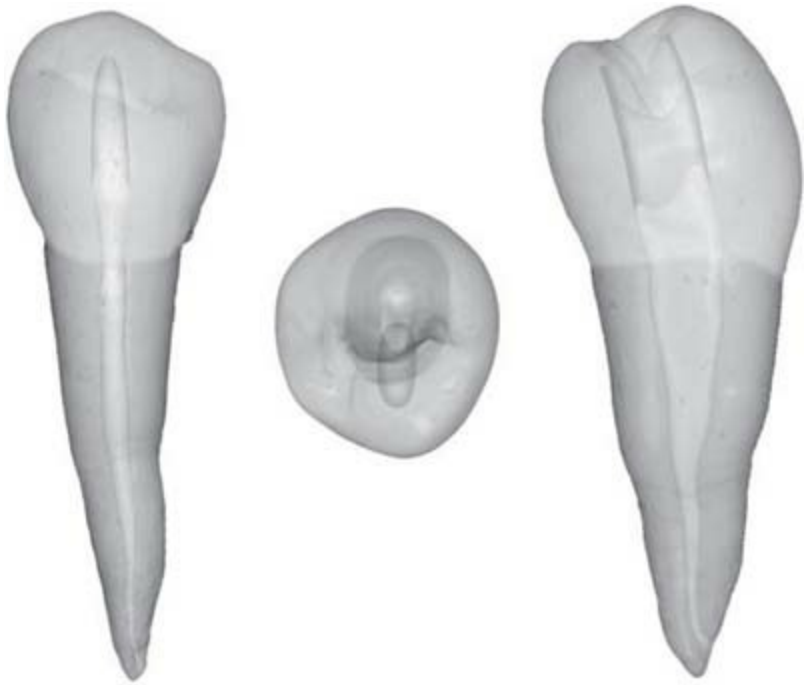

FIG. 7.71 Mandibular second premolar. Average time of eruption—11

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to 12 years; average age of calcification—13 to 14 years; average length—22.3 mm. Root curvature (most common to least common): straight, distal, buccal.

Three models represent second premolar as single-rooted tooth with the tapering end, a rounded structure, and a wide structure with two pulp horns above the pulp, respectively. Two lines from the pulp horns extend toward the crown apex.

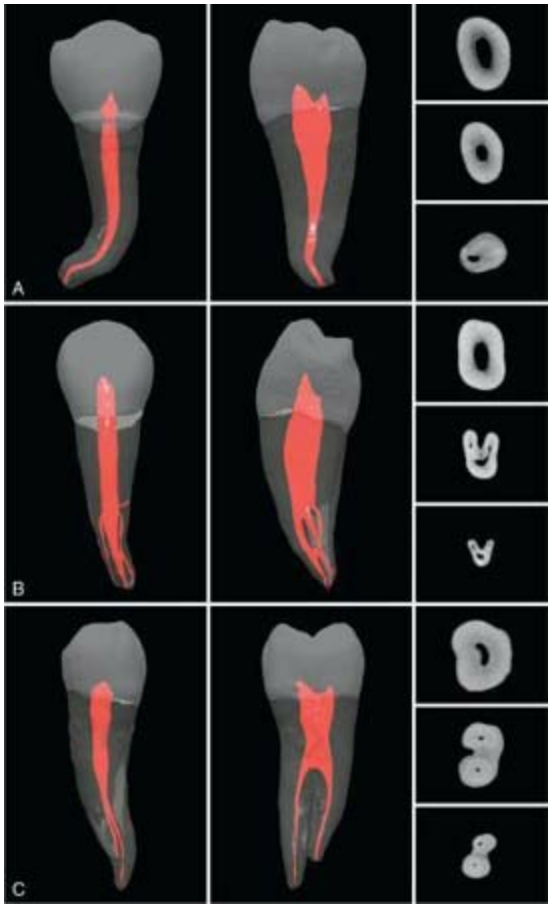

FIG. 7.72 µCT scans of mandibular second premolars. **A,** Common anatomic presentation. **B,** Second premolar with significant canal deviations in the middle to apical third. **C,** Second premolar with fused root that exhibits two distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.16 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show tapered end of the root canal and heart-shaped root canal extending from just above the crown with narrow ending, respectively.

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Three structures on the right show holes at the center.

B) Two μ CT scans show root canal with multiple branches near the root tip and interconnected branches of the root tip, respectively. The right side shows an oval structure with a hole, two-U shaped structures each with three holes. The second U-shaped structure is smaller.

C) Two μ CT scans show a root canal with two branches and two-rooted tooth. The right side shows oval structure with a vertical aperture inside, two structures each with two holes. The last structure is smallest.

Mandibular first molar

The mandibular first molar seems to be the tooth that most often requires an endodontic procedure (vital pulp capping, pulpotomy, root canal therapy); therefore, its morphology has received a great deal of attention (Fig. 7.73).[125] The occlusal appearance reflects a Y5 groove and cuspal pattern and the tooth usually has two roots, but occasionally it has three, with two or three canals in the mesial root and one, two, or three canals in the distal root (see Tables 7.24 and 7.25 online at the Expert Consult site). The μCT scans of the mandibular first molar can be seen in Fig. 7.74. (See Video 7.17 online at the Expert Consult site for rotational views of these teeth.)

FIG. 7.73 Mandibular first molar. Average time of eruption—6 years; average age of calcification—9 to 10 years; average length—21 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal.

Four model of mandibular first molar show curved roots, occlusal view, U-shaped root, and root canal extending from the neck region to apex, respectively.

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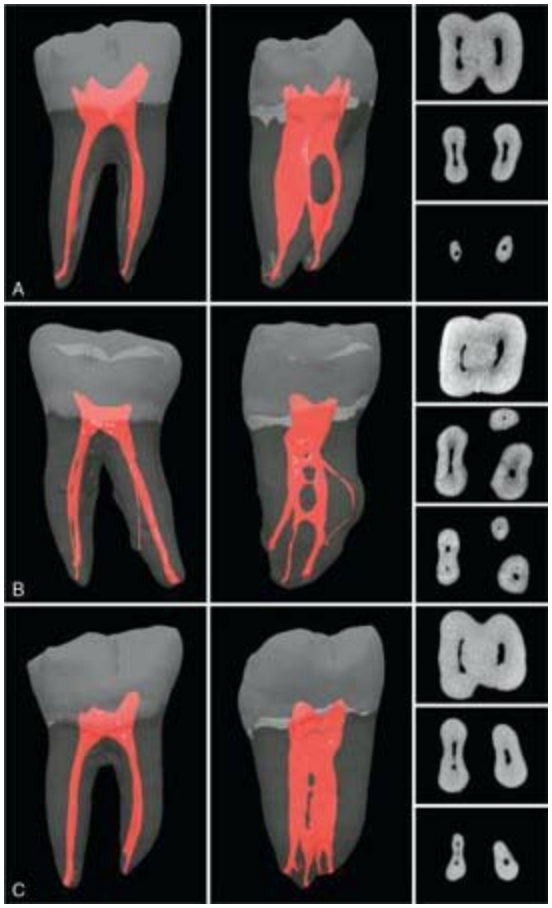

FIG. 7.74 µCT scans of mandibular first molars. **A,** Common anatomic presentation. **B,** First molar with three main canals and a deviant fourth canal/fourth root. **C,** First molar with wide connections or anastomoses between the mesial canals, demonstrating multiple canal exits. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.17 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show two root canals with three pulp horns of tooth and two-rooted tooth with an oval aperture, respectively. The right side shows a two-chambered structure, second structure with two large vertical apertures and third structure with small holes, respectively.

B) Two µ CT scans show two-rooted tooth with secondary branches, and one-rooted tooth with secondary branches and chambers. The right side shows a bilocular chamber, two sets of three structures each with one or two holes.

C) Two µ CT scans show two-rooted canal and multiple branches grouped together, respectively. The right side shows a bilocular chamber, two sets of

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two structures each with one hole. The sizes of holes differ from one another in each section.

The canals in the mesial root are the mesiobuccal and mesiolingual canals. A middle mesial canal (MMC) sometimes is present in the developmental groove between the other mesial canals,[4,12,76,79,104,107,111] but it may only represent a wide anastomosis between the two mesial canals (Fig. 7.75), or in some cases it may actually be a separate canal from orifice to apex.[89] Troughing into the groove between the mesiobuccal and mesiolingual orifices may be indicated to identify this canal,[9,43,44] if present, or it may just result in blind search within the anastomosis with the risk of a perforation or root weakening due to the commonly found external invagination found both mesially and distally. The presence of two separate distal roots is rare, but this does occur. The mesial canal orifices are usually well separated in the main pulp chamber and connected by a developmental groove.[19]

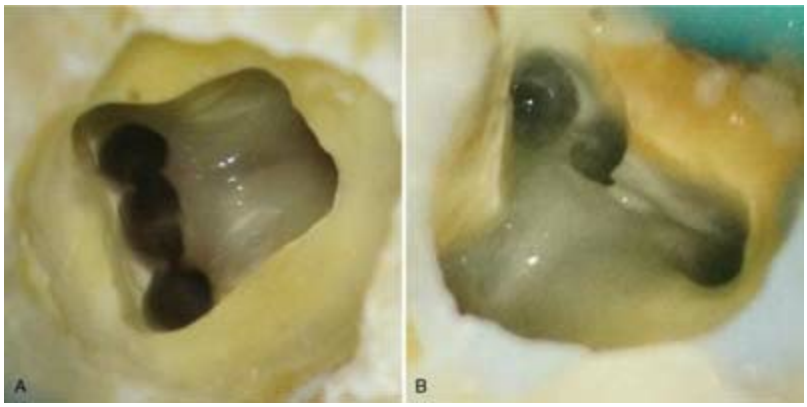

FIG. 7.75 **A,** A photograph (×8) of a mandibular molar showing fins between MB and ML canals**. B,** A photograph (×8) of a mandibular molar showing three mesial canals. *MB,* Mesiobuccal; *ML,* mesiolingual.

A) Close-up view of tooth shows a hollow chamber with three continuous orifices.

B) Close-up view of tooth shows a heart-shaped chamber with three orifices.

Source: (From Nosrat A, Deschenes RJ, Tordik PA, Hicks ML, Fouad AF: Middle mesial canals in mandibular molars: incidence and related factors, *J Endod* 41:28–32, 2015.)

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When only one distal canal is present, the orifice is oval buccolingually and the opening generally is located distal to the buccal groove. This orifice usually can be explored from the mesial with either a DG-16 explorer or a small K-file. If the file tip takes a sharp turn in a distobuccal or distolingual direction, the clinician should search for yet another orifice.

Multiple accessory foramina may be located in the furcation of the mandibular molars.[37] These foramina are usually impossible to clean and shape directly, and therefore, extensive irrigation protocols should be applied.

The traditional access cavity for the mandibular first molar typically is trapezoid or rhomboid, regardless of the number of canals present.[120] When four or more canals are present, the corners of the trapezoid or rhombus should correspond to the positions of the main orifices. A variation in root morphology is the presence of an extra distolingual root.[103] The radix entomolaris (RE) is a supernumerary root located distolingually in mandibular molars (Fig. 7.76),[7,16] whereas the radix paramolaris (RP) is an extra root located mesiobuccally.[102]

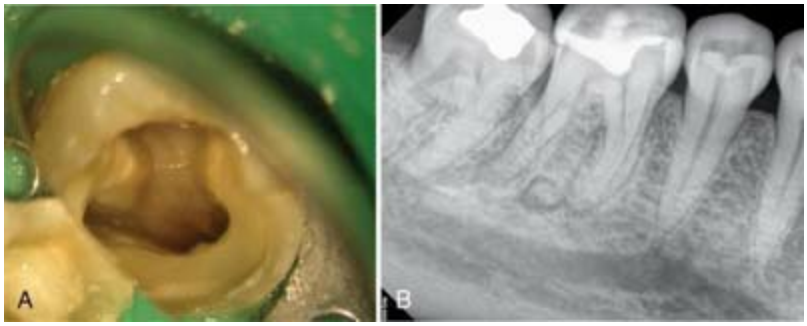

FIG. 7.76 **A,** Radix entomolaris. Notice lingual position of its orifice in relation to the two canals in the distal root. **B,** Radiograph with obvious radix entomolaris on mandibular first molar that curves to the buccal. The reader is referred to a recent publication that details extensively this type of anatomy (see following credit for part **B**). (**A,** Courtesy Dr. William J. Aippersbach, Venice, FL. **B,** From Abella F, Patel S, Durán-Sindreu F, Mercadé M, Roig M: Mandibular first molars with disto-lingual roots: review and clinical management, *Int Endod J* 45:963–978, 2012.)

A) Close-up view of tooth shows a chamber with deep opening.

B) Radiograph shows a bony tissue in the roots toward the tongue.

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Mandibular second molar

The mandibular second molar is somewhat smaller coronally than the first molar and tends to be more symmetric (Fig. 7.77). This tooth is identified by the proximity of its roots and its +4 occlusal groove and cuspal pattern. The two roots often sweep distally in a gradual curve, with the apices close together. In some teeth only one root is present. This tooth may have one, two, three, or four root canals (see Table 7.26 online at the Expert Consult site). The μCT scans for the mandibular second molar can be seen in Fig. 7.78. (See Video 7.18 online at the Expert Consult site for rotational views of these teeth.) The access cavity for a two-canal second molar is rectangular, wide mesiodistally and narrow buccolingually. The access cavity for a single-canal mandibular second molar is oval and is lined up in the center of the occlusal surface.

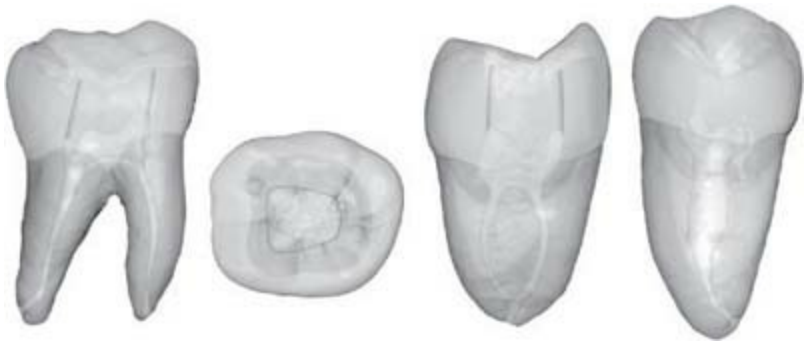

FIG. 7.77 Mandibular second molar. Average time of eruption—11 to 13 years; average age of calcification—14 to 15 years; average length —19.8 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal, mesial, buccal; single root—straight, distal, bayonet, lingual.

Four models of mandibular second molar show two-rooted tooth, occlusal view, interconnected root canals forming a chamber, and curved root canals, respectively.

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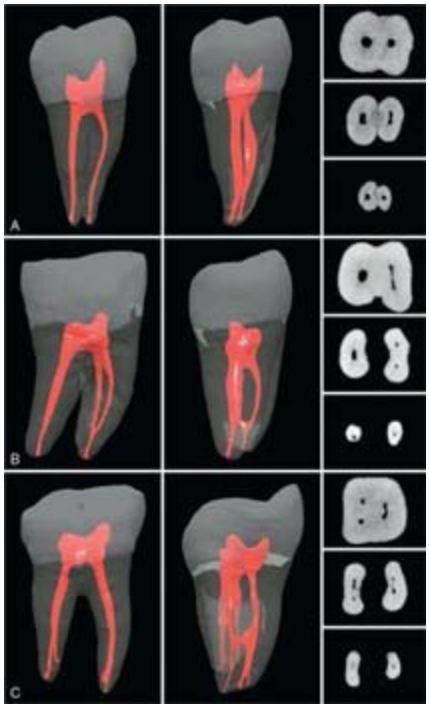

FIG. 7.78 µCT scans of mandibular second molars. **A,** Two-canal second molar with fused roots. **B,** Second molar with three initial canals ending in one canal apically in both roots. **C,** Second molar with four distinct canals. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.18 online at the Expert Consult site for rotational views of these teeth.)

A) µ CT scan shows two root canals in the tooth, which are fused in the second scan. The right side shows a dumb-bell shaped structure with two holes, followed by similar structures with smaller holes.

B) Two µ CT scans show two root canals with an addition branch forming a chamber with the right canal and interconnected root canals with a hollow chamber, respectively. The right side shows a circular structure fused to a vertical structure each with a hole inside, followed by two sets of separated structures, one with a large hole and the other with small holes.

C) Two µ CT scans show two root canals with additional root branches and interconnected root canals, respectively. The right side shows a circular structure with three holes, followed by two sets of two irregular structures each with two holes inside. The second set has smaller holes.

Mandibular third molar

The mandibular third molar is anatomically unpredictable and must be

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evaluated on the basis of its root formation (Fig. 7.79).[52] More often than not it has an occlusal pattern similar to the first molar only much smaller in overall size. Fused short, severely curved, or malformed roots often support well-formed crowns. This tooth may have one to four roots and one to six canals (see Table 7.27 online at the Expert Consult site). The μCT scans for the mandibular third molar can be seen in Fig. 7.80. (See Video 7.19 online at the Expert Consult site for rotational views of these teeth.)

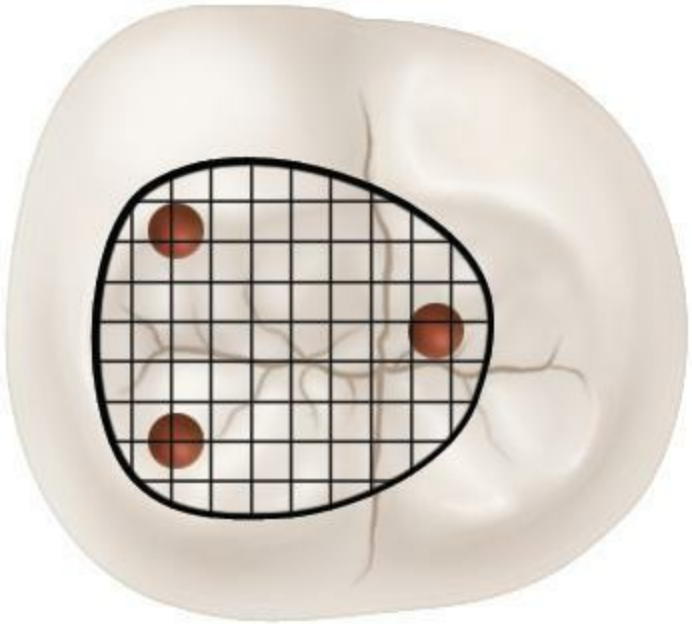

FIG. 7.79 Mandibular third molar. Average time of eruption—17 to 21 years; average age of calcification—18 to 25 years; average length—18.5 mm.

Mandibular third molar shows three holes under an ovoid net-like frame.

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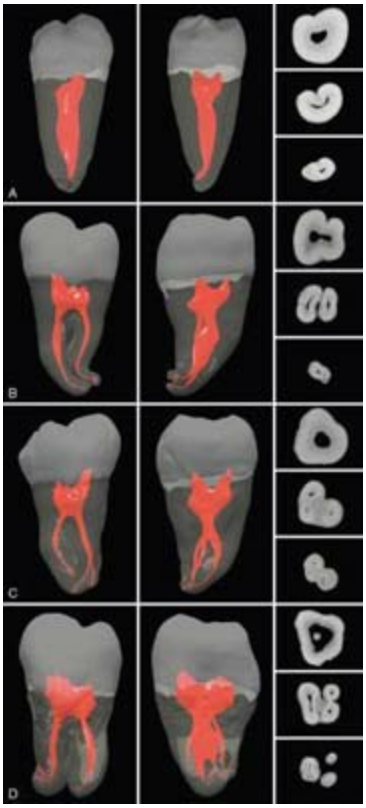

FIG. 7.80 µCT scans of mandibular third molars represent multiple variations for this tooth. **A**, Single C-shaped type canal. **B,** Complex anatomy with significant canal curvatures apically. **C,** Three canals that curve in multiple directions. **D,** Flattened, ribbon-shaped canals with significant apical curvatures. All teeth are shown from both a buccal (vestibular) and a proximal perspective, along with the cross-sectional anatomy at the coronal, middle, and apical levels. (See Video 7.19 online at the Expert Consult site for rotational views of these teeth.)

A) Two µ CT scans show root canal extending from the lower part of crown. The root is diverted in the second scan. The right side shows heart-shaped structure with a hole at the center, a C-shaped aperture in the next structure, and a small hole in the ovoid structure.

B) Two µ CT scans show root canals below the crown. The first scan shows curved root canals at the apex. The second scan shows a diverted fused root canals. The right side shows three structures with a horizontal aperture, two vertical apertures, and a small hole, respectively.

C) Two µ CT scans show two root canals with an additional branch near the apex of one root and curved multiple branches near the apex. The right side shows a structure with a deep hole at the center and two structures with three small holes. The third structure has small holes.

D) Two µ CT scans show two-rooted tooth and one-rooted tooth each with multiple branches. The right side shows a structure with a triangular cavity,

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two sets of three-fused structures each with a hole. The third structure has small holes.

C-shaped canals can also occur (Fig. 7.81).[40] Many of these teeth can be successfully root treated, regardless of anatomic irregularities; however, the long-term prognosis is determined by the root surface volume in contact with bone. The clinician must weigh the benefit of treatment against the prognosis.

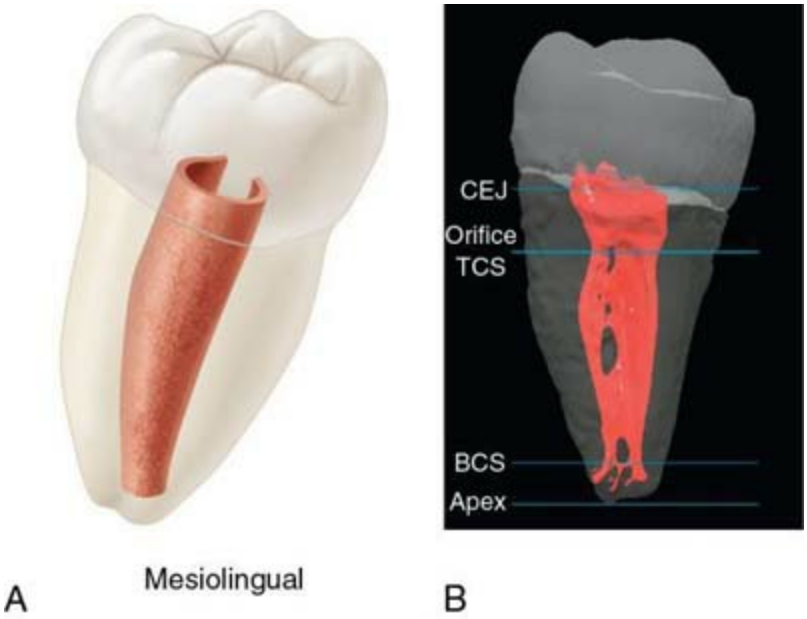

FIG. 7.81 **A,** Diagram of a C-shaped canal anatomy: one continuous canal from pulp chamber floor to apex. **B,** One large canal moving from the top of the C-shaped canal *(TCS)* to the apical-most or bottom of C-shaped canal *(BCS)*, encompassing most of the canal space. *CEJ,* Cementoenamel junction.

A) Mesiolingual view of tooth with a single root canal.

B) μ CT scan of tooth with labels as follows: CE, Orifice TCS, BCS, and Apex.

Teeth with C-shaped root canal systems

The main cause for C-shaped roots and canals is the failure of Hertwig's epithelial root sheath to fuse on either the buccal or lingual root surface (Fig. 7.82).[40] (See Videos 7.20, *A* and *B* online at the Expert Consult site for

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rotational views of these teeth.) The C-shaped canal system can assume many variations in its morphology (Fig. 7.83). Further cross section of types I, II, and III are seen in Fig. 7.83. The original classification[67] has been modified and has produced a more detailed description of C-shaped root and canal morphology. The C-shaped canal configuration can vary along the root depth so that the appearance of the orifices may not be good predictors of the actual canal anatomy.[26,28,70,117,126]

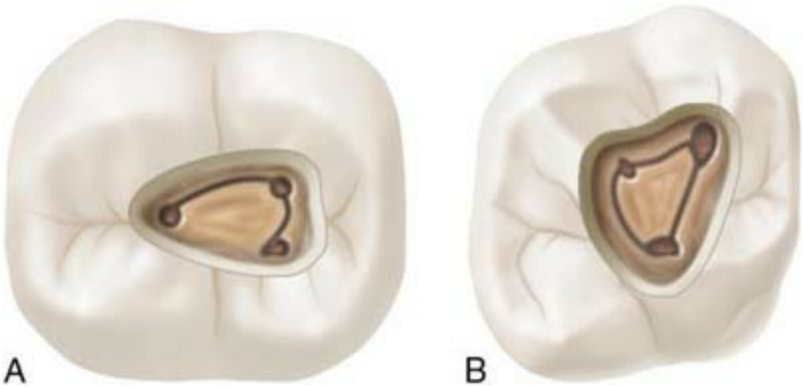

FIG. 7.82 C-shaped canal anatomy. **A,** Mandibular second molar. **B,** Maxillary first molar.

A) Tooth with a triangular cavity that has three holes connected by a string, which forms a U-shape.

B) Tooth with a heart-shaped cavity that has three holes connected by a string.

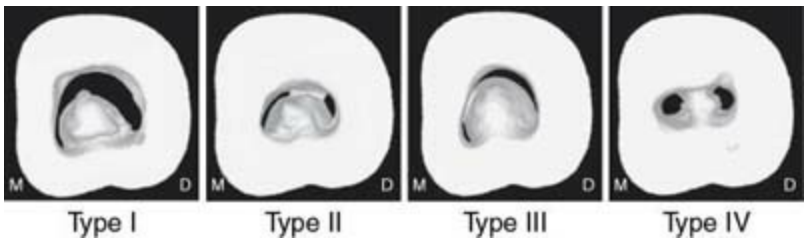

FIG. 7.83 Types of pulpal floor. *M,* Mesial side; *D,* distal side.

Type 1 shows a bell-shaped pulpal floor.

Type 2 shows a C-shaped pulpal floor.

Type 3 shows a rounded mass of pulpal floor.

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Type 4 shows pulpal floor with two holes.

Source: (From Min Y, Fan B, Cheung G, et al: C-shaped canal system in mandibular second molars. III. The morphology of the pulp chamber floor, *J Endod* 32: 1155–1159, 2006.)

Most C-shaped canals occur in the mandibular second molar (Fig. 7.84),[117] but they also have been reported in the mandibular first molar,[118] the maxillary first and second molars, and the mandibular first premolar.[26,27,29,30,75,78,116,121,123,125,126]

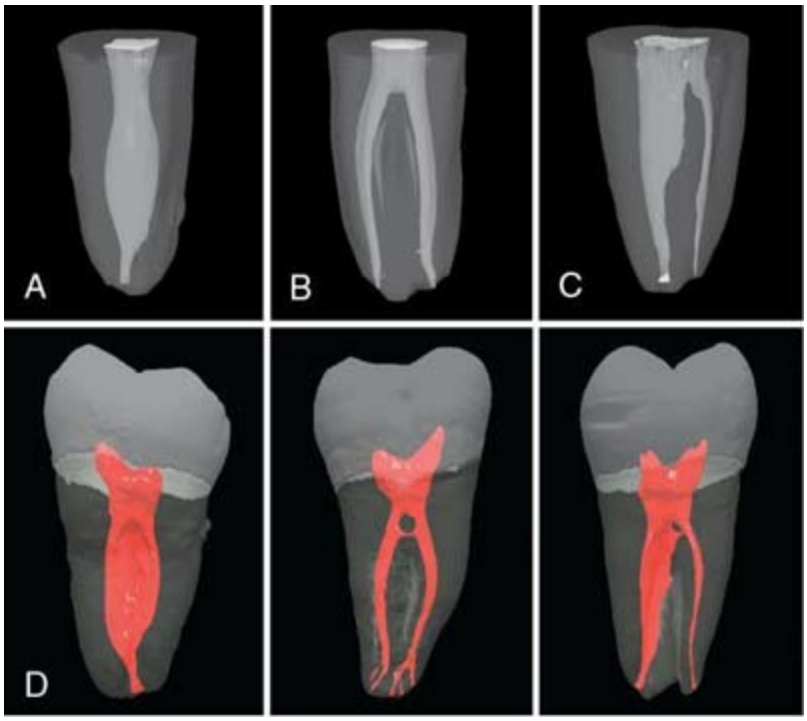

FIG. 7.84 Three-dimensional classification of C-shaped canal configuration. **A,** Merging type. **B,** Symmetrical type. **C,** Asymmetrical type. **D,** Additional C-shaped canal variations.

A) Canal shows a bulge at the center toward the root apex.

B) Canal is bifurcated.

C) Root with bifurcated canal. One canal is comparatively very thin.

D) Three μ CT scans show a depression in root canal, multiple branches at the root apex with a hole in the pulp chamber, and a bifurcated root canal extending near the edge of crown, in which the second canal is very thin.

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Source: (From Gao Y, Fan B, Cheung G, et al: C-shaped canal system in mandibular second molars. IV. Morphological analysis and transverse measurement, *J Endod* 32:1062–1065, 2006.)

The access cavity for teeth with a C-shaped root canal system varies considerably and depends on the pulp morphology of the specific tooth. Teeth with C-shaped anatomy pose a considerable technical challenge; therefore, use of the DOM during all treatment phases is recommended.[15]

All tables in this section that provide literature documentation for the studies on root canal anatomy are available online at the Expert Consult site and can be accessed accordingly.

The 3D reconstructed images in this chapter were obtained from the tooth and canal morphology database at the School of Stomatology, Wuhan University, China. The database was established by Dr. Bing Fan's group and supported by the National Natural Science Foundation of China (grant no. 30572042, 30872881, 81070821) and the Key Technologies R&D Programme of Hubei Province of China (grant no. 2007AA302B06). The microcomputed tomography machine used for scanning was the μCT-50, Scanco Medical, Bassersdorf, Switzerland. The multiple software used for 3D reconstruction included 3D-Doctor (Able Software Corp., Lexington, MA) and VGStudio MAX (Volume Graphics GmbH, Heidelberg, Germany).

Access cavity illustrations: Designed and formatted by Dr. Richard Burns, San Mateo, CA; and Dr. Eric Herbranson, San Leandro, CA.

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References

1. Ahmed HM, Dummer PM. *A new system for classifying tooth, root and canal anomalies*, Int Endod J 2018;51: 389.
2. Ahmed HM, Neelakantan P, Dummer PM, et al: *A new system for classifying accessory canal morphology*, Int Endod J 2018;51: 164.
3. Ahmed HM, Versiani MA, De-Deus G, et al: *A new system for classifying root and root canal morphology*, Int Endod J 2016;50: 761.
4. Akbarzadeh N, Aminoshariae A, Khalighinejad N, et al: *The association between the anatomic landmarks of the pulp chamber floor and the prevalence of middle mesial canals in mandibular first molars: An in vivo analysis*, J Endod 2017;43: 1797.
5. Allen C, Meyer CA, Yoo E, et al: *Stress distribution in a tooth treated through minimally invasive access compared to one treated through traditional access: a finite element analysis study*, J Conserv Dent 2018;21: 505.
6. Alovisi M, Pasqualini D, Musso E, et al: *Influence of contracted endodontic access on root canal geometry: an in vitro study*, J Endod 2018;44: 614.
7. Attam K, Nawal RR, Utneja S, et al: *Radix entomolaris in mandibular first molars in Indian population: a review and case reports*, Case Rep Dent 2012;1–7: article ID 595494.
8. Auswin MK, Ramesh S. *Truss access new conservative approach on access opening of a lower molar: A case report*, J Adv Pharm Edu Res 2017;7: 344.
9. Azim AA, Deutsch AS, Solomon CS. *Prevalence of*

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middle mesial canals in mandibular molars after guided troughing under high magnification: an in vivo investigation, J Endod 2015;41: 164.

10. Baisden MK, Kulilid JC, Weller RN. *Root canal configuration of the mandibular first premolar*, J Endod 1992;18: 505.
11. Baldassari-Cruz LA, Lilly JP, Rivera EM. *The influence of dental operating microscopes in locating the mesiolingual canal orifice*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 190.
12. Bansal R, Hedge S, Astekar M. *Morphology and prevalence of middle canals in the mandibular molars: a systematic review*, J Oral Maxillofac Pathol 2018;22: 216.
13. Buhrley LJ, Barrows MJ, BeGole EA, et al: *Effect of magnification on locating the MB-2 canal in maxillary molars*, J Endod 2002;28: 324.
14. Burch JG, Hulen S. *The relationship of the apical foramen to the anatomic apex of the tooth root*, Oral Surg Oral Med Oral Pathol 1972;34: 262.
15. Bürklein S, Schäfer E. *Minimally invasive endodontics*, Quintessence Int 2015;46: 119.
16. Calberson FL, DeMoor RJ, Deroose CA. *The radix entomolaris and paramolaris: a clinical approach in endodontics*, J Endod 2007;33: 58.
17. Calişkan MK, Pehlivan Y, Sepetçioğlu F, et al: *Root canal morphology of human permanent teeth in a Turkish population*, J Endod 1995;21: 200.
18. Cheung GS, Wei WL, McGrath C. *Agreement between periapical radiographs and cone-beam computed tomography for assessment of periapical status of root filled molar teeth*, Int Endod J 2013;46: 889.
19. Cimilli H, Mumcu G, Cimilli T, et al: *Correlation*

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between root canal patterns, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: e16.

20. Clark D, Khademi J. *Modern molar endodontic access and directed dentin conservation*, Dent Clin North Am 2010;54: 249.
21. Clark D, Khademi J, Herbranson E. *Fracture resistant endodontic and restorative preparations*, Dent Today 2013;32: 118-120.
22. Clark D, Khademi J, Herbranson E. *The new science of strong endo teeth*, Dent Today 2013;32: 112-114, 116.
23. Connert T, Zehnder MA, Amato M, et al: *Microguided endodontics: a method to achieve minimally invasive access cavity preparation and root canal location in mandibular incisors using a novel computer-guided technique*, Int Endod J 2018;51: 247.
24. Cutright DE, Bhaskar SN. *Pulpal vasculature as demonstrated by a new method*, Oral Surg Oral Med Oral Pathol 1969;27: 678.
25. Degerness RA, Bowles WR. *Dimension, anatomy and morphology of the mesiobuccal root canal system in maxillary molars*, J Endod 2010;36: 985.
26. Fan B, Cheung GSP, Fan M, et al: *C-shaped canal system in mandibular second molars. I. Anatomical fractures*, J Endod 2004;30: 899.
27. Fan B, Cheung GSP, Fan M, et al: *C-shaped canal system in mandibular second molars. II. Radiographic features*, J Endod 2004;30: 904.
28. Fan B, Pan Y, Gao Y, et al: *Three-dimensional morphologic analysis of isthmuses in the mesial roots of mandibular molars*, J Endod 2010;36: 1866.
29. Fan B, Yang J, Gutmann JL, et al: *Root canal systems in mandibular first premolars with C-shaped root*

Telegram: @dental_k

www.konkur.in

configurations. I. Microcomputed tomography mapping of the radicular groove and associated root canal cross sections, J Endod 2008;34: 1337.

30. Fan B, Ye WH, Xie EZ, et al: *Three-dimensional morphological analysis of C-shaped canals in mandibular first premolars in a Chinese population*, Int Endod J 2012;45: 1035.
31. Fasoli G, Arlotta A. *Su ll' anatomia del canali radicolari del denti umani*, L Stomatol 1913;11:.
32. Ferreira CM, de Moraes IG, Bernardineli N. *Three-rooted maxillary second premolar*, J Endod 2000;26: 105.
33. Fonseca Tavares WL, Diniz Viana AC, de Carvalho Machado V, et al: *Guided endodontic access of calcified anterior teeth*, J Endod 2018;44: 1195.
34. Görduysus MO, Görduysus M, Friedman S. *Operating microscope improves negotiation of second mesiobuccal canals in maxillary molars*, J Endod 2001;27: 683.
35. Green D. *Double canals in single roots*, Oral Surg Oral Med Oral Pathol 1973;35: 689.
36. Gutierrez JH, Aguayo P. *Apical foraminal openings in human teeth: number and location*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 769.
37. Gutmann JL. *Prevalence, location, and patency of accessory canals in the furcation region of permanent molars*, J Periodontol 1978;49: 21.
38. Gutmann JL. *Apical termination of root canal procedures - ambiguity or disambiguation?*, Evid-Based Endod 2016;1: 4:1- DOI: 10.1186/s41121-016-0004-8.
39. Gutmann JL, Lovdahl PE. Problem solving in endodontics ed 5 2011; Elsevier St Louis.
40. Gutmann JL. *C-Shaped root canal system*, Versiani MA Basrani B Sousa-Neto MD The root canal anatomy in

Telegram: @dental_k

www.konkur.in

permanent dentition, Chapter 9 2019; Springer International Publishing AG Cham, Switzerland 255.
41. Jiang Q, Huang Y, Tu X, et al: *Biomechanical properties of first maxillary molars with different endodontic cavities: a finite element analysis*, J Endod 2018;44: 1283.
42. Joseph I, Varma BR, Bhat KM. *Clinical significance of furcation anatomy of the maxillary first premolar: a biometric study on extracted teeth*, J Periodontol 1996;67: 386.
43. Keles A, Keskin C. *Detectability of middle mesial root canal orifices by troughing technique in mandibular molars: a microcomputed tomographic study*, J Endod 2017;43: 1329.
44. Keles A, Keskin C. *Deviations of mesial root canals of mandibular first molar teeth at the apical third: a micro-computed tomographic study*, J Endod 2018;44: 1030.
45. Kerekes K, Tronstad L. *Long-term results of endodontic treatment performed with a standardized technique*, J Endod 1979;5: 83.
46. Kerekes K, Tronstad L. *Morphometric observations in root canals of human premolars*, J Endod 1997;3: 74.
47. Khademi JA. *Restoratively driven, minimally invasive endodontics*, Dent Today 2019;38: 66-68.
48. Kim SM, Amponsah EK. *Impacted third molar transplantation on the malpracticed extraction socket*, Ghana Med J 2017;51: 200.
49. Krasner P, Rankow HJ. *Anatomy of the pulp chamber floor*, J Endod 2004;30: 5.
50. Krishan R, Paqué F, Ossareh A, et al: *Impacts of conservative endodontic cavity on root canal instrumentation efficacy and resistance to fracture assessed in incisors, premolars, and molars*, J Endod

Telegram: @dental_k

www.konkur.in

2014;40: 1160.
51. Kuttler Y. *Microscopic investigation of root apexes*, J Am Dent Assoc 1955;50: 544.
52. Kuzekanani M, Haghani J, Nosrati H. *Root and canal morphology of mandibular third molars in an Iranian population*, J Dent Res Dent Clin Dent Prospects 2012;6: 85.
53. Lammertyn PA, Rodfigo SB, Brunotto M, et al: *Furcation groove of maxillary first premolar, thickness, and dentin structures*, J Endod 2009;35: 814.
54. Langeland K. Tissue changes in the dental pulp: an experimental histologic study 1957; Oslo University Press Oslo.
55. Langeland K. *The histopathologic basis in endodontic treatment*, Dent Clin North Am 1967;11: 49.
56. Langeland K. *Tissue response to dental caries*, Dent Traumatol 1987;3: 149.
57. Langeland K. *Reacción tisular a los materiales de obturación del conducto*, Guldener PHA Langeland K 1995; Springer-Verlag Ibérica Endodoncia, Barcelona.
58. Lara-Mendes STO, Barbosa CFM, Machado VC, et al: *A new approach for minimally invasive access to severely calcified anterior teeth using the guided endodontics technique*, J Endod 2018;44: 1578.
59. Laramendes ST, Barbosa CD, Santarosa CC, et al: *Guided endodontic access in maxillary molars using cone-beam computed tomography and computer-aided design/computer-aided manufacturing system: A case report*, J Endod 2018;44: 875.
60. Leeb IJ. *Canal orifice enlargement as related to biomechanical preparation*, J Endod 1983;9: 463.
61. Li J, Li L, Pan Y. *Anatomic study of the buccal root with*

Telegram: @dental_k

www.konkur.in

furcation groove and associated root canal shape in maxillary first premolars by using microcomputed tomography, J Endod 2013;9: 265.

62. Loh HS. *Root morphology of the maxillary first premolar in Singaporeans*, Aust Endod J 1998;43: 399.
63. Lui N, Li X, Liu N, et al: *A micro-computed tomography study of the root canal morphology of the mandibular first premolar in a population from southwestern China*, Clin Oral Investig 2013;17: 999.
64. Manning SA. *Root canal anatomy of mandibular second molars. I*, Int Endod J 1990;23: 34.
65. Manning SA. *Root canal anatomy of mandibular second molars. II. C-shaped canals*, Int Endod J 1990;23: 40.
66. Marchesan MA, Lloyd A, Clement DJ, et al: *Impacts of contracted endodontic cavities on primary root canal curvature parameters in mandibular molars*, J Endod 2018;44: 1558.
67. Martinez-Berná A, Badanelli P. *Mandibular first molars with six root canals*, J Endod 1985;11: 348.
68. Martins JNR, Francisco H, Ordinola-Zapata R. *Prevalence of C-shaped configurations in the mandibular first and second premolars: A cone-beam computed tomographic, in vivo study*, J Endod 2017;43: 890.
69. Mauger MJ, Waite RM, Alexander JB, et al: *Ideal endodontic access in mandibular incisors*, J Endod 1999;25: 206.
70. Min Y, Fan B, Cheung GSP, et al: *C-shaped canal system in mandibular second molars. III. The morphology of the pulp chamber floor*, J Endod 2006;32: 1155.
71. Mjör IA, Nordahl I. *The density and branching of dentinal tubules in human teeth*, Arch Oral Biol 1996;41: 401.
72. Mjör IA, Smith MR, Ferrari M, et al: *The structure of*

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www.konkur.in

dentin in the apical region of human teeth, Int Endod J 2001;34: 346.

73. Monnan G, Smallwood ER, Gulabivala K. *Effects of access cavity location and design on degree and distribution of instrumented root canal surface in maxillary anterior teeth*, Int Endod J 2001;34: 176.
74. Moore B, Verdelis K, Kishen A, et al: *Impacts of contracted endodontic cavities on instrumentation efficacy and biomechanical responses in maxillary molars*, J Endod 2016;42: 1779.
75. Nallapati S. *Three canal mandibular first and second premolars: a treatment approach—a case report*, J Endod 2005;31: 474.
76. Navarro LF, Luzi A, Garcia AA, et al: *Third canal in the mesial root of permanent mandibular first molars: review of the literature and presentation of 3 clinical reports and 2 in vitro studies*, Med Oral Patol Oral Cir Bucal 2007;12: E605.
77. Neelakantan P, Khan K, Pak Hei G, et al: *Does the orifice-directed dentin conservation access design debride pulp chamber and mesial root canal systems of mandibular molars similar to a traditional access design?*, J Endod 2018;44: 274.
78. Newton CW, McDonald S. *A C-shaped canal configuration in a maxillary first molar*, J Endod 1984;10: 397.
79. Nosrat A, Deschenes RJ, Tordik PA, et al: *Middle mesial canals in mandibular molars: incidence and related factors*, J Endod 2015;41: 28.
80. Ordinola-Zapata R, Monteiro Bramante C, Gagliardi Minotti P, et al: *Micro-CT evaluation of C-shaped mandibular first premolars in a Brazilian subpopulation*,

Telegram: @dental_k

www.konkur.in

Int Endod J 2015;48: 807.
81. Özcan E, olak H, Hamid M. *Root and canal morphology of maxillary first premolars in a Turkish population*, J Dent Sci 2012;7: 390.
82. Park J-B, Kim N, Park S, et al: *Evaluation of root anatomy of permanent mandibular premolars and molars in a Korean population with cone-beam computed tomography*, Eur J Dent 2013;7: 94.
83. Patel S, Wilson R, Dawood A, et al: *The detection of periapical pathosis using digital periapical radiography and cone beam computed tomography. Part 2. A 1-year post-treatment follow-up*, Int Endod J 2012;45: 711.
84. Peters OA, Laib A, Gohring TN, et al: *Changes in root canal geometry after preparation assessed by high-resolution computed tomography*, J Endod 2001;27: 1.
85. Peters OA, Laib A, Rüegsegger P, et al: *Three-dimensional analysis of root canal geometry by high resolution computed tomography*, J Dent Res 2000;79: 1405.
86. Peters OA, Peters CI, Schonenberger K, et al: *ProTaper rotary root canal preparation: assessment of torque force in relation to canal anatomy*, Int Endod J 2003;36: 93.
87. Pineda F, Kuttler Y. *Mesiodistal and buccolingual roentgenographic investigation of 7275 root canals*, Oral Surg Oral Med Oral Pathol 1972;33: 101.
88. Ponce EH, Vilar Fernandez JA. *The cemento-dentino-canal junction, the apical foramen, and the apical constriction: evaluation by optical microscopy*, J Endod 2003;29: 214.
89. Ricucci D. *Three independent canals in the mesial root of a mandibular first molar*, Endod Dent Traumatol 1997;13: 47.

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www.konkur.in

90. Ricucci D, Langeland K. *Apical limit of root canal instrumentation and obturation. Part 2. A histologic study*, Int Endod J 1998;31: 394.
91. Rover G, Belladonna FG, Bortoluzzi EA, et al: *Influence of access cavity design on root canal detection, instrumentation efficacy, and fracture resistance assessed in maxillary molars*, J Endod 2017;43: 165.
92. Rubinstein R, Kim S. *Short-term observation of the results of endodontic surgery with the use of a surgical operation microscope and super-EBA as root-end filling material*, J Endod 1999;25: 43.
93. Saad AY, Al-Yahya AS. *The location of the cementodentinal junction in single-rooted mandibular first premolars from Egyptian and Saudi patients: a histologic study*, Int Endod J 2003;36: 541.
94. Saygili G, Uysal B, Omar B, et al: *Evaluation of relationship between endodontic access cavity types and secondary mesiobuccal canal detection*, BMC Oral Health 2018;18: 121.
95. Schilder H. *Filling root canals in three dimensions*, Dent Clin North Am 1967;11: 723.
96. Schwarze T, Baethge C, Stecher T, et al: *Identification of second canals in the mesiobuccal root of maxillary first and second molars using magnifying loupes or an operating microscope*, Aust Endod J 2002;28: 57.
97. Silva EJNL, Rover G, Belladonna FG, et al: *Impact of contracted endodontic cavities on fracture resistance of endodontically treated teeth: a systematic review of in vitro studies*, Clin Oral Investig 2018;22: 109.
98. Simon JHS. *The apex: how critical is it?*, Gen Dent 1994;42: 330.
99. Sjögren U, Hägglund B, Sundqvist G, et al: *Factors*

Telegram: @dental_k

www.konkur.in

affecting the long-term results of endodontic treatment, J Endod 1990;16: 498.

100. Smith AJ. *Dentin formation and repair*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Chicago.
101. Smulson MH, Hagen JC, Ellenz SJ. *Pulpoperiapical pathology and immunologic considerations*, Weine FS Endodontic therapy ed 5 1996; Mosby St Louis.
102. Souza-Flamini LE, Leoni GB, Chaves JF, et al: *The radix entomolaris and paramolaris: a micro computed tomographic study of 3-rooted mandibular first molars*, J Endod 2014;40: 1616.
103. Sperber GH, Moreau JL. *Study of the number of roots and canals in Senegalese first permanent mandibular molars*, Int Endod J 1998;31: 117.
104. Srivastava S, Alrogaibah NA, Aliarbou G. *cone-beam computed tomographic analysis of middle mesial canals and isthmus in mesial roots of mandibular first molars – prevalence and related factors*, J Conserv Dent 2018;21: 526.
105. Stropko JJ. *Canal morphology of maxillary molars: clinical observations of canal configurations*, J Endod 1990;25: 446.
106. Subay RK, Kayatas M. *Dens invaginatus in an immature lateral incisor: a case report of complex endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: e37.
107. Tahmasbi M, Jalali P, Nair MK, et al: *Prevalence of middle mesial canals and isthmi in the mesial root of mandibular molars: an in vivo cone-beam computed tomographic study*, J Endod 2017;43: 1080.
108. Tamse A, Katz A, Pilo R. *Furcation groove of buccal*

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www.konkur.in

root of maxillary first premolars: a morphometric study, J Endod 2000;26: 359.

109. Taylor GN. *Techniiche per la preparazione e l'otturazione intracanalare*, La Clinica Odontoiatrica del Nord America 1988;20: 566.
110. Versiani MA Basrani B Sousa-Neto MD The root canal anatomy in permanent dentition 2019; Springer International Publishing AG Cham, Switzerland.
111. Versiani MA, Ordinola-Zapata R, Keles A, et al: *Middle mesial canals in mandibular first molars: a micro CT study in different populations*, Arch Oral Biol 2016;61: 130.
112. Versiani MA, Pécora JD, Sousa-Neto MD. *The anatomy of two-rooted mandibular canines determined using micro-computed tomography*, Int Endod J 2011;44: 682.
113. Vertucci FJ. *Root canal anatomy of the human permanent teeth*, Oral Surg Oral Med Oral Pathol 1984;58: 589.
114. Vertucci FJ, Williams RG. *Furcation canals in the human mandibular first molar*, Oral Surg Oral Med Oral Pathol 1974;38: 308.
115. Von der Lehr WN, Marsh RA. *A radiographic study of the point of endodontic egress*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1973;35: 705.
116. Von ZM, Jnr M, Berti L, et al: *Worldwide prevalence of mandibular second molar C-shaped morphologies evaluated by cone-beam computed tomography*, J Endod 2017;43: 1442.
117. Wang Y, Guo J, Yang HB, et al: *Incidence of C-shaped root canal systems in mandibular second molars in the native Chinese population by analysis of clinical methods*, Int J Oral Sci 2012;4: 161.
118. Weine FS, Members of the Arizona Endodontic

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Association. *The C-shaped mandibular second molar: incidence and other considerations*, J Endod 1998;24: 372.

119. Weine FS, Pasiewicz RA, Rice RT. *Canal configuration of the mandibular second molar using a clinically oriented in vitro method*, J Endod 1988;14: 207.
120. Wilcox LR, Walton RE, Case WB. *Molar access: shape and outline according to orifice location*, J Endod 1989;15: 315.
121. Xuan Y, Bin G, Ke-Zeng L, et al: *Cone-beam computed tomography study of root and canal morphology of mandibular premolars in a western Chinese population*, BMC Med Imag 2012;12: 18.
122. Xu T, Tay FR, Gutmann JL, et al: *Micro–Computed tomography assessment of apical accessory canal morphologies*, J Endod 2016;42: 798.
123. Yang H, Tian C, Li G, et al: *A cone-beam computed tomography study of the root canal morphology of mandibular first premolars and the location of root canal orifices and apical foramina in a Chinese subpopulation*, J Endod 2013;39: 435.
124. Yuan K, Niu C, Xie Q, et al: *Comparative evaluation of the impact of minimally invasive preparation vs. conventional straight-line preparation on tooth biomechanics: a finite element analysis*, Eur J Oral Sci 2016;124: 591.
125. Zhang R, Wang H, Tian YY, et al: *Use of cone-beam computed tomography to evaluate root and canal morphology of mandibular molars in Chinese individuals*, Int Endod J 2011;44: 990.
126. Zheng Q, Zhang L, Zhou X, et al: *C-shaped root canal system in mandibular second molars in a Chinese population evaluated by cone-beam computed*

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tomography, Int Endod J 2011;44: 857.

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8: Cleaning and shaping of the root canal system

Ove A. Peters, Christine I. Peters, Bettina Basrani

CHAPTER OUTLINE

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Introduction

Clinical endodontics encompasses a number of treatments that share the common goal of preventing and treating microbial contamination of pulps and root canal systems. Treatment of traumatic dental injuries and vital pulp treatment are fundamentally different from pulpectomies and root canal instrumentation of teeth with infected pulps (see Chapter 1 for more details).

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Endodontic therapy is directed toward one specific set of aims: to cure or prevent periradicular periodontitis.[309,459] The ultimate goal is to aid in the long-term retention in patients' natural teeth in function and esthetics.

To date, available pertinent clinical trials[89,317,326,341] and in vitro studies suggest that certain practices in canal preparation and disinfection are more appropriate than others. This chapter provides a summary of this information.

Orthograde root canal treatment is a predictable and usually highly successful procedure, both in relatively straightforward (Fig. 8.1) and more complex cases (Fig. 8.2). In recent studies and reviews, favorable outcome rates of up to 95% were reported for the treatment of teeth diagnosed with irreversible pulpitis;[37,93,139] favorable outcome rates of up to 85% were reported for teeth with infected, necrotic pulps.[92,140,301,326,358]

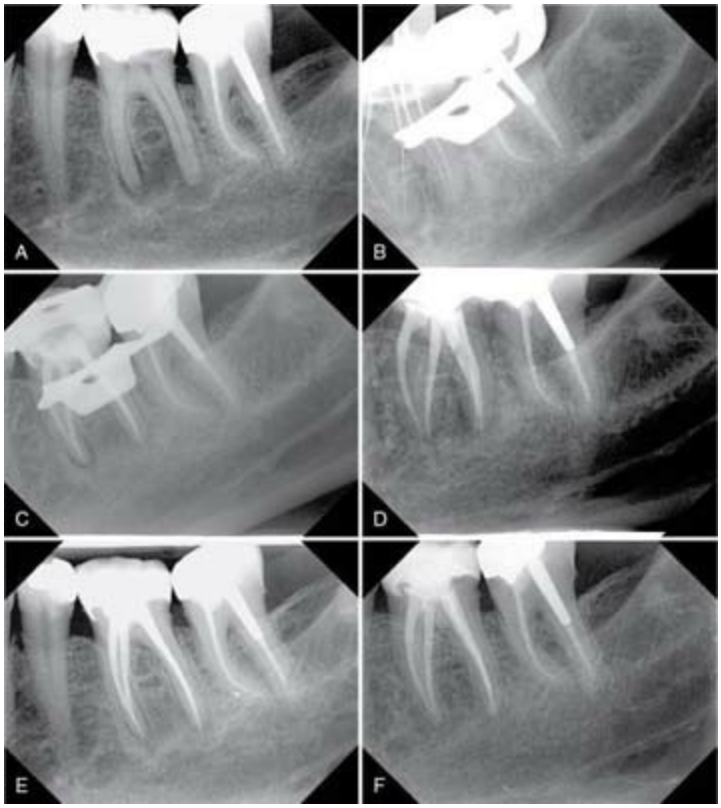

FIG. 8.1 Effect of routine root canal treatment of a mandibular molar. **A,** Pretreatment radiograph of tooth #19 shows radiolucent lesions adjacent to both mesial and distal root apices. **B,** Working length radiograph shows two separate root canals in the mesial root and two merging canals in the distal root. **C,** Posttreatment radiograph after shaping of root canal systems with nickel-titanium rotary files and

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obturation with thermoplasticized gutta-percha. **D,** Six-month recall radiograph after restoration of tooth #19 with an adhesively inserted full ceramic crown; some periradicular bone fill can be seen. **E,** One-year recall radiograph displays evidence of additional periradicular healing. **F,** Five-year recall radiograph; tooth not only is periapically sound but also clinically asymptomatic and fully functional.

Set of six radiographs depict pre- and post-treatment results of routine root canal treatment of a mandibular molar.

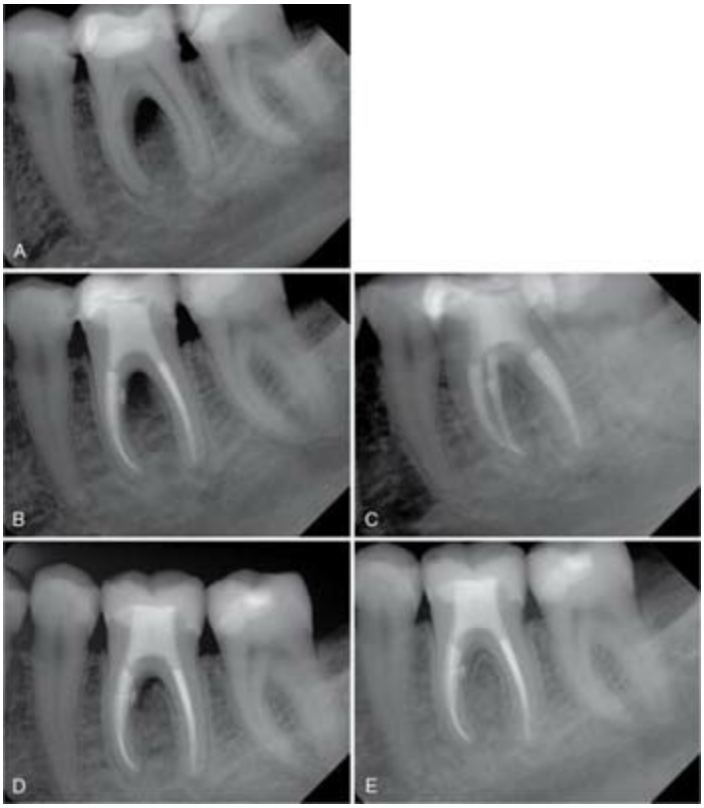

FIG. 8.2 Root canal treatment in a case of apical and interradicular pathosis. **A,** Pretreatment radiograph of tooth #19 shows an interradicular lesion. **B–C,** Posttreatment radiographs after root canal preparation and obturation. Note the lateral canal in the coronal third of the root canal. **D–E,** Two-month recall radiograph suggests rapid healing.

A) Radiograph of tooth shows a radiolucent crown with a white radiopaque smudge visible on the left crown.

B) Radiograph of tooth shows a radiolucent crown and neck with a white radiopaque canals. An outline of white radiopaque large smudge visible on the left crown.

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C) Radiograph of tooth shows a radiolucent crown with a white radiopaque smudge on the left. Radiolucency is visible in neck, vertically segmented left canal and right canal.

D) Radiograph of tooth shows a radiolucent crown with radiopaque neck and canals.

E) Radiograph of tooth shows a radiolucent crown with radiopaque neck and canals.

Source: (Courtesy Dr. H. Walsch.)

The ultimate cause of pulpal pathosis are microorganisms that can breach dental hard-tissue barriers through several avenues—the most common being dental caries (Fig. 8.3). Shaping and cleaning procedures (Box 8.1) as part of root canal treatment are directed against microbial challenges to the root canal system. However, proper disinfection per se does not guarantee long-term retention of root canal–treated teeth; there is good evidence that this outcome is closely related to placement of an adequate coronal restoration.[20,300,352,369] Moreover, the impact of preservation of radicular structural strength on tooth longevity has been emphasized.[154]

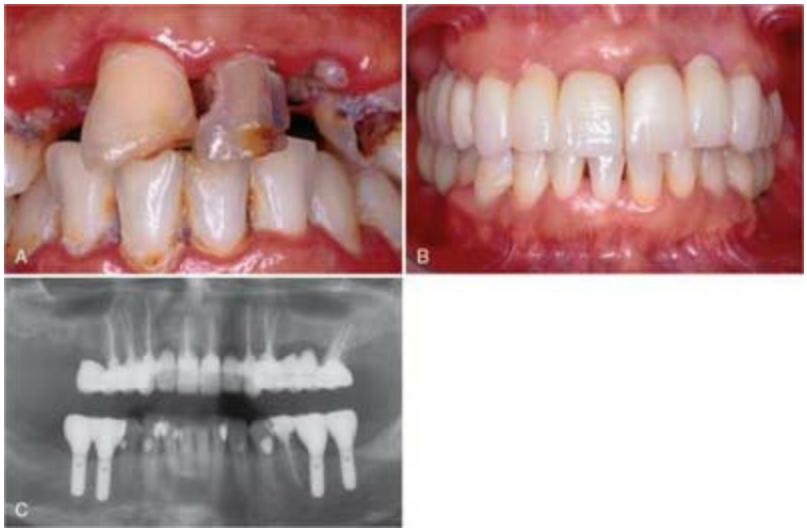

FIG. 8.3 Root canal therapy as part of a comprehensive treatment plan. The patient, who was recovering from intravenous drug addiction, requested restorative dental treatment. Because of extensive decay, several teeth had to be extracted, and nine teeth were treated endodontically. Root canal treatment was aided by nickel-titanium rotary instruments, and obturation was done with lateral compaction of

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gutta-percha and AH26 as the sealer. Microsurgical retrograde therapy was performed on tooth #8, and the distobuccal root of #14 had to be resected. Metal-free adhesively luted restorations were placed, and missing mandibular teeth were replaced by implants. **A,** Pretreatment intraoral status, showing oral neglect. **B,** Posttreatment intraoral status at 4-year follow-up, showing fully functional, metal-free, tooth-colored reconstructions. **C,** Panoramic radiograph at 4-year recall shows sound periradicular tissues in relation to endodontically treated teeth.

A) Close-up of frontal upper and lower jaw exposes extensively decayed central and lateral incisors.

B) Close-up of frontal upper and lower jaw exposes central, lateral incisors, canines and molars.

C) Radiograph of frontal upper and lower jaw shows radiopaque lower molars with extended radiopaque spirally textured posts for root treatment visible in the periradicular tissues.

Source: (Restorations done by Dr. Till N. Göhring.)

Box 8.1

Basic Objectives in Cleaning and Shaping

The primary objectives in cleaning and shaping the root canal system are to:

- Remove infected soft and hard tissue
- Give disinfecting irrigants access to the apical canal space
- Create space for the delivery of medicaments and subsequent obturation
- Retain the integrity of radicular structures

Principles of cleaning and shaping

Endodontists agree that a major biologic aim of root canal therapy is to address apical periodontitis by disinfection and subsequent sealing of root canal systems. However, considerable disagreement exists over how this goal should be achieved. Although the terms “cleaning and shaping” are commonly used to describe root canal treatment procedures, reversing the order to “shaping and cleaning” may more correctly reflect the fact that enlarged canals direct and facilitate the cleaning action of irrigants.

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Microorganisms in the pulp cavity and coronal root canal may be readily killed by irrigants early in a procedure, but bacteria and their toxins in less accessible canal areas can survive and elicit or maintain apical periodontitis. In current everyday practice, these persisting bacteria can be targeted only after root canal preparation.

Mechanical objective

An ideal mechanical objective of root canal instrumentation is complete and centered incorporation of the original canals into the prepared shape, meaning that all root canal surfaces are mechanically prepared (*green areas* in Fig. 8.4, *A* and *B*); however, this goal is unlikely to be met with current techniques.[312,340]

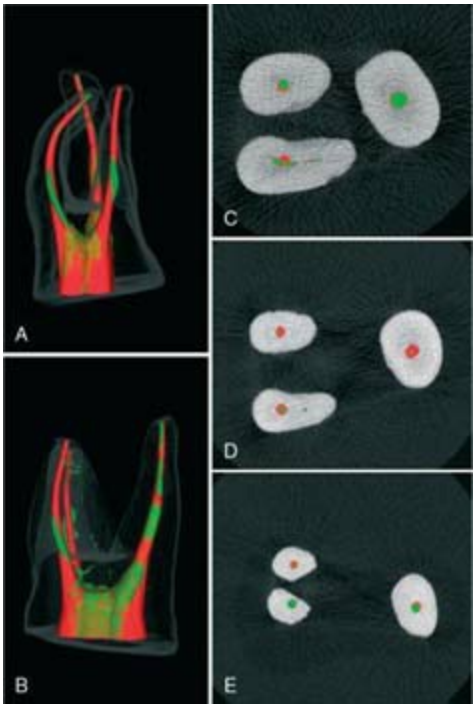

FIG. 8.4 Example of a desired shape, with the original root canal fully incorporated into the prepared outline. **A–B,** Micro–computed tomography reconstructions in clinical and mesiodistal views of a maxillary molar prepared with a NiTi rotary system. The *green area* indicates the pretreatment shape, and the *red area* indicates the posttreatment shape. Areas of *mixed red and green* indicate no change (i.e., no removal of radicular dentin). **C–E**, Cross sections of the coronal, middle, and apical thirds; the pretreatment cross sections *(green)* are encircled by the posttreatment outlines *(red)* in most areas.

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A) Scan shows a red tooth neck, root canals with green pigments towards the left and central canal. Green vertical curve is visible over the left and right canal and on the outlines of central canal.

B) Scan shows a red tooth neck, root canals with green pigments towards the right and central canal. Green vertical curves are visible over the left and right canal and around central canal.

C) Cross-section of coronal canal shows scattered red dots in the center and a streak of green over it; Cross-section of middle canal shows green dot in the center and an outline of red; and Cross-section of apical canal shows a green dot in the center and an outline of red on one side.

D) Cross-section of coronal canal shows a red dot in the center and green dot inside it with a green dot on the side; Cross-section of middle canal shows red dot in the center and green dot inside it; and Cross-section of apical canal shows red dot in the center and green dot inside it.

E) Cross-section of coronal canal shows a green dot with an outline of red on one side; Cross-section of middle canal shows green dot in the center with an outline of red on one side; and Cross-section of apical canal shows red dot in the center and green dot inside it.

Source: (***A–B***, From Hübscher W, Barbakow F, Peters OA: Root-canal preparation with FlexMaster: canal shapes analysed by micro-computed tomography, *Int Endod J* 36:740–747, 2003.)

Preparation errors such as deviations, zipping, and perforations should be absent. Although these negative effects of canal shaping and other procedural mishaps (see later) may not directly affect the probability of a favorable outcome,[254] they leave parts of the root canal system inaccessible for disinfection and are undesirable for that reason alone.

Another important mechanical objective is to retain as much cervical and radicular dentin as possible so as not to weaken the root structure, thereby preventing root fractures. Before root canal shaping, dentin wall thickness dimensions of 1 mm and below have been demonstrated in anatomical studies.[113] Straightening of canal paths can lead to precarious thinning of curved root walls (Fig. 8.5). Although no definitive minimal radicular wall thickness has been established, 0.3 mm is considered critical by some authors.[252] To avoid overpreparation and outright perforations, adequate access cavity preparation and optimal enlargement of the coronal third of the root canal has to be ascertained (see upcoming discussion).

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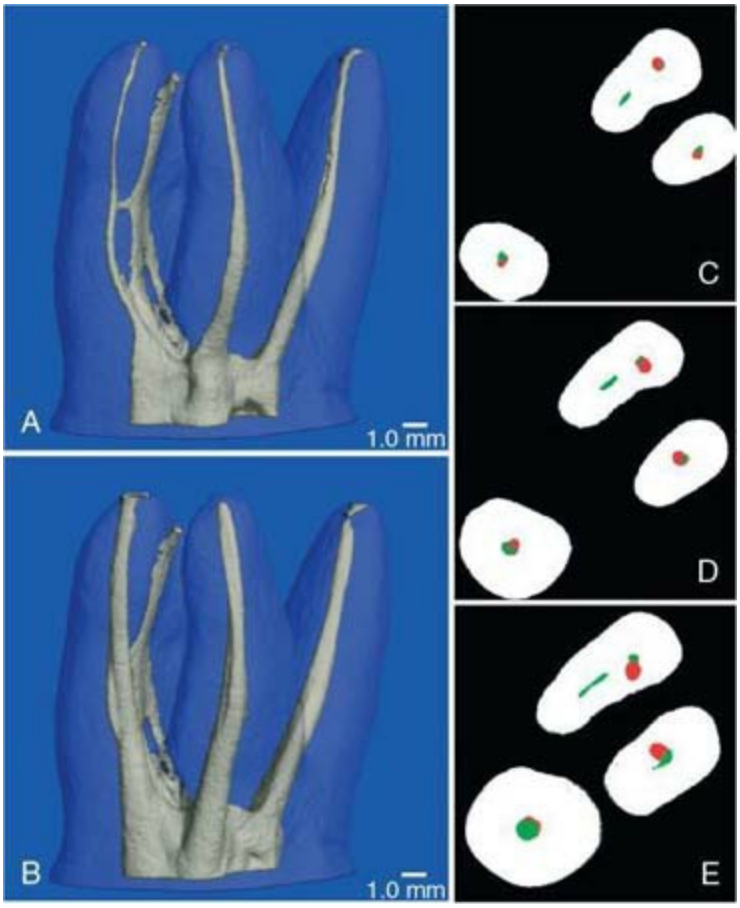

FIG. 8.5 Example of excessive thinning of dental structure during root canal treatment. **A–B**, Micro–computed tomography reconstructions show pretreatment and posttreatment root canal geometry of a maxillary molar. **C–E**, Cross sections of the coronal, middle, and apical thirds with pretreatment canal cross sections. Note the transportation and thinning, in particular, in the main mesiobuccal canal.

A) Scan of a maxillary molar shows a laterally split canal.

B) Scan of a maxillary molar shows partially filled and reconstructed split canal.

C) Cross-section of coronal canal shows red dot in the center with a green dot in it with a green streak on the side; Cross-section of middle canal shows overlapping green and red dots in the center; and Cross-section of apical canal shows a red dot in the center and an outline of green on one side.

D) Cross-section of coronal canal shows red dot in the center with an outline of green on one side and a green streak on the side; cross-section of middle canal shows overlapping green and red dots in the center; and cross-section of apical canal shows a red dot in the center and green dot in it.

E) Cross-section of coronal canal shows red and green dot in the center with a

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green streak on the side; Cross-section of middle canal shows green dot in the center with an outline of red on one side; and Cross-section of apical canal shows overlapping red and green dots in the center.

Biologic objective

Schilder suggested that canals should be prepared to a uniform and continuous taper;[386] however, this guideline was aimed at facilitating obturation rather than targeting antimicrobial efficacy. For optimal disinfection, preparation shape and antimicrobial efficacy are intimately related through efficient removal of infected pulp and dentin (Fig. 8.6) and creation of space for delivery of irrigants.

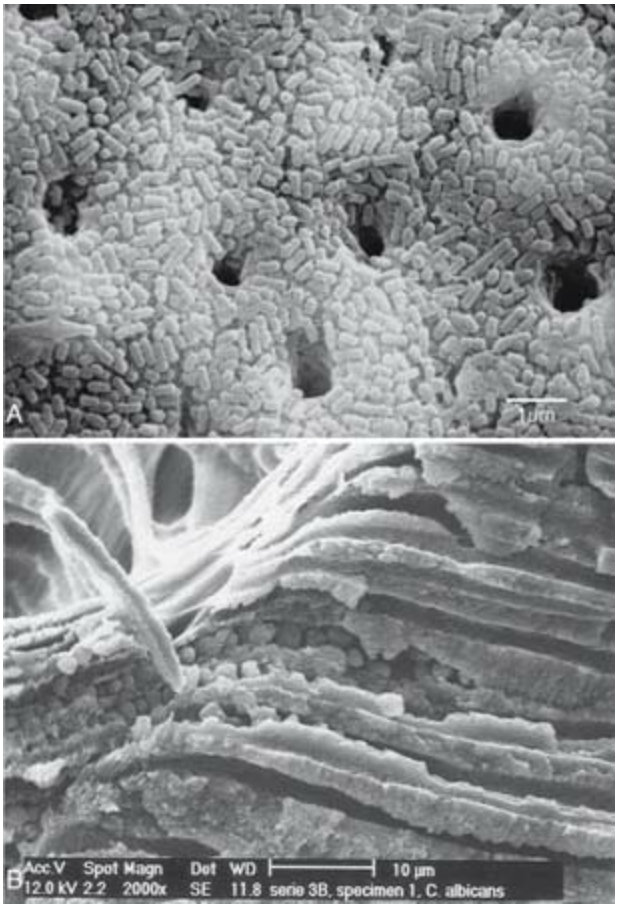

FIG. 8.6 Presence of microorganisms inside the main root canal and dentinal tubules. **A,** Scanning electron micrograph of a root canal surface shows a confluent layer of rod-shaped microbes (×3000). **B,** Scanning electron micrograph of a fractured root with a thick smear

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layer and fungi in the main root canal and dentinal tubules.

A) Micrograph of a root canal surface shows a layer of rod-shaped surface with ruptures of black over it.

B) Micrograph of a laterally segmented root surface with granular section and ruptures.

Source: (***A***, Courtesy Professor C. Koçkapan; ***B***, Courtesy Professor T. Waltimo.)

Traditionally, fluids have been dispensed passively into root canals by syringe and needle (Fig. 8.7). When delivered with passive needle irrigation, solutions have been shown to progress only about 1 mm farther than the tip of the needle.[169,350,370] Enlarged apical canals and finer more flexible needles allow increasingly deeper needle placement, and this improves débridement and disinfection of canals.[14,464] Irrigation methods and solution are discussed in detail elsewhere in this chapter.

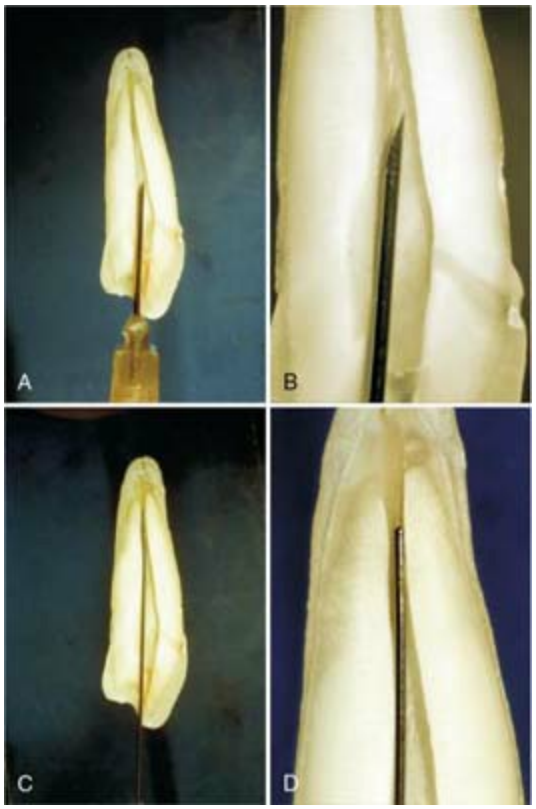

FIG. 8.7 Irrigation needles inserted into prepared root canals. **A–B**, A 27-gauge needle barely reaches the middle third. **C–D**, A 30-gauge,

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side-venting needle reaches the apical third in adequately enlarged canals.

Close-up of a tooth with an exposed root canal shows an irrigation needle inserted into it as labeled from A through D.

Technical objective

While a continuous taper that encompasses the original shape and curvature of a given root canal is an accepted goal, final apical preparation size remains a much-disputed entity in root canal therapy, as does final taper of the preparation.[43] Arguments were made for better disinfection with larger sizes (i.e., #50 or greater)[79,363] in combination with smaller tapers of.02 to.05. Others found no difference whether the selected final size was small or large.[95,500]

Clinical issues

A wide spectrum of possible strategies exists for attaining the goal of removing the canal contents and eliminating infection. Lussi and colleagues[264] introduced an approach to removing canal contents and accomplishing disinfection that did not involve the use of a file: the so-called non-instrumentation technique. This system consisted of a pump, a hose, and a special valve that was cemented into the access cavity (Fig. 8.8, *A*) to provide oscillation of irrigation solutions (1% to 3% sodium hypochlorite) at a reduced pressure. Although in vitro data[263] suggested that canals could be cleaned and filled using this noninvasive system (see Fig. 8.8, *B* and *C*), clinical results were not as convincing (see Fig. 8.8, *D*).[25] Recently, a strategically similar technique was introduced, the GentleWave (Sonendo, Orange, CA, USA) unit, described in detail later.

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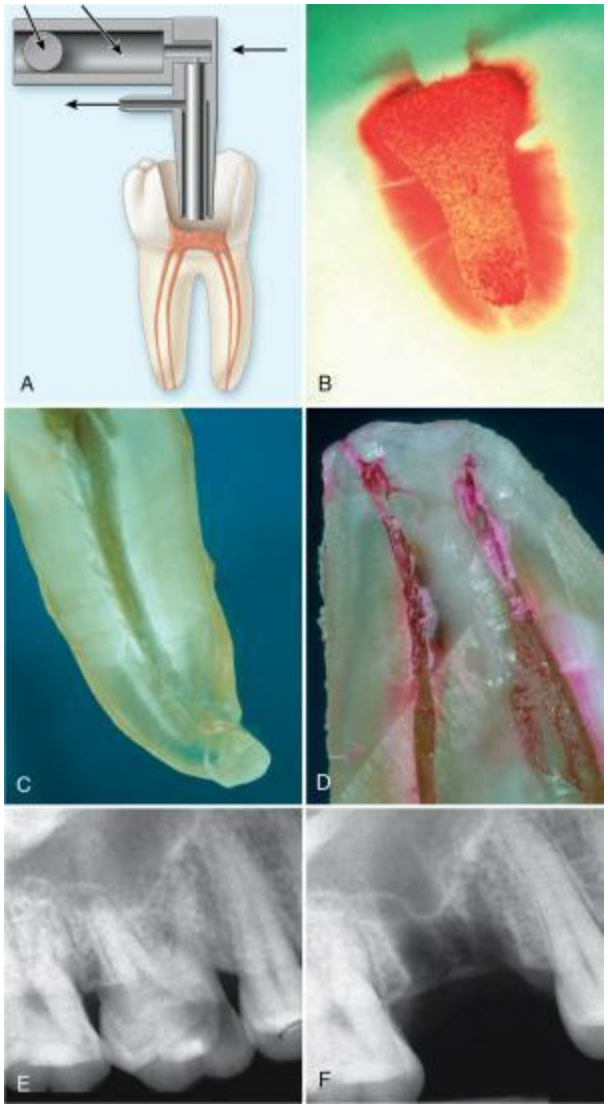

FIG. 8.8 Spectrum of strategies for accomplishing the primary aim of root canal treatment: elimination of infection. **A,** Schematic diagram of minimally invasive therapy using the noninstrumentation technique (NIT). **B,** Example of teeth cleaned in vitro using NIT. Note the clean intracanal surface, which is free of adhering tissue remnants. **C–D**, Examples of teeth cleaned in vivo and later extracted to investigate the clinical effects of NIT. Note the relatively clean, tissue-free canal space in **C** and the significant tissue revealed by rhodamine B staining in **D. E–F**, Course of maximally invasive therapy; apically involved tooth #30 was extracted, effectively removing the source of periradicular inflammation.

A) Diagram of a molar shows an exposed crown, neck and root canals. A non-instrumentation system accesses the crown, with arrows pointing to the pump, hose, and a special valve of the system.

B) Spectrum of clean tooth shows orange color and outlines of yellow.

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C) Close-up of a clean tooth with an exposed the root canal.

D) Close-up of a tooth shows two exposed and inflamed root canals.

E) Radiograph of tooth shows a radiolucent crown with radiopaque canals.

F) Radiograph of tooth shows a missing tooth with a dark patch around the gingival surface.

Source: (***A–B***, Courtesy Professor A. Lussi. ***C–D***, Courtesy Professor T. Attin. ***E–F***, Courtesy Dr. T. Kaya.)

At the opposite end of the spectrum of débridement strategies is to remove all intraradicular infection through extraction of the tooth in question. Almost invariably, periradicular lesions heal after extraction of the involved tooth.

Clinical endodontic therapy takes place somewhere along this spectrum of treatment strategies. This is reflected in some of the controversies that surround the cleaning and shaping process. Once the decision has been made to initiate endodontic treatment, a clinician must integrate their knowledge of dental anatomy, immunology, and bioengineering science with clinical information.

Endodontic therapy has been compared with a chain of events, wherein the chain is only as strong as each individual link. For the purpose of this chapter, shaping and cleaning of the root canal system is considered a decisive link, because shaping determines the efficacy of subsequent procedures. It includes mechanical débridement, the creation of space for the delivery of medicaments, and optimized canal geometries for adequate obturation.[326] These tasks are attempted within a complex anatomic framework, as recognized in the early 20th century by Walter Hess (Fig. 8.9; see also Chapter 7 for a complete description of root canal anatomy).[196]

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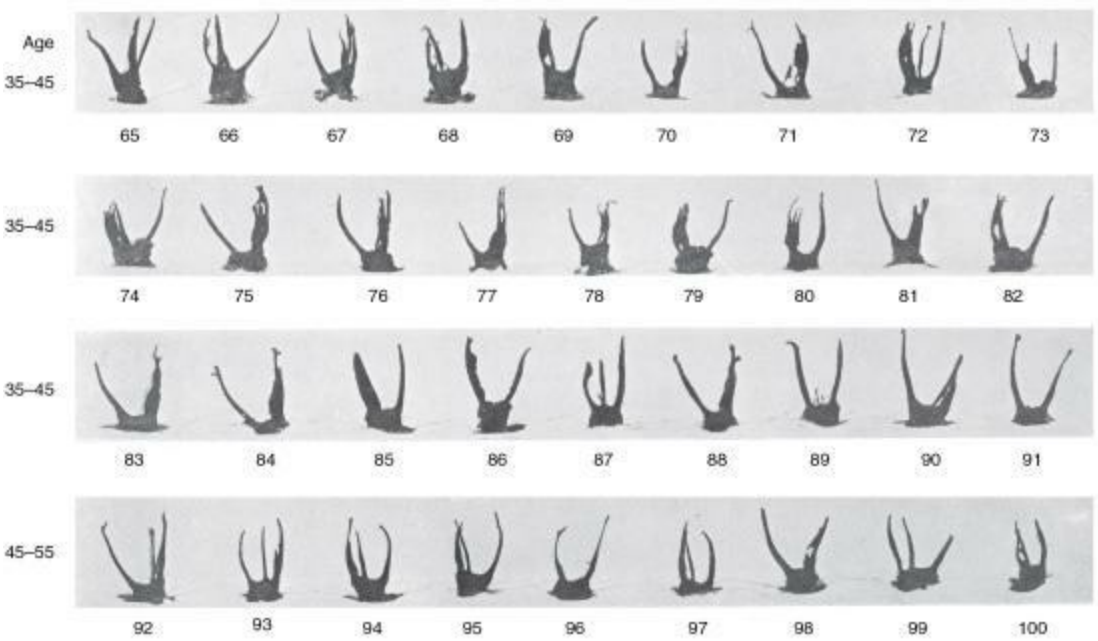

FIG. 8.9 Panel of 36 anatomic preparations of maxillary molars from the classic work by Professor Walter Hess of Zurich. Note the overall variability of root canal systems and the decrease of canal dimensions with age.

Set of four panel images show the types of maxillary molars and their canal systems in the age range of 35 to 45 and 45 to 55 years.

Source: (From Hess W: *The anatomy of the root canals of teeth of the permanent dentition*, London, 1925, John Bale, Sons & Danielsson).

A clinician must choose appropriate strategies, instruments, and devices to overcome challenges and accomplish precise preparation in shape, length, and width. This allows endodontic therapy to address various forms of the disease processes described previously (Fig. 8.10). Recall radiographs taken at appropriate intervals predictably demonstrate longevity and favorable outcomes (see Figs. 8.1, 8.2, and 8.11) if a systematic approach to root canal shaping is adhered to (see Box 8.1).

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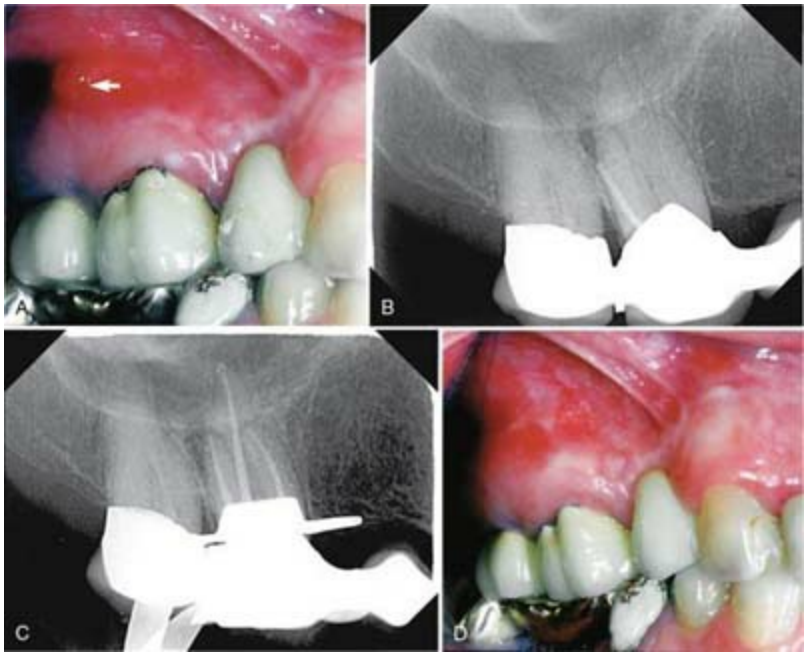

FIG. 8.10 Sinus tract as a sign of a chronic apical abscess and effect of routine root canal treatment. **A,** Intraoral photograph of left maxillary region with draining sinus tract *(arrow)* periapical to tooth #14. **B,** Pretreatment radiograph with gutta-percha point positioned in the sinus tract, pointing toward the distobuccal root of #14. **C,** Finished root canal fillings after 2 weeks of calcium hydroxide dressing. **D,** Intraoral photograph of the same region as in **A,** showing that the sinus tract had closed by the time obturation was performed.

A) Close-up of an open mouth shows a set of anterior teeth with an arrow pointing to the draining sinus tract.

B) Radiograph of teeth shows a white radiopaque outline of sinus tract over the crowns with radiolucent pulp. A radiopaque outline of a gutta-percha point is visible.

C) Radiograph of teeth shows a white radiopaque outline of sinus tract over the crowns. A radiopaque outline of canal filling and the gutta-percha point is visible. Radiolucency is visible in canals.

D) Close-up of an open mouth shows a set of anterior teeth with a closed sinus tract.

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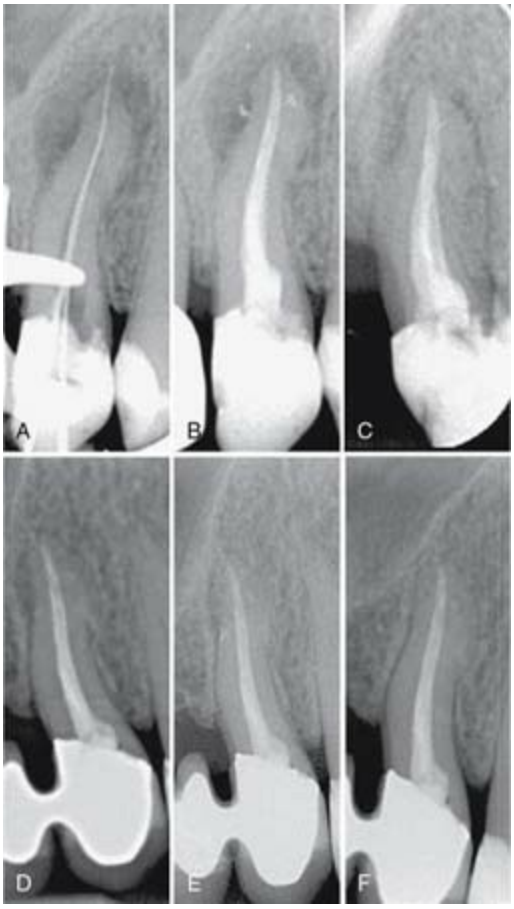

FIG. 8.11 Relationship of radicular anatomy and endodontic disease as shown by filled accessory canals. **A,** Working length radiograph of tooth #13 shows lesions mesially and distally but not apically. **B,** Posttreatment radiograph shows the accessory anatomy. **C,** Six-month recall radiograph before placement of the restoration. **D,** Two-year recall radiograph after resection of the mesiobuccal root of tooth #14 and placement of a fixed partial denture. Excess sealer appears to have been resorbed, forming a distal residual lesion. **E,** Four-year recall radiograph shows almost complete bone fill. **F,** Seven-year recall radiograph; tooth #14 is radiologically sound and clinically within normal limits.

A) Radiograph of tooth shows a white radiopaque crown with a radiolucent thread-like canal.

B) Radiograph of tooth shows a white radiopaque crown, radiolucent part of the neck and radiopaque thick canal.

C) Radiograph of tooth shows a white radiopaque crown with smudge of black on the top, radiolucent part of the neck and radiopaque canal with radiolucent pulp.

D) Radiograph of tooth shows a gray radiopaque U-shaped patch on the crown

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with radiolucent part of the neck and radiopaque canal.

E) Radiograph of tooth shows a gray radiopaque U-shaped patch on the crown with radiopaque neck and radiopaque canal.

F) Radiograph of tooth shows a gray radiopaque U-shaped patch on the crown with radiopaque neck and radiopaque canal.

Endodontic files traditionally have been manufactured according to empiric designs, and most instruments still are conceived based on individual clinicians' philosophies rather than developed through an evidence-based approach. Similar to the development of composite resins in restorative dentistry, the development of new files is a fast and market-driven process. With new instruments becoming available, the clinician may find it difficult to pick the file and technique most suitable for an individual case. Clinicians must always bear in mind that all file systems have specific benefits and weaknesses. Ultimately, clinical experience, handling properties, usage safety, and case outcomes, rather than marketing or the inventor's name, should drive decisions toward a particular design. The following section describes typical instruments used in root canal shaping.

Endodontic instruments

General characteristics

Design elements

Root canal preparation instruments such as K-files and nickel-titanium rotary instruments follow certain design principles that relate to drills and reamers used for work in wood and metal, respectively, while other instruments such as broaches and Hedström files do not find a direct technological correlate. Design elements such as the tip, flutes, and cross sections are considered relevant for files and reamers used in rotary motion. These pertinent aspects are briefly described later; for a more detailed review, the reader is referred to the literature.[162,365,371,423]

Tip design.

In root canal preparation, an instrument tip has two main functions: to guide

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the file through the canal and to aid the file in penetrating deeper into the canal. A clinician unfamiliar with the tip design, in particular of a rotary instrument, may do either of the following: (i) transport the canal (if the tip is capable of enlarging the canal and is used too long in one position in a curved canal) or (ii) encounter excessive torsion and break the file (if a noncutting tip is forced into a canal with a smaller diameter than the tip).

The angle and radius of its leading edge and the proximity of the flute to its actual tip end determine the cutting ability of a file tip. Cutting ability and file rigidity determine the propensity to transport the canal. The clinician must keep in mind that as long as a flexible file with a noncutting tip is engaged, 360-degree canal transportation is unlikely to occur.[360]

Studies have indeed shown that tip design affects file control, efficiency, and outcome in the shaping of root canal systems.[283,284] The tip of the original K-file resembled a pyramid; instrument tips have been described as *cutting*, *noncutting*, and *partially cutting*, although no clear distinction exists among the three types (Fig. 8.12).

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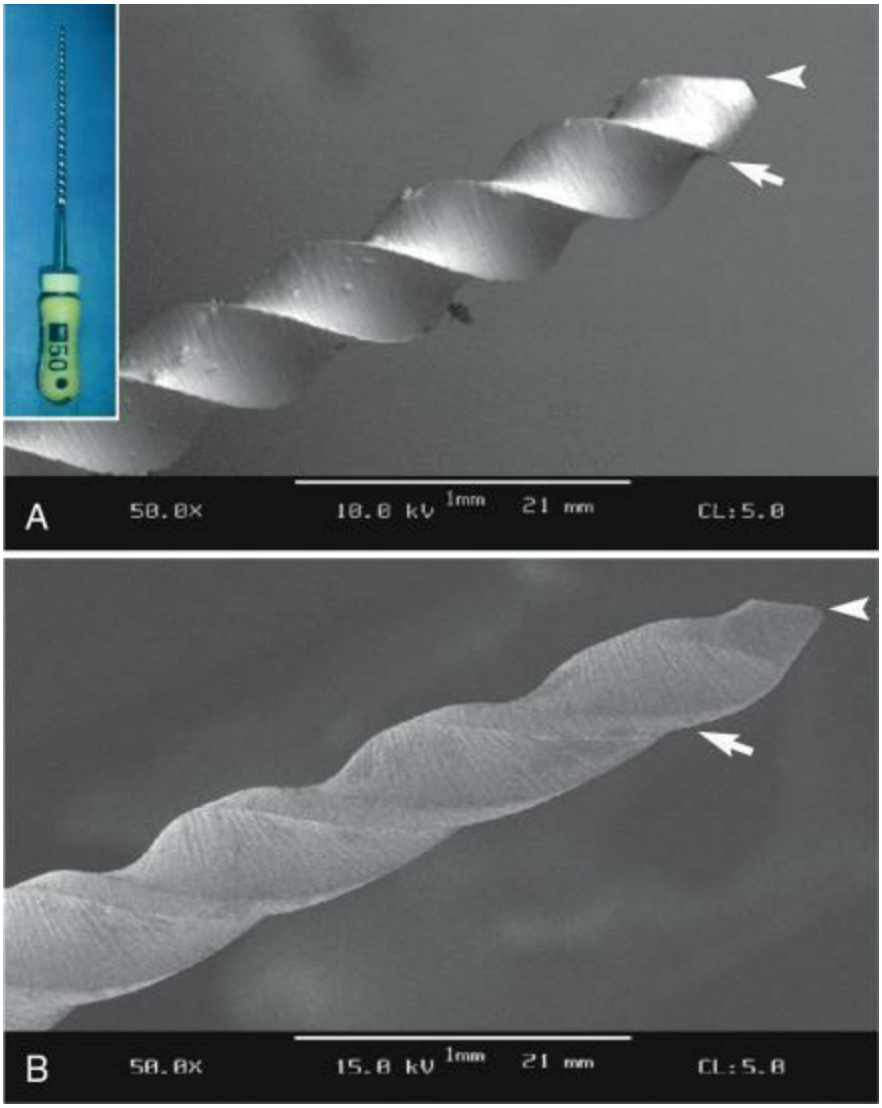

FIG. 8.12 Comparison of the flute geometry and tip configuration of a hand file *(insert)* and a NiTi rotary instrument. **A,** K-file with sharp cutting edges *(arrow)* and Batt tip *(arrowhead).* **B,** GT rotary file with rounded, noncutting tip *(arrowhead),* smooth transition, and guiding radial lands *(arrow).*

A) Close-up of a rotatory hand file shows arrows pointing to the conical flat tip and the sharply textured spiral surface.

B) Close-up of a rotatory hand file shows arrows pointing to the conical curved tip and the flatly textured spiral surface.

Noncutting tips, also called *Batt tips*, are created by grinding and smoothing the apical end of the instrument (see Fig. 8.12, *A*). A tip modification was introduced with the Flex-R file, which was manufactured

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fully by grinding so that the transitional angles between the tip and the instrument's working parts blend smoothly.[361] Similar techniques are required to manufacture NiTi K-files.[449]

For NiTi rotary files, typically rounded noncutting tips are used (see Fig. 8.12, *B*), which are effective in preventing preparation errors that were found with earlier so-called safe cutting tips.[210] One exception to this rule are rotaries specifically designed as retreatment instruments; cutting tips in that case facilitate removal of the existing root canal filling material and are sufficiently safe.

Longitudinal and cross-sectional design.

The *flute* of the file is the groove in the working surface used to collect soft tissue and dentin chips from the wall of the canal. The effectiveness of a flute depends on its depth, width, configuration, and surface finish. The surface with the greatest diameter that follows the groove (where the flute and land intersect) as it rotates forms the *leading (cutting) edge* or the *blade* of the file. The cutting edge creates and deflects dentin chips along the wall of the canal and severs or snags soft tissue. Its effectiveness depends on its angle of incidence and sharpness.

Some instruments have a feature between trailing and cutting edge that forms a larger contact area with the radicular wall; this surface is called a radial *land* (Fig. 8.13). This design is believed to reduce the tendency of the file to thread into the canal. It also supports the cutting edge and limits the depth of cut; its position relative to the opposing cutting edge and its width determine its effectiveness. On the other hand, landed files are typically less cutting efficient compared with triangular cross sections.[335]

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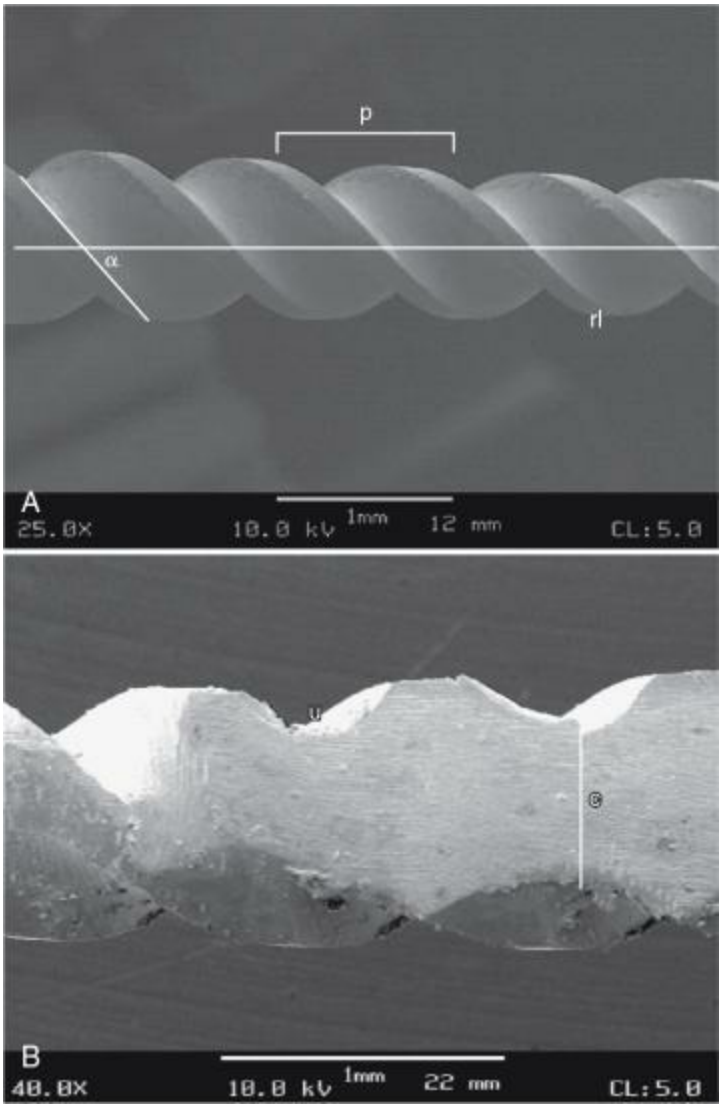

FIG. 8.13 Design characteristics of nickel-titanium rotary instruments. **A,** Lateral view showing the details of the helical angle, pitch *(p),* and the presence of guiding areas, or radial lands *(rl)* (scanning electron micrograph, ×25). **B,** Ground-working part of the instrument in **A,** showing U-shaped excavations and the dimension of the instrument core *(c).*

Set of two images show close-up of spirally textured geometry of nickel-titanium rotary file as labeled through A to B.

To reduce frictional resistance, some of the surface area of the land that rotates against the canal wall may be reduced to form the *relief.* The angle the cutting edge forms with the long axis of the file is called the *helical angle* (see Fig. 8.13).

If a file is sectioned perpendicular to its long axis, the *rake angle* is the

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angle formed by the leading edge and the radius of the file through the point of contact with the radicular wall. The rake angle may be *positive* or *cutting* (Fig. 8.14, *A*). If the angle formed by the leading edge and the surface to be cut is 90 degrees, the rake angle is said to be *neutral*. It may also be *negative* or *scraping* (see Fig. 8.14, *C*). The *cutting angle* is considered a better indication of a file's cutting ability and is determined by measuring the angle formed by the leading edge of the file and a tangent to the radicular wall in the point of contact. The clearance angle corresponds to the cutting angle at the trailing edge of the file and, in case of reciprocating action, becomes the cutting angle. The sum of cutting angle and rake angle is 90 degrees.

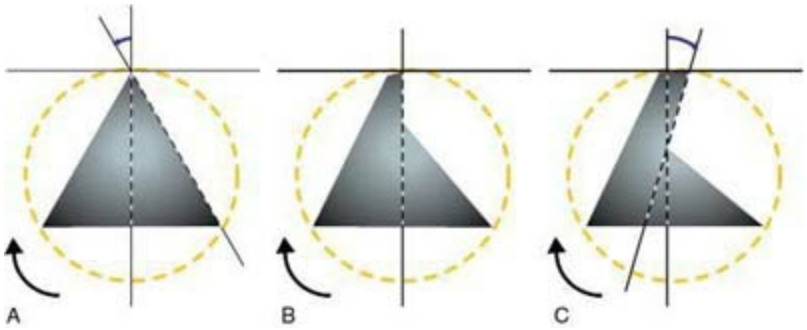

FIG. 8.14 The cutting angle of an endodontic file can be negative (**A**), neutral (**B**), or positive (**C**).

A) Diagram shows two halves of a triangle segmented vertically with a lined circle enclosing it. Right side of the triangle creates an anti-clockwise torque.

B) Diagram shows two halves of a triangle segmented vertically with a lined circle enclosing it. The right half of the triangle is smaller than the left half (which has slightly flat top).

C) Diagram shows two halves of a triangle segmented vertically with a lined circle enclosing it. The right half of the triangle is smaller than the left triangle which extends to the right half creating a clockwise torque.

The *pitch* of the file is the distance between a point on the leading edge and the corresponding point on the adjacent leading edge (the distance from one "spiral twist" to the next; see Fig. 8.13). The smaller the pitch or the shorter the distance between corresponding points, the more spirals the file has and the greater the helix angle. While K-files have a constant pitch typically in the range of 1 mm, many NiTi rotaries have a variable pitch—one that

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changes along the working surface. When variable pitch is used, for example to modify the helical angle, usually tighter spirals are located close to the tip of the file and more space between the flutes is located toward the coronal part of the file. A longitudinal section through an instrument reveals the *core* (see Fig. 8.13). The outer diameter of a tapered instrument increases from the file tip toward the handle. Depending on core dimensions, flutes may become proportionately deeper, resulting in a *core taper* that may be different from the *external taper*.

Cutting angles, helix angles, and external and core tapers may vary along the working surface of the file, and the ratios of these quantities can vary among instruments of the same series.

Taper.

The *taper* usually is expressed as the amount that the file diameter increases each millimeter along its working surface from the tip toward the file handle. For example, a size #25 file with a.02 taper would have a 0.27 mm diameter 1 mm from the tip, a 0.29 mm diameter 2 mm from the tip, a 0.31 mm diameter 3 mm from the tip, and so forth. Instruments can have constant or variable taper. Current instrument developments include variations in helical angle, pitch, and taper along the cutting portion, which along with variations in alloy and rotational speed (rpm) all affect cutting behavior.[335] The ability to determine cross-sectional diameter at a given point on a file can help the clinician determine the file size in the point of curvature and the relative stress being placed on the instrument. Instruments with greater tapers are designed so that the tip of the instrument functions as a guide, and the middle and coronal part of the instrument's working part is the one engaging the canals walls.

International Standards Organization norms

Standardized specifications have been established to improve endodontic instrument quality.[212] For example, the International Standards Organization (ISO) has worked with the Fédération Dentaire Internationale (FDI) and other stakeholders to define specifications. These standards are designated with an ISO number. The American Dental Association (ADA) also has been involved in this effort, as has the American National Standards Institute

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(ANSI); these standards are designated with an ANSI number. However, new instrument designs have resulted in a need for reconsideration of the standards.

Two ISO standards pertain to endodontic instruments: ISO No. 3630-1 deals with K-type files (as does ANSI No. 28), Hedström files (ANSI No. 58), and barbed broaches and rasps (ANSI No. 63). ISO No. 3630-3 deals with condensers, pluggers, and spreaders (ANSI No. 71); however, the term *ISO-normed instruments* is often synonymous for K-files (Fig. 8.15).

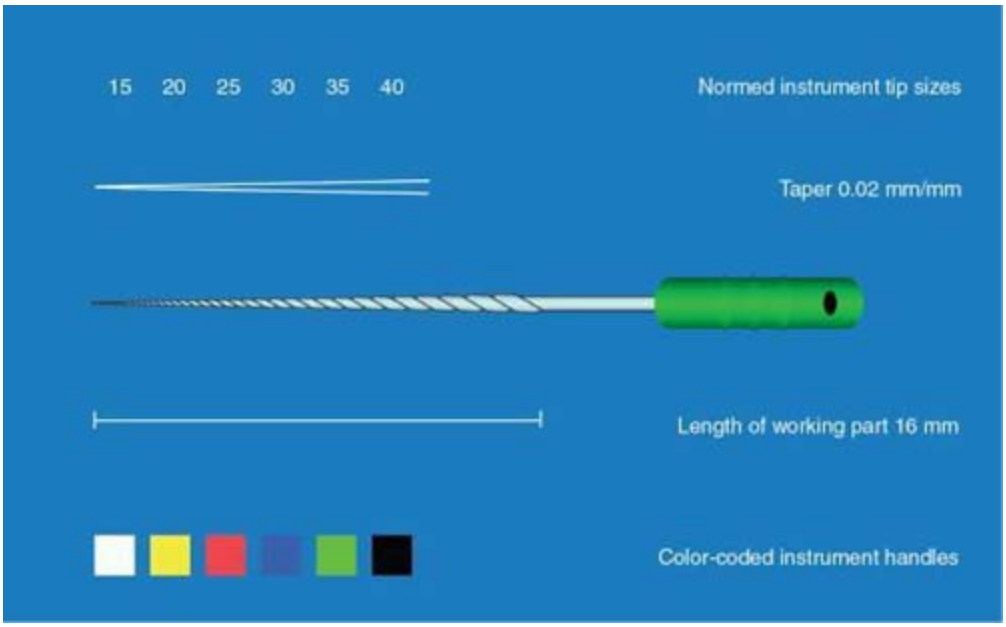

FIG. 8.15 Schematic drawing of an ISO-normed hand instrument size #35. Instrument tip sizing, taper, and handle colors are regulated by the ISO/ANSI/ADA norm.

Diagram of a hand file shows a spirally textured surface and tip with a green handle. The hand file come in sizes 15, 20, 25, 30, 35 and 40 with work length of 16 millimeters and color codes of white, yellow, red, purple, green and black.

One important feature of ISO-normed hand instruments is a defined increase in tip diameter of 0.05 or 0.1 mm, depending on the instrument size (Fig. 8.16). ISO-normed K- and Hedström files (Fig. 8.17) are available in different lengths (21, 25, and 31 mm), but all have a 16-mm-long section of cutting flutes (see Figs. 8.12 and 8.15). The cross-sectional diameter at the

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first rake angle of any file is labeled D_0. The point 1 mm coronal to D_0 is D_1, the point 2 mm coronal to D_0 is D_2, and so on up to D_{16}. The D_{16} point is the largest diameter of an ISO-normed instrument. Each file derives its numeric name from the diameter at D_0 and is assigned a specific color code (see Fig. 8.15). Another aspect of ISO files is the standard taper of 0.32 mm over 16 mm of cutting blades, or 0.02 mm increase in diameter per millimeter of flute length (.02 taper).

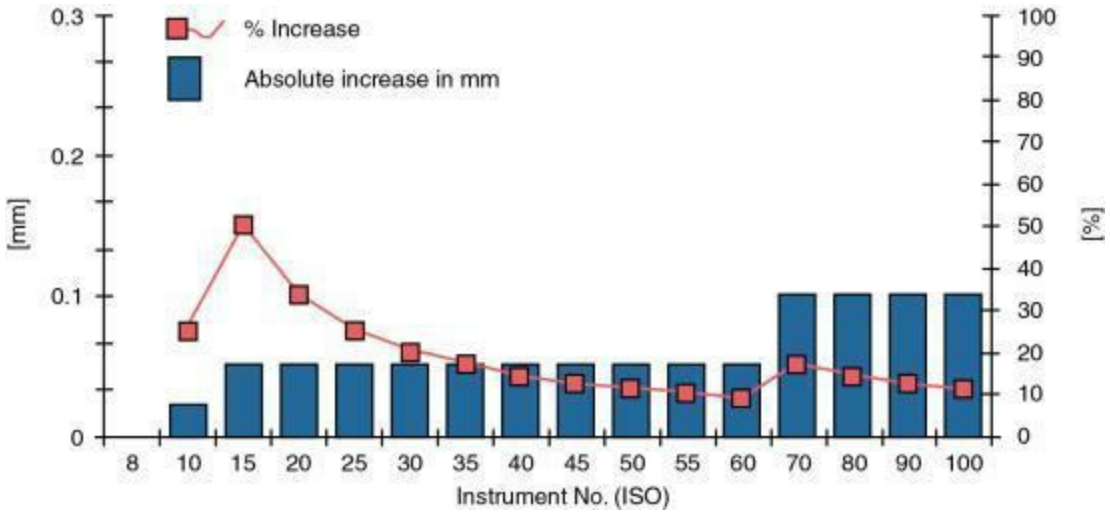

FIG. 8.16 Increase in tip diameter in absolute figures and in relation to the smaller file size. Note the particularly large increase from size #10 to size #15.

Plot shows instrument numbers versus with absolute increase in tip diameters (in millimeters) and increase (in percentage). The horizontal axis shows instrument numbers ranging from 0 to 100. The left vertical axis shows diameters (in millimeters) ranging from 0 to 0.3 in increments of 0.1. The right vertical axis shows percentage ranging from 0 to 100 in increments of 10.

The data for absolute increase depicted as bars is as follows:

10: 7, 15 to 60: 18; and 70 to 100: 34.

The data for percentage increase is as follows:

10: 0.066, 15: 0.135, 20: 0.1, 25: 0.066, 30: 0.65; decreasing to 0.33 at 60, which increases to 0.05 at 70 decreasing to terminate at 0.04 at 100.

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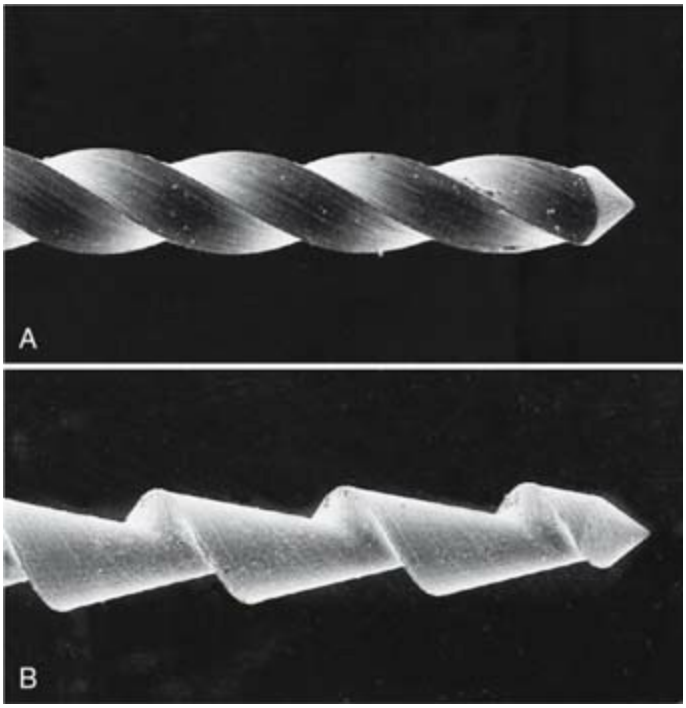

FIG. 8.17 Scanning electron micrographs of endodontic hand files fabricated by twisting (K-file size #40, **A**) and grinding (Hedström file #50, **B**).

A) Micrograph of a K- file shows a sharply textured spiral surface body with a sharp conical tip.

B) Micrograph of a hedstrom file shows conically textured surface body with a sharp conical tip.

Source: (***A–B***, Courtesy Dentsply Maillefer, Ballaigues, Switzerland.)

The ISO-normed design is a simplification that has specific disadvantages, and it may explain the clinical observation that enlarging a root canal from size #10 to #15 is more difficult than the step from size #55 to #60. The introduction of K-type files with tip sizes between the ISO-stipulated diameters seemed to solve the problem. However, the use of such file is not universally recommended, perhaps because the approved machining tolerance of 0.02 mm could negate any intended advantages. Moreover, although 0.02 mm tolerance is stipulated by the ISO norm, most manufacturers do not adhere to it.[223,389,437,511] Another suggested modification relates to tips with a constant 29% of diameter increments.

Further changes to the numbering system for files with different sizes have been implemented by several manufacturers. One system has introduced

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“half” sizes in the range of #15 through #60, resulting in instruments in sizes #15, #17.5, #20, #22.5, and so on.

Alloys

There are currently two principally different types of alloys used for endodontic instruments: stainless steel and nickel-titanium. Most manually operated endodontic instruments are fabricated from stainless steel and have considerable resistance to fracture. A clinician who is careful in applying force and adheres to a strict program of discarding instruments after use should have few instrument fractures. Stainless steel files are comparably inexpensive so that adequate cleaning and sterilization for reuse of files in sizes up to #60 may not be cost effective. Indeed, files in the range up to #60 have been considered disposable instruments.[429]

Several burs and instruments designed for slow-speed handpiece operation such as Gates Glidden drills, Peeso burs, and pilot drills for intraradicular posts are also manufactured from stainless steel. Instruments designed for rotary root canal instrumentation, however, are typically made of nickel-titanium.[393] This alloy offers unique properties, specifically flexibility, and corrosion resistance.

Physical and chemical properties of steel and nickel titanium alloys.

Basic engineering terms relate to metals and their behavior when used to manufacture endodontic instruments. Stress-strain diagrams describe the response of metal wires under loading depending on their crystal configuration (Fig. 8.18).

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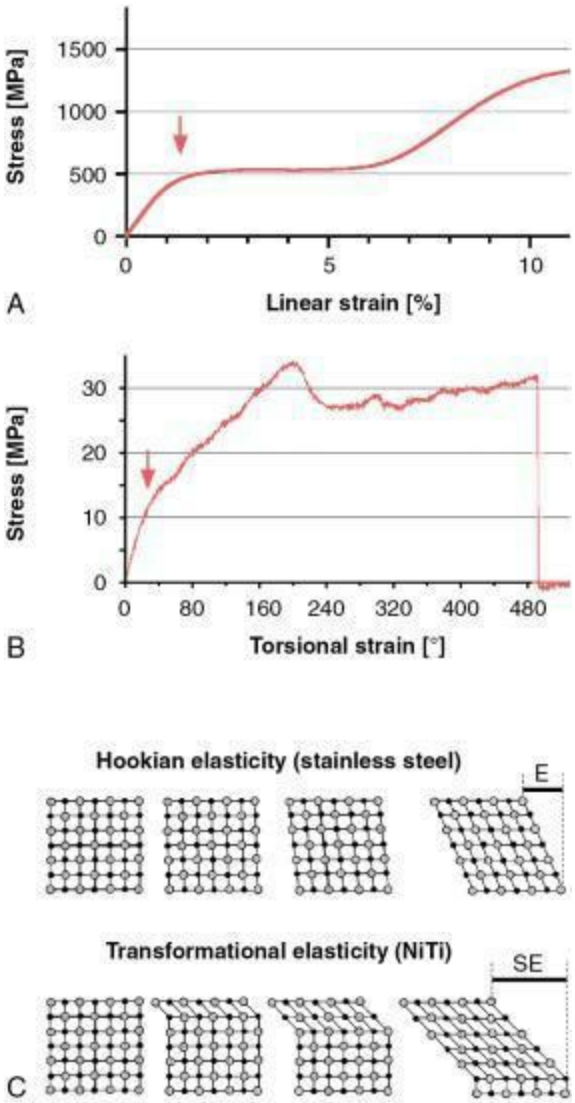

FIG. 8.18 Stress-strain behavior of nickel-titanium alloy. **A,** Schematic diagram of linear extension of a NiTi wire. **B,** Torque to failure test of a size #60,.04 taper ProFile NiTi instrument. Note the biphasic deformation, indicated by arrows in **A–B. C,** Comparison of stainless steel and nickel-titanium crystal lattices under load. Hookian elasticity accounts for the elastic behavior *(E)* of steel, whereas transformation from martensite to austenite and back occurs during the pseudoelastic *(PE)* behavior of NiTi alloy.

A) Line-graph of linear strain (in percents) versus stress (in megapascals). A curve begins from (0,0) to (11, 1350) showing an increasing trend from (1.2, 500) pointed by an arrow.

B) Line-graph of torsional strain (in percents) versus stress (in megapascals). A curve begins from (0,0) to (0, 530) showing an increasing trend from (40, 14) pointed by an arrow, declining abruptly at (500, 32).

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C) Schematic diagrams of crystal lattices for hookian elasticity in stainless steel shows increasing strain in the entire lattice and crystal lattices for transformational elasticity in nickel titanium shows increasing strain in the upper lattice.

Source: (***C***, Modified from Thompson SA: An overview of nickel-titanium alloys used in dentistry, *Int Endod J* 33:297–310, 2000.)

During the development of the equiatomic *nitinol* (this acronym is derived from **ni**ckel-**ti**tanium investigated at the **N**aval **O**rdinance **L**aboratory) alloy (55% [by weight] nickel and 45% [by weight] titanium), several effects were noted that relate to its specific crystal arrangement with two stable main phases, *austenite* and *martensite* (Fig. 8.19): a shape memory effect as temperature- and strain-dependent pseudoelasticity, all attributable to specific thermodynamic properties of the new alloy.[69,119,310,331,449]

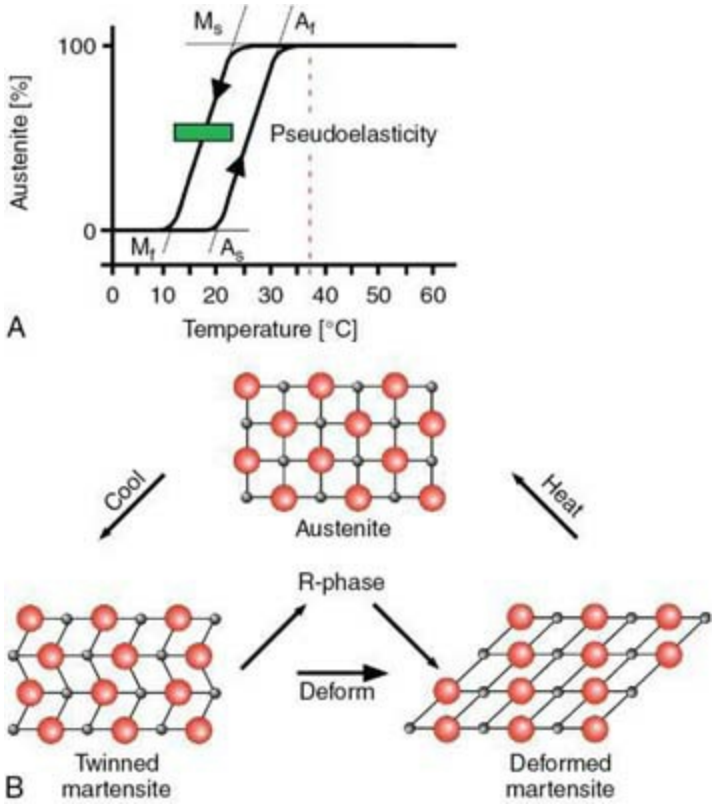

FIG. 8.19 Pseudoelastic behavior of nickel titanium is based on the two main crystal configurations, martensite and austenite, which depend on temperature (**A**) and applied strain (**B**). Formation of the respective configuration initiates at the start temperatures, M_s and A_s.

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A) Plot shows temperature (in Celsius) versus austenite (in percents). The horizontal axis ranges from 0 to 60 in increments of 10. The vertical axis ranges from 0 to 100. A curve M sub 1 increases from (0,0) to (65, 100) with a green box at (15, 50). A curve A sub S increases from (0,0) to (65, 100). An vertical pseudoelasticity line increases from (36.5, 0) to (36.5, 100).

B) Twinned martensite with zigzag lattice deforms through R-phase into deformed martensite with rhomboidal lattice. Deformed martensite is heated to form austenite with square lattice which cools backs to twinned martensite.

Walia and others[474] thought that the pseudoelastic properties of 55-nitinol might prove advantageous in endodontics and initially tested hand instruments. They found that size #15 NiTi instruments were two to three times more flexible than stainless steel instruments; moreover, the instruments showed superior resistance to angular deflection.[474]

Furthermore, hardly any plastic deformation of cutting flutes was recorded when an instrument was bent up to 90 degrees, and forces required to bend endodontic files to 45 degrees were reduced by 50% with nickel titanium, also known as NiTi.[393,474] Serene and others[393] speculated that heat, probably during sterilization cycles, could even restore the molecular structure of used NiTi files, resulting in an increased resistance to fracture. Such a behavior is claimed to occur for current martensitic instruments.[332]

These unusual properties are the result of a molecular crystalline phase transformation in specific crystal structures of the austenitic and martensitic phases of the alloy.[449] External stresses transform the austenitic crystalline form of NiTi into a martensitic crystalline structure that can accommodate greater stress without increasing the strain. As a result, a NiTi file has transformational elasticity, also known as *pseudoelasticity*, or the ability to return to its original shape after being deformed (see Fig. 8.19, *B*). This property dictates that typical NiTi instruments are manufactured by milling rather than twisting. Twisting incorporates plastic deformation and is used, for example, to produce stainless steel K-files. Another recently introduced manufacturing technique is electric discharge machining, which apparently does not introduce defects.[344]

Similar to the application of deforming forces, temperature changes also result in phase transformation (see Fig. 8.19, *A*) from austenite to martensite and vice versa.[190,282] Moreover, thermal heat applied either during the

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production of the raw wire or after manufacturing of the file can be used to modify its properties, most importantly, its flexibility.[172,399] For austenitic endodontic instruments a recoverable elastic response of up to 7% is expected (Fig. 8.20). However, more martensitic instruments will have less of an elastic range and are more likely to plastically deform during use.[332,399]

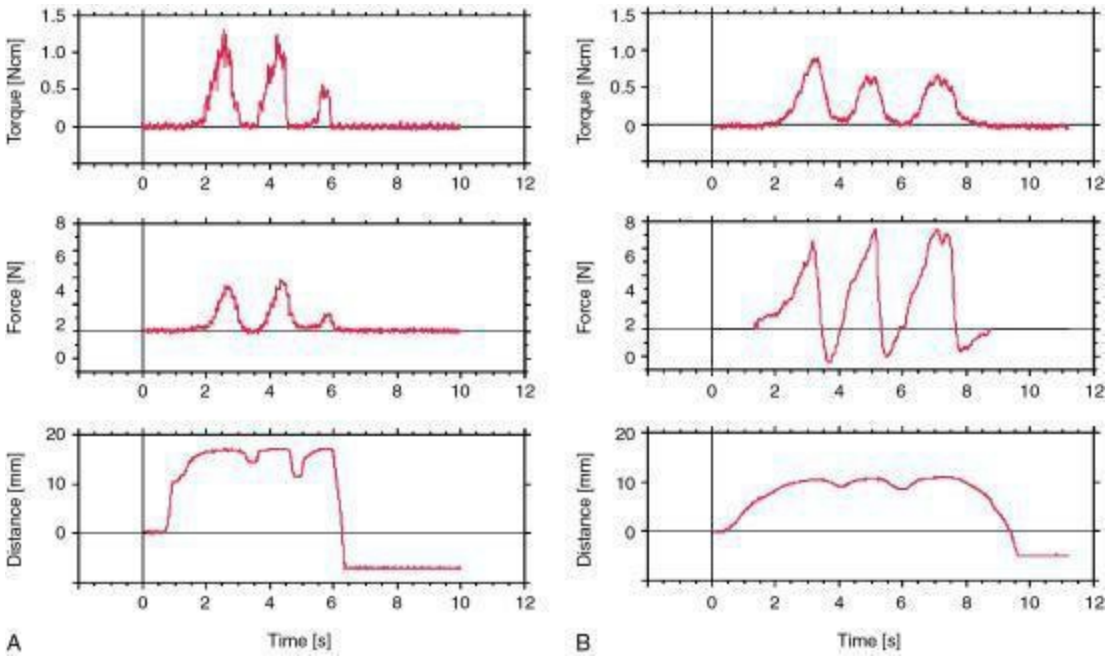

FIG. 8.20 Physical factors (torque, axial force, and insertion depth) that affect root canal instrumentation documented with a torque-testing platform. **A,** ProFile size #45,.04 taper used in a mildly curved canal of a single-rooted tooth, step-back after apical preparation to size #40. **B,** FlexMaster size #35,.06 taper used in a curved distobuccal canal of a maxillary first molar, crown-down during the initial phase of canal preparation.

A) Line graph of torque (in Newton centimeter) against time (in seconds) shows a curve with pointed crests and U-shaped troughs. The curve begins at (0, 2) and ends at (10, 0).

B) Line graph of force (in Newtons) against time (in seconds) shows a curve with U-shaped crests and troughs. The curve begins at (0, 2) and ends at (10, 0).

C) Line graph of distance (in millimeters) against time (in seconds) shows a U-shaped curve with internal crests and troughs. The curve begins at (0, 0) and ends at (10, negative 4).

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D) Line graph of torque (in Newton centimeter) against time (in seconds) shows a curve with pointed crests and U-shaped troughs. The curve begins at (0, 2) and ends at (11, 0).

E) Line graph of force (in Newtons) against time (in seconds) shows a curve with zigzag crests and troughs. The curve begins at (1.5, 2) and ends at (8.9, 2).

C) Line graph of distance (in millimeters) against time (in seconds) shows a U-shaped curve with internal crests and troughs. The curve begins at (0, 0) and ends at (11.8, negative 5).

NiTi instruments may have imperfections such as milling marks, metal flash, or rollover.[474] Some researchers have speculated that fractures in NiTi instruments originate at such surface imperfections.[13]

Surface irregularities may provide reservoirs of corrosive substances—most notably sodium hypochlorite (NaOCl). Chloride corrosion may lead to micropitting[372] and possibly subsequent fracture in NiTi instruments.[184] Immersion in various disinfecting solutions for extended periods (e.g., overnight soaking) produced corrosion of NiTi instruments and subsequent decreased torsional resistance.[304,424] For ProTaper,[46] RaCe, and ProFile[339] instruments, also 2-hour immersion damaged the integrity of the alloy.

In the majority of studies sterilization procedures did not appear to negatively impact torsional strength[199,407] or fatigue resistance[71,198] of most NiTi instruments: austenitic[470] and martensitic[81] alloy behaved grossly similarly in this aspect. Some NiTi instruments are available in presterilized form, which eliminates the need to process and sterilize before use and provides a simpler workflow overall. There is an ongoing discussion over the impact of other aspects of clinical usage on the mechanical properties of NiTi rotaries. Most likely, clinical usage leads to some changes in the alloy, potentially through work hardening,[12,224]

Another strategy to improve file characteristics is electropolishing. Surface coatings and ion implantation have been tried. Electropolishing is a process that removes surface irregularities such as flash and bur marks. It is believed to improve material properties, specifically fatigue and corrosion resistance; however, the evidence for both these claims is contradictory. One study[19] found an extension of fatigue life for electropolished instruments, while others found no improvement of fatigue resistance of electropolished

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instruments.[70,195] Boessler et al.[54] suggested a change in cutting behavior with an increase of torsional load after electropolishing.

Perhaps more relevant than surface treatment are modifications of the base alloy that significantly alter material properties within the atomic ratio.[323] The first commercialized alloy to exploit this was M-Wire (SportsWire, Langley, OK, USA), which was shown to have higher fatigue resistance with similar torsional strength.[220]

More recently, most instrument are manufactured from drawn standard NiTi wires, which are then worked by micro milling and finally submitted to specific annealing and cooling steps.[331,399] Instruments manufactured with this workflow tend to be relatively more martensitic at room and possible at body temperature during root canal treatment, as defined by their M_s temperature (see Fig. 8.19, A). Typically, such heat-treated alloys are more flexible[447] and present higher fatigue resistance.[332] Examples are the so-called gold and blue alloys types (Dentsply Sirona, York, PA, USA) or so-called controlled memory alloy used in Hyflex instruments (Coltene Endo, Cuyahoga Falls, OH, USA).

Manually operated instruments

Endodontic trays contain many items familiar from general dentistry, but certain hand instruments are designed specifically for endodontic procedures. This includes instruments employed for procedures inside the pulp space—for example, hand- and engine-driven instruments for root canal preparation and energized instruments for root canal shaping. Special instruments and devices for root canal obturation are selected for filling prepared canal spaces.

K-type instruments

Manually operated instruments are generically called *files.* Defined by function, files are instruments that enlarge canals with apico-coronal insertion and withdrawal motions.

Files were first mass produced by the Kerr Manufacturing Co. of Romulus, Michigan, in the early 1900s—hence the name K-type file (or K-file). K-files and K-reamers were manufactured by the same process (i.e., by twisting square or triangular metal blanks along their long axis; see Fig. 8.17, *A*).

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Three or four equilateral, flat surfaces were ground at increasing depths on the sides of a piece of wire, producing a tapered pyramidal shape. The wire then was stabilized on one end, and the distal end was rotated to form the spiral instrument. The number of sides and the number of spirals determine whether the instrument is best suited for filing or reaming. In general, a three-sided configuration with fewer spirals (e.g., 16 per 16 mm working portion) is used for reaming (i.e., cutting and enlarging canals with rotational motions). A file has more flutes per length unit (e.g., 20) than a reamer, whereas a three-sided shape is generally more flexible than a four-sided one.[381]

K-type instruments are useful for penetrating and enlarging root canals. In general, a reaming motion (i.e., constant file rotation) causes less transportation than a filing motion (translational or "in-and-out" motion).[153] *Transportation* in root canal preparation may be defined in general as the movement away from the initial canal axis and is described in more detail later in this chapter.

Stainless steel K-files may be precurved by overbending; this procedure subjects the file to substantial strain and should therefore be done carefully. Permanent deformation occurs when the flutes become wound more tightly or opened more widely (Fig. 8.21). When such deformation occurs, an instrument should no longer be used; file fracture is likely to occur during clockwise motion after plastic deformation.[395]

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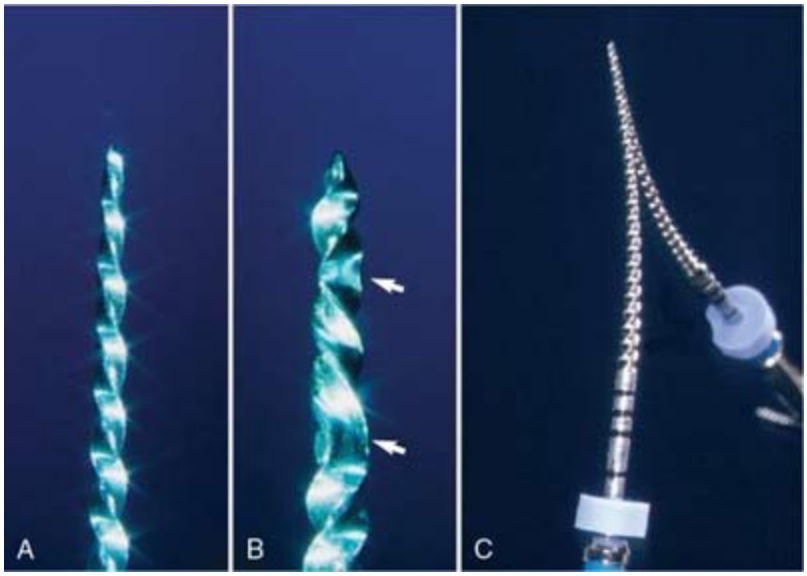

FIG. 8.21 Deformation of endodontic instruments manufactured from nickel-titanium alloy. **A** and **B,** Intact and plastically deformed ProFile instruments (*arrow* indicates areas of permanent deformation). **C,** ProFile instrument placed on a mirror to illustrate elastic behavior.

A) Close-up of a nickel titanium file shows a tight spirally textured sharp body with a sharp tip.

B) Close-up of a deformed nickel titanium file with arrows pointing to the large and small curvature of the spiral scales after deformation.

C) Close-up of a spirally textured instrument shows a mirror on one end and a hook on the other end.

Interestingly, although the force required for failure is the same in both directions of rotation,[244] failure occurs in the counterclockwise direction at half the number of rotations required for failure in the clockwise direction. Therefore K-type instruments should be operated more carefully when pressure is applied in a counterclockwise direction.

Cross-sectional analysis of a K-file reveals why this design allows careful application of clockwise and counterclockwise rotational and translational working strokes: The cross section is symmetrical with negative rake angles, allowing dentin to be adequately cut in both clockwise and counterclockwise direction.

H-type instruments

H-type instruments, also known as Hedström files (see Fig. 8.17, *B*), are

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milled from round, stainless steel blanks. These files are very efficient for translational strokes[381] because of a positive rake angle and a blade with a cutting rather than a scraping angle. Rotational working movements are discouraged because of the possibility of fracture. Hedström files up to size #25 can be efficiently used to relocate canal orifices and, with adequate filing strokes, to remove overhangs. Similarly, wide oval canals can be instrumented with Hedström files, as well as with rotary instruments. On the other hand, overzealous filing can lead to considerable thinning of the radicular wall and strip perforations (Fig. 8.22).

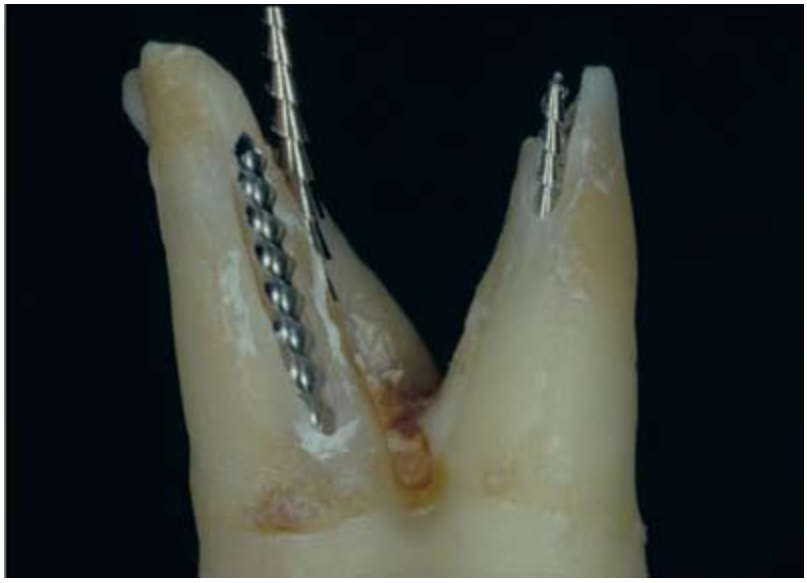

FIG. 8.22 Result of an overenthusiastic attempt at root canal treatment of a maxillary second molar with large stainless steel files. Multiple strip perforations occurred, so consequently the tooth had to be extracted.

Close-up of a maxillary second molar with roots exposing spirally and conically textured stainless steel files.

Precurving Hedström files results in points of greater stress concentration than in K-type instruments. Such prestressed areas may lead to the propagation of cracks and ultimately fatigue failure.[182] Note that clinically, fatigue fractures may occur without visible signs of deformation.

Hedström files are produced by grinding a single continuous flute into a tapered blank. Computer-assisted machining technology has allowed the development of H-type instruments with very complex forms. Because an H-

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file generally has sharper edges than a K-file, it has a tendency to thread into the canal during rotation, particularly if the instrument's blades are nearly parallel. Awareness of threading-in forces is important to avoid instrument failure, also with NiTi rotary instruments.

Effectiveness and wear of instruments.

The ability of an endodontic hand instrument to cut and machine dentin is essential; however, no standard exists for either the cutting or machining effectiveness of endodontic files, nor have clear requirements been established for resistance to wear. In any study of the effectiveness of an instrument, two factors must be investigated: (1) effectiveness in cutting or breaking loose dentin and (2) effectiveness in machining dentin.

Attempts have been made to evaluate the effectiveness of an instrument when used with a linear movement,[383] and these studies showed that instruments might differ significantly, not only when comparing brands and types but also within one brand and type. For K-files, effectiveness varies 2 to 12 times between files of the same brand. This variation for Hedström files is greater, ranging from 2.5 to more than 50 times.[436]

During canal preparation, a file's rake edge shaves off dentin that accumulates in the grooves between the rake edges. The deeper and larger this space, the longer the stroke can be before the instrument is riding on its own debris, making it ineffective. These design variations and the rake angle of the edges determine the effectiveness of a Hedström file. Of the hybrid files, the K-Flex (Kavo Kerr Endo, Orange, CA, USA) file has properties similar to those of K-files. The Flex-R file (Integra Miltex, Plainsboro, NJ, USA), which is a ground instrument with a triangular cross section similar to a K-file, more closely resembles a Hedström file in its variations in cutting behavior. It also is more effective at substrate removal than the K-files but cannot measure up to the H-files' ability to machine radicular dentin.[436]

Barbed broaches

Barbed broaches (Fig. 8.23) are produced in a variety of sizes and color codes. They are manufactured by cutting sharp, coronally angulated barbs into metal wire blanks. Broaches are intended to remove vital pulp from root canals, and in cases of mild inflammation and a large canal space, they work

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well for severing pulp at the constriction level in toto. The use of broaches has declined since the advent of NiTi rotary instruments, but broaching occasionally may be useful for expediting emergency procedures (see Chapter 19) and removing materials (e.g., cotton pellets or absorbent points) from root canals.

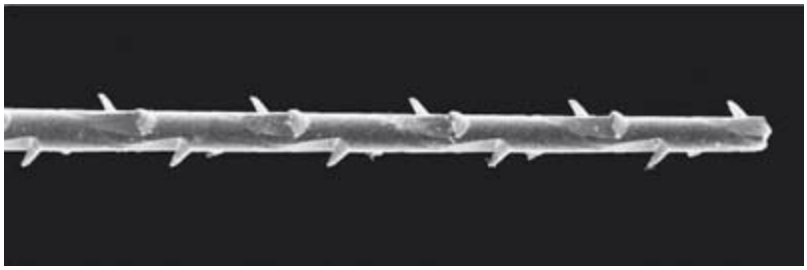

FIG. 8.23 Scanning electron micrograph of a barbed broach.

Close-up of a barbed broach shows a tapered round shaft with lifted up portions of metal shaft.

Source: (Courtesy Moyco Union Broach, York, PA, USA.)

Low-speed engine-driven instruments

Burs

Specialized burs are available for endodontic access cavities. These burs are used in both high-speed and slow-speed handpieces and are manufactured from stainless steel. (Access cavity preparation and used materials are described in detail in Chapter 7.)

Gates-Glidden drills

Gates-Glidden (GG) drills (Fig. 8.24) have been used for almost 150 years without noteworthy design changes. GG drills are typically used to enlarge coronal canal areas.[107] When misused, GG drills can dramatically reduce radicular wall thickness.[155,213]

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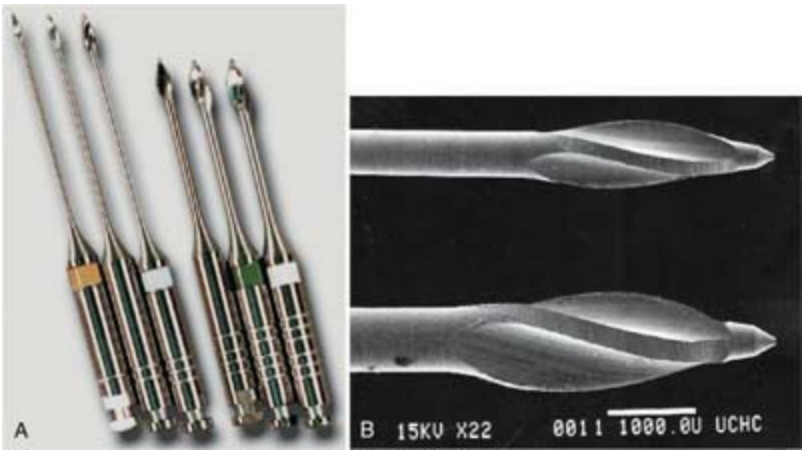

FIG. 8.24 Various Gates-Glidden (GG) burs made of stainless steel (**A**) and scanning electron micrograph (**B**, working tip).

A) Close-up of gates-glidden burs with long and short bodies.

B) Micrograph of gates-glidden bur shows an axially twisted top with a conical tip.

Source: (***A***, From Johnson WT: *Color atlas of endodontics*, St Louis, 2002, Saunders.)

GG instruments are available in six sizes and various lengths, by several manufacturers. Each instrument has a long, thin shaft with parallel walls and a short, oval cutting head with safety tips (see Fig. 8.24, *B*); these drills with tip diameters of 0.5 to 1.1 mm are produced in stainless steel and NiTi varieties. Because of their design and physical properties,[62] GG drills are side-cutting instruments; they can be used to cut dentin as they are withdrawn from the canal (i.e., on the outstroke) and should be used only in the straight portions of a canal.[456]

Two procedural sequences have been proposed: With the step-down technique, the clinician starts with a large drill and progresses to smaller ones; conversely, with the step-back technique, the clinician starts with a small drill and progresses to larger ones. Either technique efficiently opens root canal orifices and works best when canals exit the access cavity without severe angulations.

When used adequately, GG instruments are inexpensive, safe, and clinically beneficial tools. However, high speeds excessive pressure, an incorrect angle of insertion, and the use of GG instruments to aggressively drill into canals have resulted in mishaps such as strip perforation.

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Peeso reamers and similar drills

Peeso reamers and other drills are occasionally used in root canal preparation for coronal flaring or during postpreparation. At this point, these drills are manufactured mainly from stainless steel and commonly by milling blanks. Peeso drills are available with cutting and noncutting tips but should be used with caution to avoid excessive preparation and thinning of radicular dentin walls.[8] Materials and clinical procedures for post space generation are described in Chapter 23.

Engine-driven instruments for canal preparation

Instrument types

Engine-driven instruments for root canal preparation made of stainless steel have been in use for more than half a century—the first decades mainly in handpieces that permitted reciprocation (alternating clockwise-counterclockwise motion). The major two problems with this type of instrument were canal transportation and file fracture. This changed with the advent of NiTi rotaries in the early 1990s; the much more flexible alloy allowed continuous rotation and reduced both canal preparation errors and instrument fracture compared with earlier engine-driven techniques.

Currently more than 100 types of rotary instrument systems are marketed, and more continue to be developed.[148] The instruments vary greatly in terms of design, alloy used, and recommended cutting movement (Table 8.1). Various built-in features may help prevent procedural errors, increase efficiency, and improve the quality of canal shaping. For example, a longer pilot tip may guide the instrument and help to stay centered in the canal long axis.

Table 8.1

Grouping of Instruments According to Their Mode of Cutting and Manufacturer Details

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Group	Enlargement Potential	Preparation Errors	Fracture Resistance	Clinical Performance
I: ProFile,[1] ProSystem GT, GTX,[1] Quantec,[2] Pow-R,[3] Guidance,[4] K3[2] LightSpeed var.*[,2]	+, Depending in sizes, often time consuming	++, Low incidence, usually <150 μm canal transportation	±, Fatigue +, Torsional load, depending on system	++, Good, depending on treatment conditions. No difference between instruments shown so far, except for inexperienced clinicians, who perform better with landed instruments
II: ProTaper var.,[1] RaCe,[5] Hero 642,[6] FlexMaster,[7] Mtwo,*[,7] Sequence,**[,8] Alpha[9]... ProFile Vortex[1] Twisted File[1]	±, Good with use of hybrid techniques	±, Overall more demanding in clinicians' ability	+, Fatigue ±, Torsional load, depending on taper, handling	
III: EndoEZE AET[10], WaveOne,[1] Reciproc[7] OneShape[6] SAF, TRUShape,[1] TruNatomy,[1] XP-Shaper,[8] XP-Finisher[8]	Limited	Varies EndoEZE AET—WaveOne, Reciproc+	Varies +, With WaveOne, Reciproc	Varies

Group I consists of radial-landed instruments with reaming action. Group II instruments are such with triangular cross section and cutting action, while Group III is made up of atypical instruments with unusual geometry, movement, or sequence

Manufacturers:

[1]Dentsply TSirona, York, PA, USA/Dentsply Maillefer, Ballaigues Switzerland.

[2]Analytic Endodontics, Orange, CA, USA/SKavo Kerr, Orange, CA, USA.

[3]Moyco Union Broach, York, PA, USA.

[4]Guidance Endo, Albuquerque, NM, USA.

[5]FKG, La Chaux-De-Fonds, Switzerland.

[6]MicroMega, Besançon, France.

[7]VDW, Munich, Germany ** originally by Sweden & Martina, Padova, Italy.

[8]BrasselerUSA, Savannah, GA * instruments made by FKG.

[9]Brasseler, Lemgo, Germany.

[10]Ultradent, South Jordan, UT, USA.

Another direction of instrument development is the prevention of instrument fractures (Fig. 8.25). There are several ways to modify an instrument to make it less likely to fracture—for example, increasing the core diameter will increase torsional resistance. Another approach is to use a

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torque-limiting motor (see later). Alternatively, a zero taper or nearly parallel and fluted working portion of the file may be provided for curved canals so that the apical portion of the canal can be enlarged without undue file stress and compression of debris. Changing continuous rotation to reciprocation motion when applied to NiTi rotary instruments has been successful in preventing threading-in and instrument fractures in general. Another direction of instrument development is to improve shaping as it relates to circumferential root canal wall contact, also described as conforming shaping. One example for this strategy is a file manufactured from an expandable and flexible hollow NiTi tube, the so-called Self-adjusting File (SAF; ReDent-Nova, Raanana, Israel) (Fig. 8.26).

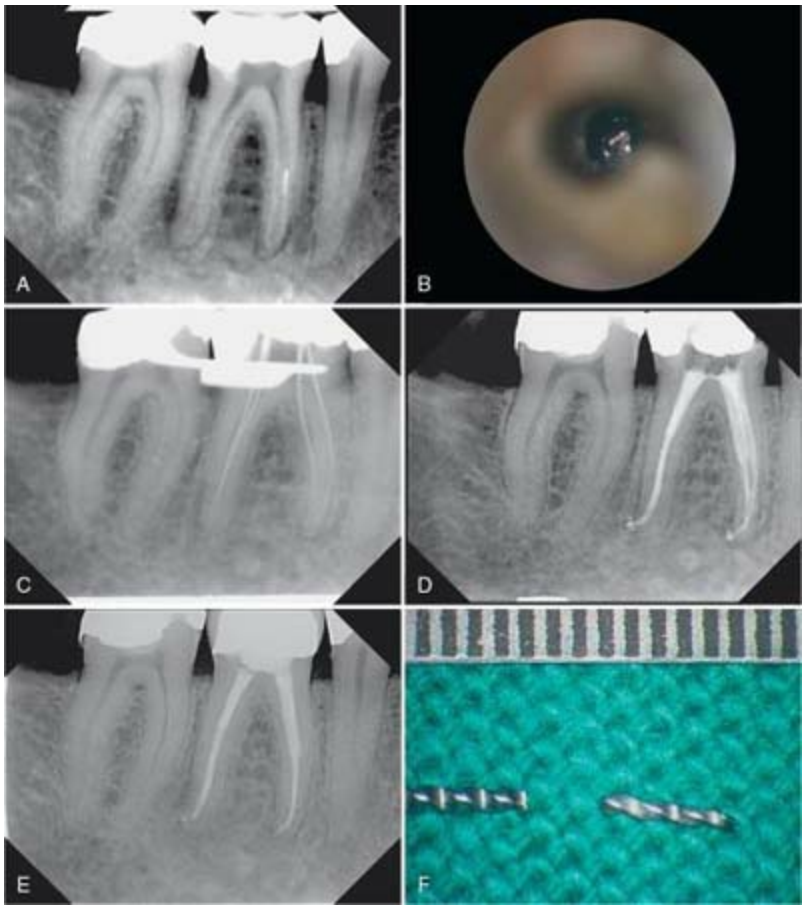

FIG. 8.25 Removal of a fractured NiTi instrument from a mesiolingual canal of a mandibular molar. **A,** Fragment located in the middle third of the root. **B,** Clinical aspect of the fragment after enlargement of the coronal third of the root canal with modified Gates-Glidden drills, visualized with an operating microscope (×25). **C,** Radiograph taken

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after removal of the fragment; four hand files have been inserted into the canals. **D,** Final radiograph shows slight widening of the coronal third of the mesiolingual canal and fully sealed canal systems. A full crown was placed immediately after obturation. **E,** Recall radiograph 5 years after obturation shows sound periradicular tissues. **F,** Removed fragment and separated file (gradation of ruler is 0.5 mm).

A) Radiograph of tooth shows a white radiopaque patch on the crown with a radiolucent outline of titanium-nickel file in the right canal.

B) Micrograph of an exposed root canal shows a silver speck in the center.

C) Radiograph of tooth shows a white radiopaque patch on the crown with a radiolucent outline of four hand files in the canals. A radiopaque outline of clamp is visible.

D) Radiograph of tooth shows a white radiopaque crown, radiolucent neck and radiopaque canals.

E) Radiograph of tooth shows a white radiopaque crown, neck and canals.

F) Close-up of the removed laterally segmented hand file.

FIG. 8.26 The SAF instrument. The instrument is made as a hollow, thin NiTi lattice cylinder that is compressed when inserted into the root canal and adapts to the canal's cross section. It is attached to a vibrating handpiece. Continuous irrigation is applied through a special hub on the side of its shank.

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An image of two self adjusting files show sharp pointed and conical tips composed of nickel-titanium lattice.

Source: (Courtesy ReDent-Nova, Raanana, Israel.) Insert shows the abrasive surface of the instrument.

More recently, a substantial S-shape was imposed onto a flexible NiTi rotary. This strategy provides an adaptable, larger envelope of motion while maintaining a limited maximum flute diameter (TRUShape, Dentsply Sirona; XP Shaper, Brasseler, Savannah, GA, USA). Marketed files vary greatly in terms of their specific design characteristics, such as tip sizing, taper, cross section, helix angle, and pitch (see Fig. 8.13). Some of the early systems have been removed from the market or relegated to minor roles.

Most instruments described in the following section are manufactured by multiaxial grinding or micromilling processes, although some are produced by laser etching and yet others by plastic deformation under specific heating and cooling processes or electric discharge machining.

Many variables and physical properties influence the clinical performance of NiTi rotaries.[239,329,393,449] Clinical practice has generated much of what is known about NiTi instruments, including reasons for instrument fracture and the application of specific instrument sequences; NiTi rotary instruments have substantially reduced the incidence of relevant canal shaping errors.[89]

Table 8.1 and the following sections describe the instrument groups most widely used for root canal preparation at this point in time. Typical clinical strategies apply to all NiTi rotary instruments, regardless of the specific design or brand. However, three design groups will be analyzed separately next: group I, instruments designed for passive preparation; group II, rotary instruments designed for active cutting; and group III, unique designs that do not fit in either group I and II.

Group I: Passive preparation; presence of radial lands.

The first commercially successful rotary instruments were ProFile (Dentsply Sirona), Lightspeed (marketed in its current form by Kavo Kerr, Orange, CA, USA), and GT rotaries (Dentsply Sirona), and have in common a cross section with so-called radial lands (Fig. 8.27, red arrow). These are created by three round excavations, also known as the U-shape. The design of the instrument tip and also the lateral file surface (radial land) guide the file as it

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progresses apically. This makes rotaries listed in group I fairly safe regarding preparation errors. On the other hand, it results in a reaming action rather than cutting of dentin, and this makes them less efficient. Moreover, the smear layer produced with radial-landed rotaries is different in consistency and amount compared with the debris and smear created by cutting files.[318]

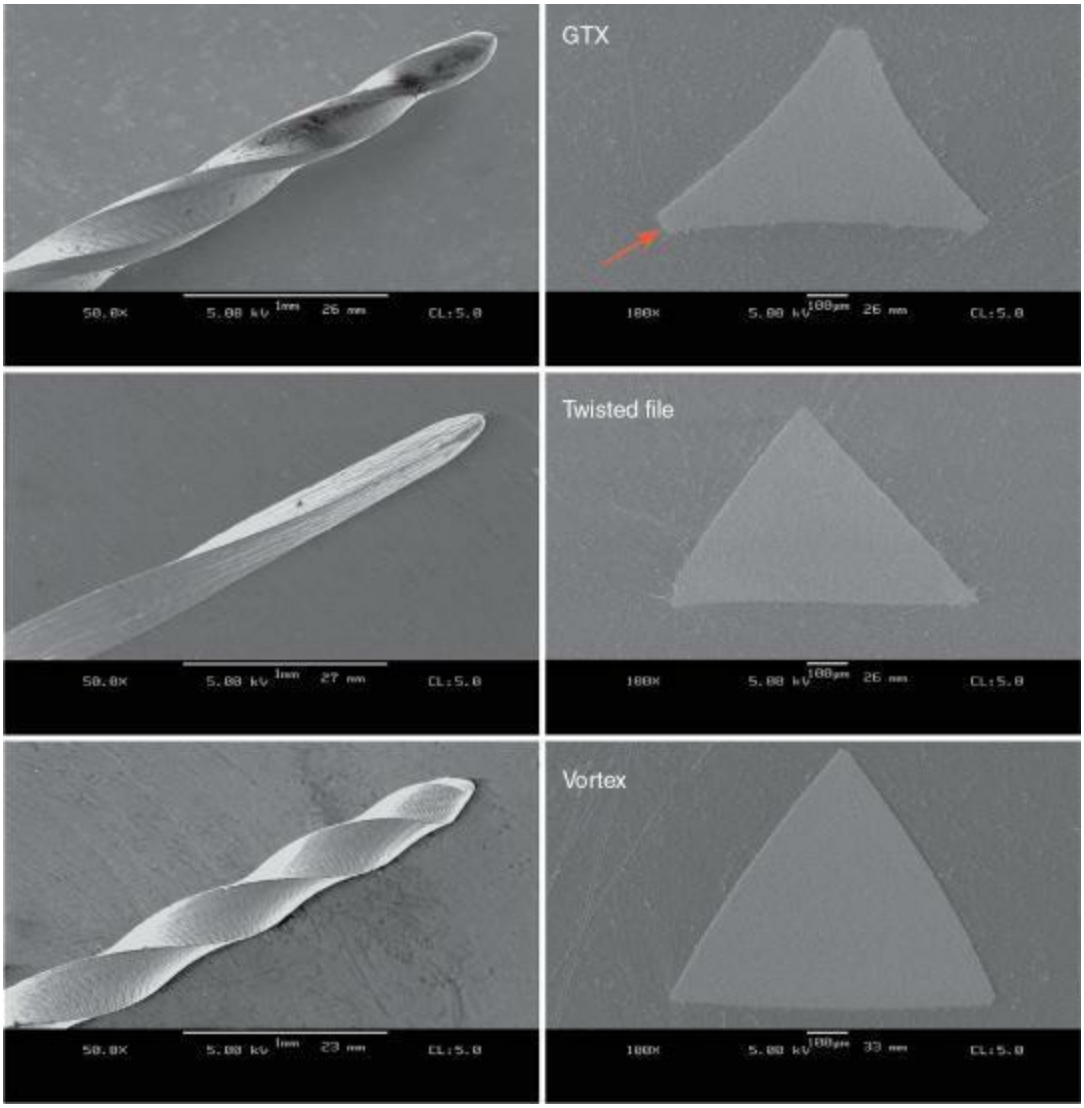

FIG. 8.27 Scanning electron micrographs (SEM) of current nickel-titanium rotary instruments, seen laterally (*left panel*, ×50) and in cross sections (*right panel*, ×100). Note the radial landed cross section of the *GTX* instrument (*red arrow*).

A) Micrograph of a nickel titanium instrument shows a flatly textured loose spiral surface body with a hemispherical tip. Cross-section of GTX shows a triangular shape with inward sides and flat corners. An arrow points to a flat

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corner.

B) Micrograph of a nickel titanium instrument shows axially textured surface body with a sharp conical tip. Cross-section of twisted file shows a triangular shape with straight sides and two sharp and one irregular corner.

C) Micrograph of a nickel titanium instrument shows sharp textured tight spiral surface body with a conical tip. Cross-section of vortex shows a triangular shape with outward sides and partially sharp corners.

LightSpeed.

The LightSpeed file, developed by Drs. Steve Senia and William Wildey in the early 1990s and later known as *LS1*, was introduced as an instrument with a thin, noncutting shaft and short anterior cutting head. The same design principles apply to the subsequently marketed LSX instrument (Kavo Kerr) that is manufactured not by milling but by a stamping process. A full set consists of 25 LightSpeed LS1 instruments in sizes #20 to #100, including half sizes (e.g., 22.5, 27.5); LSX does not have half sizes, and a set includes sizes #20 to #80.

ProFile.

The ProFile system (Dentsply Sirona) was introduced by Dr. Ben Johnson in 1994. ProFile instruments have increased tapers compared with conventional hand instruments. The tips of the original ProFile Series 29 rotary instruments had a constant proportion of diameter increments (29%). Later, a ProFile series with ISO-sized tips (Dentsply Maillefer, Ballaigues, Switzerland) was developed and marketed in Europe.

Cross sections of a ProFile instrument show a U-shape design with radial lands (Fig. 8.28) and a parallel central core. Lateral views show a 20-degree helix angle, a constant pitch, and bullet-shaped noncutting tips (see Fig. 8.12). Together with a slightly negative rake angle, this configuration facilitates a reaming action on dentin rather than cutting. ProFile instruments shaped canals without major preparation errors in a number of in vitro investigations.[66,67,451,452]

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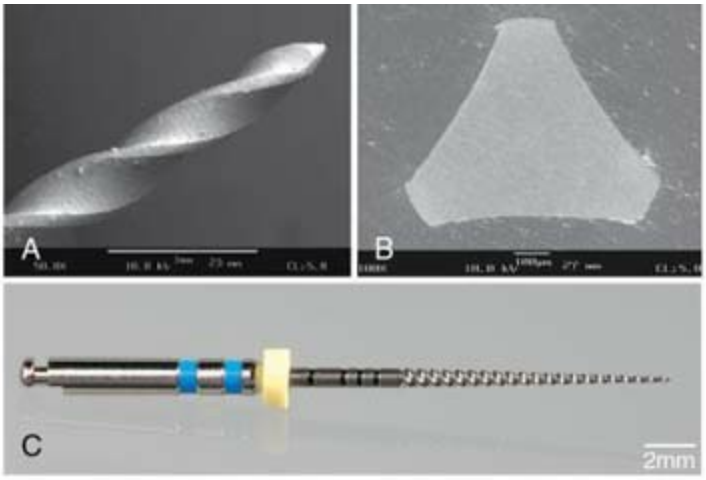

D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
Orifice Shapers: 6	20–80	10; from 60: 20	150–350, low apical force, torque to fracture and working torque dependent on instrument size	19 mm
ProFile .06: 6	15–40	5		21 mm, 25 mm, some 31 mm
ProFile .04: 9	15–90	5; from #45: 15; from #60: 30		
ProFile .02: 6	15–45	5		
Profile Series 29	13–100	Varies, 29%		21 mm, 25 mm

FIG. 8.28 Design features of a ProFile instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

A) Micrograph of a ProFile instrument shows a flatly textured tight spiral surface body with a conical tip.

B) Cross-section of ProFile instrument shows a triangular shape with inward sides and flat corners.

C) Close-up of a ProFile instrument shows a spiral surface body with a conical tip.

D) The data for number of instruments, tip sizes, size increments, r.p.m. (recommended) and lengths are as follows:

Orifice Shapers (6): 20 to 80: 10; from 60 is to 20: 150 to 350 (low apical force, torque to fracture and working torque dependent on instrument size): 19 millimeters ProFile.06 (6): 15 to 40: 5: blank: 150 to 350 (low apical force, torque to fracture and working torque dependent on instrument size): 21, 25, some 31 millimeters.

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ProFile.04 (9): 15 to 90: 5; from number 45 is to 15; from number 60 is to 30: blank: blank.

ProFile.02 (6): 15 to 45: 5: blank: blank Profile Series 29: 13 to 100: varies 29 percent: blank: 21 millimeters, 25 millimeters.

GT and GTX files.

The Greater Taper, or GT file, was introduced by Dr. Steve Buchanan in 1994. This instrument incorporates a radial landed cross-sectional design and was marketed as ProFile GT (Dentsply Sirona). The instruments were available in four tapers (.06,.08,.10, and.12), and the maximum diameter of the working part was 1 mm. The design limited the maximum shank diameter and decreased the length of the cutting flutes with increasing taper. The instruments had a variable pitch and a growing number of flutes in progression to the tip; the apical instrument diameter was 0.2 mm.

K3.

In a sequence of design developments by their inventor, Dr. McSpadden, the Quantec 2000 files were followed by the Quantec SC, the Quantec LX, and the current K3 system (all by Kavo Kerr Endo). K3, similar to ProFile instruments, includes instruments with.02,.04, and.06 tapers. The most obvious difference between the Quantec and K3 models is the K3's unique cross-sectional design: a slightly positive rake angle for greater cutting efficiency, wide radial lands, and a peripheral blade relief for reduced friction. Unlike the Quantec, a two-flute file, the K3 features a third radial land to help prevent threading-in. K3 features a round safety tip, but the file is about 4 mm shorter than other files (although it has the same length of cutting flutes) because of the so-called Axxess handle. The instruments are coded by ring color and number.

In the lateral aspect, the K3 has a variable pitch and variable core diameter, which provide apical strength. Manufacturing this intricate design can result in some metal flash.

Summary.

Radial landed rotary instruments are considered very safe, even when accidentally taken beyond the confines of the root canal. Fracture resistance

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to torsional loading and cyclic varies depending on specific instrument design. The limited cutting efficacy of these files was perceived as a downside and is a reason that the market share has diminished.[50] However, their track record in clinic and research continue to support the use of rotaries listed in group I.

Group II: Active cutting; triangular cross section.

Rotary instruments in group II all have a more active cutting flute design in common and have the largest market share. Radial lands are absent (see Fig. 8.13), and this fact results in a higher cutting efficacy. This translates to a higher potential for preparation errors, in particular when the instrument is taken through the apical foramen, thus eliminating the guide derived from the noncutting tip.

ProTaper Universal, Gold.

The ProTaper system originally comprised six instruments: three shaping files and three finishing files. This set is now complemented by two larger finishing files and a separate set of three retreatment rotaries. ProTaper instruments were designed by Drs. Cliff Ruddle, John West, and Pierre Machtou. In cross sections, ProTaper shows a convex triangle with sharp cutting edges and no radial lands. The cross section of finishing files F3, F4, and F5 is slightly relieved for increased flexibility. The three shaping files have tapers that increase coronally, and the reverse pattern is seen in the five finishing files.

Shaping files Nos. 1 and 2 have tip diameters of 0.185 mm and 0.2 mm, respectively, 14-mm-long cutting blades, and partially active tips. The finishing files (F1 to F5) have tip diameters of 0.2, 0.25, 0.3, 0.4, and 0.5 mm, respectively, between D_0 and D_3, and the apical tapers are.07,.08,.09,.05, and.04, respectively. The finishing files have rounded noncutting tips.

Two aspects of handling have been emphasized for ProTaper. The first is the preparation of a glide path, either manually[321] or with special rotary instruments.[47] An enlargement to a size approaching the subsequent rotaries' tips, at least larger than the file's core diameter, prevents breakage and allows assessment of the canal size.[321] This means that a glide path should

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correspond to a size #15 or 20. Glide path preparation is currently recommended for most NiTi instruments.

The second specific recommendation for ProTaper use is the use of a more laterally directed “brushing” working stroke. Such a stroke allows the clinician to coronally direct larger files away from danger zones and counteract any “threading-in” effect.[53] Brushing strokes should be applied judiciously and away from the furcation in molars to avoid thinning of radicular structure.

In a study using plastic blocks, ProTaper created acceptable shapes more quickly than GT rotary, ProFile, and Quantec instruments[502] but also created somewhat more aberrations. A study using micro-CT showed that the original ProTaper file created consistent shapes in constricted canals, without obvious preparation errors, although wide canals may be insufficiently prepared with this system.[338]

A newer version, called ProTaper Next, was introduced in 2013. Current research suggests that mechanical properties of these instruments, manufactured from M-Wire, are better than ProTaper Universal.[23,121,322]

HERO 642, HERO Shaper.

Several systems in group II (see Table 8.1) were designed with positive rake angles, which provide greater cutting efficiency. HERO instruments (MicroMega, Besançon, France) are an example, the original version was known as *HERO 642* (the name *HERO* is an acronym for *h*igh *e*lasticity in *ro*tation); the name has now changed to HERO Shaper, with few apparent differences in the instrument design.

Cross sections of HERO instruments show geometries similar to those of an H-file without radial lands. Tapers of.02,.04, and.06 are available in sizes ranging from #20 to #45. The instruments are relatively flexible but maintain an even distribution of force into the cutting areas.[460,461] HERO instruments have a progressive flute pitch and a noncutting passive tip, similar to other NiTi rotary systems. The instruments are coded by handle color.

RaCe, BioRaCe, BT RaCe.

The RaCe file has been manufactured since 1999 by FKG and was later distributed in the United States by Brasseler. The name, which stands for

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r*eamer with* a*lternating cutting* e*dges*, describes just one design feature of this instrument. Light microscopic imaging shows flutes and reverse flutes alternating with straight areas; this design is aimed at reducing the tendency of files to thread into a root canal. Cross sections are triangular or square for.02 instruments with size #15 and #20 tips. The lengths of cutting parts vary from 9 to 16 mm.

The surface quality of RaCe instruments has been modified by electropolishing, and the two largest files (size #35,.08 taper and size #40,.10 taper) are also available in stainless steel. The tips are round and noncutting, and the instrument handles are color-coded and marked by milled rings. RaCe instruments have been marketed in various packages to address small and large canals; recently they are sold as BioRaCe, purportedly to allow larger preparation sizes, with an emphasis on the use of.02 tapered instruments. A new variant, BT RaCe was recently introduced and incorporates different tip designs, as well as different sequences.

Few results of in vitro experiments comparing RaCe to other contemporary rotary systems are available;[384,385] canals in plastic blocks and in extracted teeth were prepared by the RaCe system with less transportation than with ProTaper files.[384] In another study, ProTaper and RaCe performed similarly when canals were prepared to an apical size #30.[313] As with any rotary system, this file may be included in a hybrid hand-rotary technique. BioRace instruments prepared S-shaped canals in plastic blocks (to size #40) similarly to ProTaper and MTwo but were superior when combined with S-Apex.[56]

EndoSequence.

The EndoSequence rotary instrument is produced by FKG in Switzerland and marketed in the United States by Brasseler. This instrument adheres to the conventional length of the cutting flutes, 16 mm, and to larger tapers,.04 and.06, to be used in a crown-down approach. The overall design, including the available tapers and cross sections, is thus similar to many other files; however, the manufacturer claims that a unique longitudinal design called *alternating wall contact points* (ACP) reduces torque requirements and keeps the file centered in the canal. It also has varying comparatively small helical angles. Another feature of the Sequence design is an electrochemical treatment after manufacturing, similar to that of RaCe files, resulting in a

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smooth, polished surface. This is believed to promote better fatigue resistance; hence a rotational speed of 600 rpm is recommended for EndoSequence.[231] Most in vitro results, however, suggest that EndoSequence is not superior to other files in cyclic fatigue resistance.[195,243,353]

Twisted file.

In 2008, SybronEndo, now Kavo Kerr Endo, presented the first fluted NiTi file manufactured by plastic deformation, a process similar to the twisting process that is used to produce stainless steel K-files: the Twisted File (TF). According to the manufacturer, a thermal process allows twisting during a phase transformation into the so-called R-phase of nickel-titanium. The instrument is currently available with size tip from 25 to 50 and in tapers from.04 to.12.

The unique production process is believed to result in superior physical properties; indeed, early studies suggested significantly better fatigue resistance when size #25.06 taper TFs were compared with K3 instruments of the same size GTX instrument.[144] Moreover, as determined by bending tests according to the ISO norm for hand instruments, 3630-1, TFs size #25.06 taper were more flexible than ProFiles of the same size.[143] Others found similar levels for fatigue resistance for TF and Profile of similar size.[243] A more recent development for TF is the use of an electric motor that allows different file movement, both continuous rotation and reciprocation, depending on the clinical situation (TF Adaptive, Kavo Kerr).

Profile Vortex.

Profile Vortex instruments are manufactured from NiTi. Two versions are on the market—one made from M-Wire and another from so-called blue wire (Vortex Blue, which showed greater resistance to cyclic fatigue and increased torque resistance) and have varying helical angles to counteract the tendency of nonlanded files to thread into the root canal. Vortex instruments are recommended to be used at 500 rpm; higher rotational speed results in less torque generated.[35] Canal preparation with ProFile Vortex in vitro was similar to other rotary instruments.[75,493] Vortex instruments are available in sizes 15 to 50 and in.04 and.06 tapers.

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MTwo.

This instrument entered the European market in 2004. It possesses a two-fluted S-shaped cross section. The original strategy allowed for three distinct shaping approaches after the use of a basic sequence with tip sizes from #10 to #25, and tapers ranging from.04 to.06. Subsequent enlargement was meant to either create apical sizes up to #40.04, alternatively to #25.07 or to larger apical sizes with so-called apical files. MTwo is a well-researched and cutting-efficient instrument; clinically it is an example for the so-called single length technique. Canal shapes with MTwo were similar to other contemporary root canal instruments, either in rotation or reciprocation.[72]

Edge files.

A selection of instruments in designs that are similar to other designs, made primarily from heat-treated NiTi alloys, are available from Edge Endo (Albuquerque, NM, USA). There is comparatively limited research available for the individual files but extended fatigue life has been reported for them, at least at room temperature.[22]

Summary.

The market share for rotary files without radial lands continues to expand because of perceived higher efficacy. The overall incidence of clinically relevant preparation errors (see later for details) appears to be low, in spite of more aggressive cutting by files without radial lands. Instrument fractures remain a concern as does the tendency of continuously rotating instruments to thread or pull into the canal, specifically as working length (WL) is approached.

Group III, atypical instruments

WaveOne, Reciproc, Gold, Blue.

A way to mitigate problems with continuous rotation (e.g., taper lock, fatigue fracture, threading-in) is to replace continuous rotation by reciprocation action. A case report[496] described this approach using ProTaper instruments. Based on experiments evaluating the maximal rotational angle before plastic deformation for the selected instrument, a forward angle of 144 degrees,

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followed by a reverse rotation of 72 degrees, was recommended.[496]

Subsequently, two instruments specifically designed for reciprocation were brought to the market (WaveOne, Dentsply Sirona) and Reciproc (VDW, Munich, Germany, currently not available in the United States). WaveOne instruments are available in three tip sizes, #21, #25, and #40, with tapers of.06 and.08, respectively. Corresponding sizes for Reciproc are tips of #25, #40, and #50, with tapers of.08,.06, and.05, respectively. Both instruments feature variable tapers that are largest toward the tips. The main WaveOne cross section is triangular, similar to ProTaper, while Reciproc is a two-fluted file with a design similar to MTwo (VDW).

Special motors are used for both systems to provide reciprocation action with alternating counterclockwise and clockwise rotations of about 150 to 170 and 30 to 50 degrees, respectively.[134]

Both files are machined with left-leaning flutes, and therefore the cutting direction for both is clockwise. One problem that may occur with this design is the transportation of dentin debris into the apical area, rather than moving debris coronally. There is mixed evidence for this phenomenon[73,110] in vitro. Clinically, frequent careful cleaning of the cutting blades with a moist gauze is recommended.

Shaping ability of these systems seems, according to current in vitro data, similar to established systems with continuous rotation.[72,278,469]

Self-adjusting File.

The SAF (ReDent-Nova) represented a completely different approach, both in file design and mode of operation.[280] It is a cylindrical, hollow design with a thin-walled NiTi lattice displaying a lightly abrasive surface (see Fig. 8.26). An initial glide path to a #20 K-file allows insertion of the SAF file. The file is operated with a handpiece that generates in-and-out movements (4000 per minute) and 0.4 mm amplitudes.

In vitro data for this system suggest that indeed more wall contact is made compared with rotary files,[336] resulting in better débridement and superior antimicrobial efficacy.[415] Shaping quality is also on par with rotary instruments.[336]

TRUShape.

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Two strategies have recently emerged in file design, which are both present in the TRUShape (Dentsply Sirona) instrument: so-called conforming shaping and a limited maximum flute diameter. The strong S-shaped longitudinal design allows TRUShape to expand and contract during rotation, similar to what had been described for the SAF, in an attempt to increase wall contact. TRUShape is marketed in sizes #20, #25, #30, and #40, all with a maximum fluted diameter of 0.8 mm, thus limiting coronal dentin removal. Used in continuous rotation at 300 rpm, TRUShape has been shown to create adequate shapes in vitro during initial canal preparation in molars[328] and premolars.[216] Specifically in root canal retreatment, the ability of TRUShape files to create more canal wall contact has attracted attention.

Further design developments led to the TruNatomy system (Dentsply Sirona), which includes a dedicated Orifice Modifier, Glide Path file, and three shaping instruments, as well as a special irrigation needle and conforming gutta percha. TruNatomy is operated at 500 rpm and 1.5 Ncm and with gentle stroking motion.

XP-Shaper, XP-Finisher.

A similar design is incorporated in the XP instruments. Both have strong curves in the longitudinal aspect and a small taper, so that the maximum fluted diameter is limited. The files are made of a NiTi solution that changes its conformation in the transition from room to body temperature, which in turn leads to some stiffening and specific preparation outcomes. The finisher is thought of as an irrigation enhancement device and is run at about 800 rpm. Initial reports suggest that using the XP-Shaper resulted in acceptable canal preparation[28] and that the use of the finisher had débridement potential that is similar or better than with other shaping and cleaning strategies.[122]

Sonic and ultrasonic shaping instruments.

An alternative way of instrumenting root canals was introduced when clinicians became able to activate files by electromagnetic ultrasonic energy. Piezoelectric ultrasonic units are also available for this purpose (Fig. 8.29). These units activate an oscillating sinusoidal wave in the file with a frequency of about 30 kHz.

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FIG. 8.29 Example of an ultrasonic unit (Neutron P5 piezoelectric).

A Neutron P5 piezoelectric ultrasonic device consisting of a control unit, a slim hand piece comprising of ceramic transducer and a tip or a file.

Source: (Courtesy Satelec, Merignac Cedex, France.)

Two types of units, ultrasonic and sonic, are marketed. Ultrasonic devices, which operate at 25 to 30 kHz, include the magnetostrictive Cavi-Endo (Dentsply Caulk, Milford, DE, USA), the piezoelectric ENAC (Osada, Tokyo), the EMS Piezon Master 400 (Electro Medical Systems, Vallée de Joux, Switzerland), and the P5 Neutron (Satelec, Merignac Cedex, France).

Sonic devices, which operate at 2 to 3 kHz, include the Sonic AirMM 1500 (Micro Mega, Prodonta, Geneva, Switzerland), the Megasonic 1400 (Megasonic Corp, House Springs, MO, USA), and the Endostar (Syntex Dental Products, Valley Forge, PA, USA).

The piezoelectric transducer transfers more energy to the file than does the magnetostrictive system, making it more cutting efficient. The file in an ultrasonic device vibrates in a sinus wavelike fashion. A standing wave has areas with maximal displacement (i.e., antinodes) and areas with no displacement (i.e., nodes). The tip of the instrument exhibits an antinode. If powered too high, especially with no contact with the canal wall, the instrument may break because of the intense vibration. Therefore, files must be used only for a short time, must remain passive within the canal, and the

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power must be controlled carefully. The frequency of breakage in files used for longer than 10 minutes may be as high as 10%, and the breakage normally occurs at the nodes of vibrations.[9] Ultrasonic devices have been linked to a higher incidence of preparation errors and to reduced radicular wall thickness.[260,277,512]

Summary.

An abundance of NiTi systems is currently on the market, Table 8.1 lists these instruments systematically and illustrates some of the most relevant properties. Most systems included in Table 8.1 have files with tapers greater than the.02 stipulated by the ISO norm, while some have variable tapers and yet others have canal-conforming shapes and limited maximum flute diameters. Differences exist in tip designs, cross sections, and manufacturing processes. In vitro tests continue to identify the effect of specific designs on shaping capabilities and fracture resistance. An attractive goal is the instrumentation of more canal surface, in an effort to make biofilm more susceptible to subsequent chemical disinfection. At the same time, instrumentation paradigms that conserve more dentin appear desirable to promote long-term function.[154] Meaningful differences in clinical outcomes in regard to various specific design variations have yet to be ascertained.

Motors

Motors for rotary instruments have become more sophisticated since the simple electric motors of the first generation in the early 1990s (Fig. 8.30, *A*). Electric motors with gear reduction are most suitable for rotary NiTi systems, because they ensure a constant rpm level and constant torque. They can also be programmed to provide alternative rotation patterns—for example, reciprocation with freely selectable angles of rotation.[134] Electric motors often have presets for rpm and torque and are capable of delivering torques much higher than those required to break tips. Some authors believe that torque-controlled motors (see Fig. 8.30, *B–D*) increase operational safety.[142] Others have suggested that such motors may mainly benefit inexperienced clinicians.[499] These motors probably do not reduce the risk of fracture caused by cyclic fatigue, and even if the torque is below the fracture load at D_3, a fracture at a smaller diameter (D_2) is still possible.

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FIG. 8.30 Examples of motors used with rotary nickel-titanium endodontic instruments. **A,** First-generation motor without torque control. **B,** Fully electronically controlled second-generation motor with sensitive torque limiter. **C,** Frequently used simple torque-controlled motor. **D,** Newer-generation motor with built-in apex locator and torque control.

Set of four images shows types of motors with hand-held dental drills labeled as A through D.

To complicate matters further, a differential exists between torque at failure at D_3 and the working torque needed to operate an instrument effectively (Fig. 8.31 and Box 8.2).[52,329,376] In many cases, the working torque is greater than the torque required to fracture the instrument's tip.

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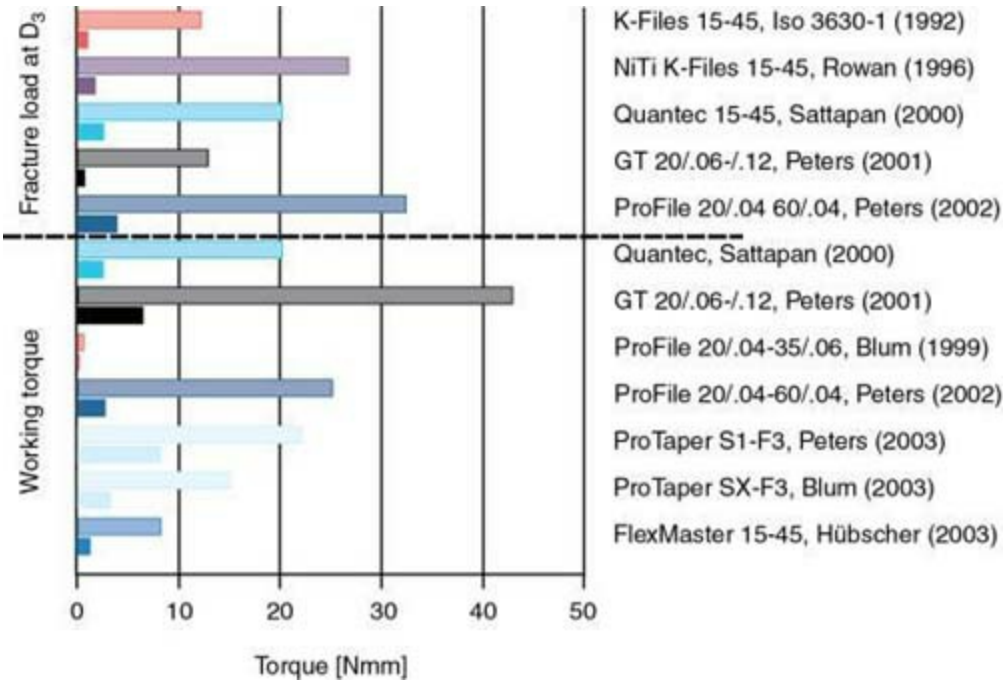

FIG. 8.31 Diagram comparing fracture loads at D_3 *(upper section of graph)* to torques occurring during preparation of root canals *(lower section of graph)*. Filled columns represent the largest file in each set, and open columns show the scores of the most fragile file (see text and Box 8.3 for details).

Plot shows torque [Newton millimeter] against working torque and fracture load at D sub 3. The horizontal axis ranges from 0 to 50 in increments of 10. The vertical axis shows working torque and fracture load at D sub 3.

The data is as follows:

Working torque:

FlexMaster 15 to 45, Hübscher (2003): 2, ProTaper SX-F3, Blum (2003): 4, ProTaper S1 to F3, Peters (2003): 8, ProFile 20/.04 to 60/.04, Peters (2002): 4, ProFile 20/.04 to 35/.06, Blum (1999): 0.5, GT 20/.06 to /.12, Peters (2001): 7, Quantec, Sattapan (2000): 3.5, ProFile 20/.04 60/.04, Peters (2002): 5, GT 20/.06 to /.12, Peters (2001): 1, Quantec 15 to 45, Sattapan (2000): 4, NiTi K-Files 15 to 45, Rowan (1996): 3 and K-Files 15 to 45, Iso 3630-1 (1992): 1.5.

Fracture load at D sub 3.:

FlexMaster 15 to 45, Hübscher (2003): 8, ProTaper SX-F3, Blum (2003): 16, ProTaper S1 to F3, Peters (2003): 23, ProFile 20/.04 to 60/.04, Peters (2002): 26, ProFile 20/.04 to 35/.06, Blum (1999): 1, GT 20/.06 to /.12, Peters (2001): 44, Quantec, Sattapan (2000): 20, ProFile 20/.04 60/.04, Peters (2002): 34, GT 20/.06 to /.12, Peters (2001): 14, Quantec 15 to 45, Sattapan (2000): 20.5, NiTi K-Files 15 to 45, Rowan (1996): 26 and K-Files 15 to 45, Iso 3630-1 (1992):

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Box 8.2

Instrument Breakage With Torsional Load (MacSpadden Factor)

For rotary instrument tips, susceptibility to breakage is governed by the quotient of torque needed to fracture divided by working torque. Simply put, the larger the value, the safer the file.

This differential between working torque and tip fracture load is especially large with files with a taper equal to or greater than.06; therefore, these files are rather ineffective in most torque-controlled motors. Some contemporary motors offer microprocessor-controlled features, for example, information about the specific instrument being used, such as the presets and the usage history, as well additional connectivity for example to an apex locator.

Some motors have built-in apex locators (see Fig. 8.30, *D*). This type of motor may be programmed to stop a rotary instrument when WL is reached or go into reverse gear. Similar changes in movement can occur in an adaptive fashion, depending on the torsional load that the instrument experiences in the root canal. The TF Adaptive motor (Kerr Endo) is an example for this technology that appeared only recently.

Some of the factors besides the motor itself that may influence the incidence of fracture in engine-driven NiTi rotary instruments are lubrication, specific instrument motion, and speed of rotation. It cannot be overemphasized that NiTi rotary instruments should only be used in canals that have been flooded with irrigants. Although lubricants such as RC-Prep (Premier, Norristown, PA) and Glyde (Dentsply Maillefer) have been recommended in the past, their benefit appears to be restricted to plastic blocks,[18] and could not be demonstrated when rotary instruments engage dentin surfaces. To the contrary, experimental data suggests that the use of gel-type lubricants fails to reduce torque values during simulated canal preparation.[55,330] Finally, because of chemical interactions between NaOCl and ethylenediamine tetra-acetic acid (EDTA),[164] alternating irrigants and using lubricants that contain EDTA may even be counterproductive. Details

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regarding irrigation solution interactions are described later in this chapter.

For instrument motion, some manufacturers recommend a pecking, up-and-down motion. This not only prevents threading in of the file; it is also believed to distribute stresses away from the instrument's point of maximum flexure where fatigue failure would likely occur.[250,348] However, such in-and-out movements did not significantly enhance the lifespan of ProFile size.04 or GT rotary instruments rotated around a 5-mm radius cylinder with a 90-degree curve.[329,333] Furthermore, large variations were noted in the lengths of the fractured segments.[203,462] This suggests that ductile fractures may originate at points of surface imperfections. Files made of more martensitic alloy tend to work better with longer sweeping or brushing strokes. The notion of "brushing" is not directly related to a paintbrush motion, since this would predispose a file to bending and subsequent fatigue.[346] Rather it should be considered as stroking against the wall away from the danger zone—the inner curvature of a curved root. Specifically, highly martensitic files may benefit from a low-impact gentle brushing with low torque setting.

Rotational speed may influence instrument deformation and fracture. Some studies indicated that ProFile instruments with ISO-norm tip diameters failed more often at higher rotational speed,[117] whereas other studies did not find speed to be a factor.[222] For Vortex, 300 rpm and 400 rpm appeared to reduce the generated torque and force; this higher rpm preset did not result in an increase of instrument fractures in vitro.[35] As mentioned before, there is a trend to using higher rotational speeds and comparatively low torques—for example, for XP-Shaper and BioRace (Brasseler) and for TruNatomy (Dentsply Sirona) instruments.[28]

Clinicians must fully understand the factors that control the forces exerted on continuously rotating NiTi instruments (Box 8.3). To minimize fractures and prevent taper lock, motor-driven rotary instruments should not be forced in an apical direction. Similarly, acute apical curves limit the use of instruments with higher tapers because of the risk of cyclic fatigue. The incidence of instrument fracture can be reduced to an absolute minimum if clinicians use data from well-designed torque and stress studies. Adequate procedural strategies, such as an adequate glide path, a detailed knowledge of anatomic structures, with avoidance of extreme canal configurations, and

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specific instrumentation sequences may also improve shaping results.

Box 8.3

Factors Governing the Potential for Nickel-Titanium Rotary Instrument Fractures

- Clinician's handling (most important)
- Combination of torsional load, bending, and axial fatigue
- Root canal anatomy
- Manufacturing process and quality

Certain procedures have evolved for removing fractured instruments from root canals (see Fig. 8.25); these are discussed in detail elsewhere in this book (see Chapter 19). Most of those methods require the use of additional equipment, such as a dental operating microscope and ultrasonic units. However, the best way to deal with instrument fracture is prevention. An understanding of the anatomy of the root canal system, together with a clear plan for selecting, sequencing, and using shaping instruments, can certainly help prevent procedural mishaps.

Steps of cleaning and shaping

Access—principles

Access cavity preparation is essential for root canal treatment and is described in detail elsewhere in this book (see Chapter 7). However, it should be emphasized at this point that mishaps during access (e.g., perforation) significantly affect the long-term outcome of a root canal–treated tooth. Overenlargement or gouging during access significantly reduces structural strength[354] and may lead to root fracture and nonrestorable conditions.

Rationally, the use of a cylindrical diamond bur, followed by a tapered fluted bur with noncutting tip and perhaps a small round bur is recommended (Fig. 8.32). A recent experiment suggested that more conservative access cavities, in particular in premolars, may increase fracture resistance and permit similar shaping outcomes.[238] The use of ultrasonically powered tips

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under magnification is aimed at achieving ideal access cavities (Fig. 8.33), including discovery and perhaps relocation of the canal orifices.

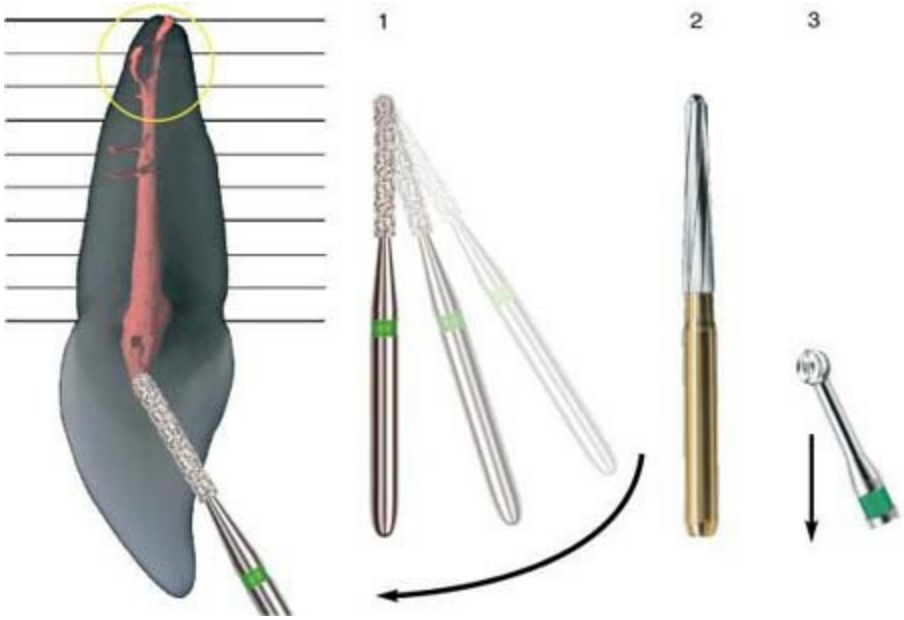

FIG. 8.32 Sequence of instruments used for optimal preparation of an access cavity (e.g., in an incisor). A parallel-sided diamond or steel bur is used to remove overlying enamel in a 90-degree angle toward the enamel surface *(1).* The bur is then tilted vertically to allow straight-line access to the root canal *(arrow).* A bur with a noncutting tip (e.g., Endo-Z bur or ball-tipped diamond bur) is then used to refine access *(2).* Overhangs or pulp horns filled with soft tissue are finally cleared with a round bur used in a brushing or pulling motion *(3).*

Set of four images show the steps of accessing a cavity in the root of a canal as numbered from 1 to 4.

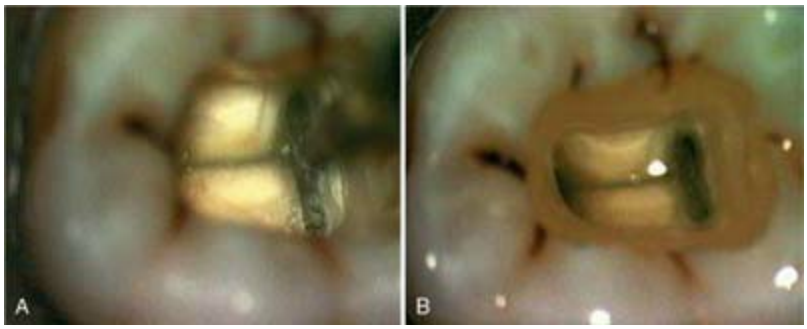

FIG. 8.33 Clinical views of an access cavity in a mandibular molar as

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seen through an operating microscope (×20). **A,** Modification with an ultrasonically activated tip (**B**).

A) Close-up of a molar shows an open crown exposing a golden filled vertical cavity.

B) Close-up of a molar shows an open crown exposing a vertical cavity with a circumference of hard filling around the crown.

Coronal modification

The extension of an access cavity into the coronal-most portion of the root canal has been called “coronal flaring” but may be more appropriately described as orifice modification. If a canal is constricted, mineralized, or difficult to access, directed enlargement of the coronal portion prior to any deep entry into the root canal is beneficial. This canal modification should be preceded by a scouting step, in which a small (e.g., size #10) K-file is passively placed several millimeters into the root canal. Tools for preflaring include Gates-Gliddens and dedicated NiTi instruments (Fig. 8.34). Specific orifice shaping rotaries have been developed that are used as a single instrument, rather than in a sequence.

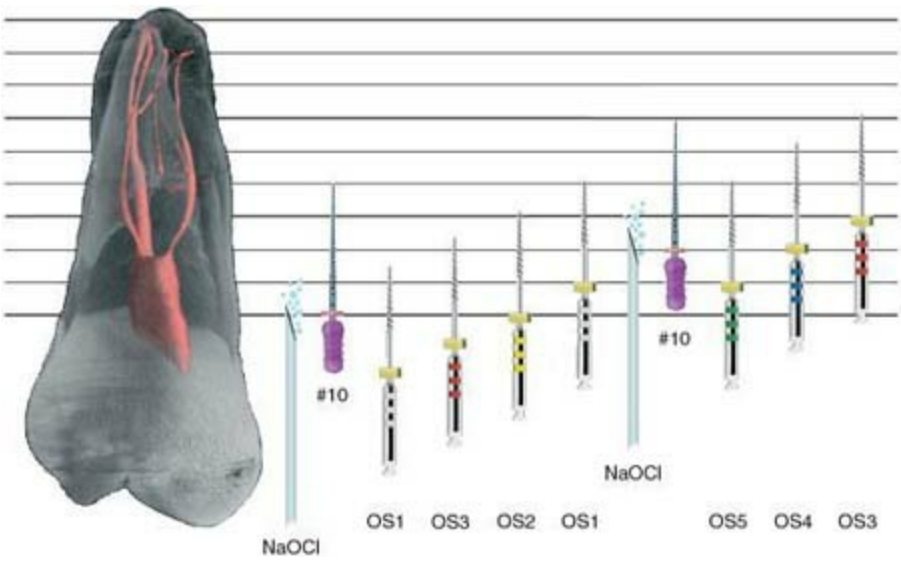

FIG. 8.34 Diagram of coronal enlargement in a more complicated maxillary posterior tooth. This maxillary molar presents several difficulties, including a narrow mesiobuccal canal that exits the pulp cavity at an angle. A possible approach in a case involving difficult

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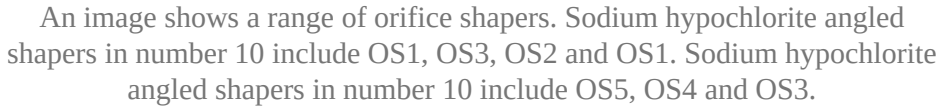
entry into the root canal system is to use a small orifice shaper *(OS1)* after ensuring a coronal glide path with a K-file. Use of a sequence of orifice shapers *(OS3 to OS1)* then allows penetration into the middle third of the root canal. Wider canals can accept a second sequence of orifice shapers. Copious irrigation and securing a glide path with a size #10 K-file are prerequisites for use of NiTi rotary instruments.


An image shows a range of orifice shapers. Sodium hypochlorite angled shapers in number 10 include OS1, OS3, OS2 and OS1. Sodium hypochlorite angled shapers in number 10 include OS5, OS4 and OS3.

The use of laterally cutting NiTi rotaries allows clinicians to modify the root canal orifice to form a receptacle for subsequent instrumentation. This is related to removal of coronal mineralization and to relocation of the canal pathway by removal of restrictive dentin. For example, a second mesiobuccal canal of a maxillary molar typically exits the pulp chamber toward the mesial under a dentin shelf and then curves towards the distal. It is this curve in a small canal that often leads to formation of a ledge, which in turn prevents shaping to length.

Preenlargement of the coronal portion of a root canal allows files unimpeded access to the apical one third and gives the clinician better tactile control in directing small, adequately precurved negotiating files (Fig. 8.35).

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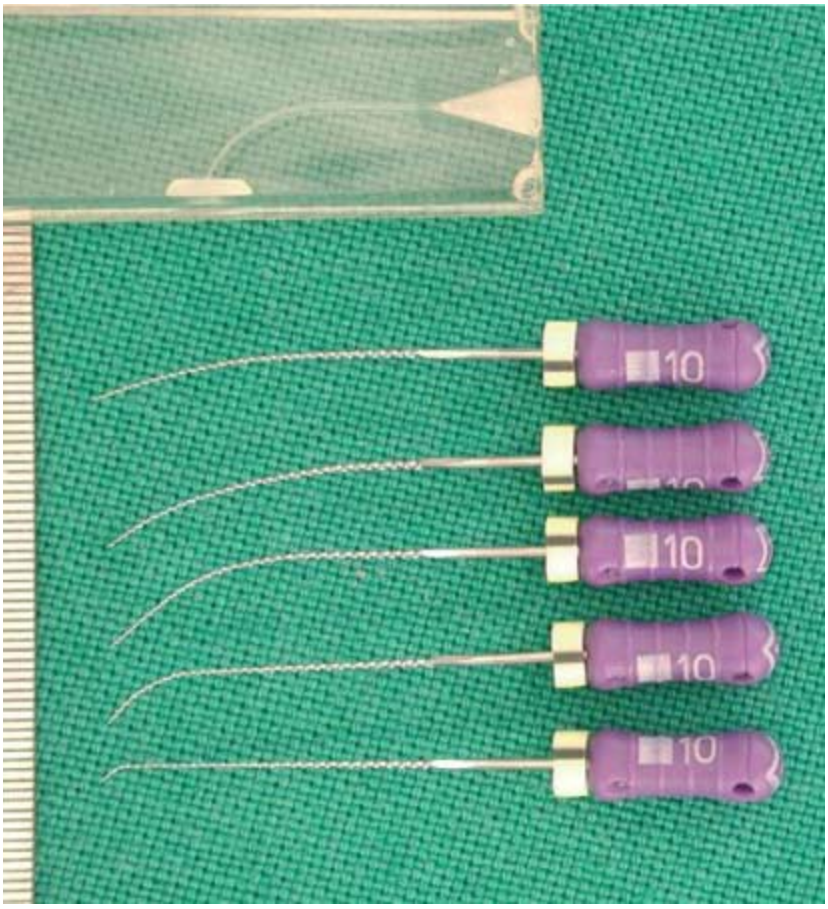

FIG. 8.35 Various precurved, stainless steel hand files for path finding and gauging. Compare the curves in the instruments to the ones in a plastic training block (gradation of ruler is 0.5 mm).

An image of five spirally textured hand files in size ten.

One of the purported benefits of orifice modification is early access of disinfecting irrigation solutions and prevention of pushing contaminated coronal debris apically. This has not been confirmed in experiments. On the other hand, a documented benefit of coronal flaring is mitigation of WL changes during canal preparation.[435]

Comparatively little is known about the efficacy of individual flaring instruments. A recent study compared the cutting efficacy of radial-landed and triangular cross sections.[292] The latter were shown to be more cutting efficient compared with radial-landed design; somewhat surprisingly, flexible martensitic alloy was cutting more rapidly compared with conventional NiTi alloys.

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Patency and glide path preparation

A *patency file* is a small K-file (usually a size #10 or #15) that is passively extended just through the apical foramen. Demonstration of apical patency has been suggested as a procedural for most rotary techniques. It is believed to remove accumulated debris, to help maintain WL, and thereby to translate into greater clinical success.[301] One concern with the patency file was that instead of having a cleaning effect, the file would drive contaminated debris through the foramen. However, an in vitro study suggested that the risk of inoculation was minimal when canals were filled with NaOCl.[214] Maintaining patency throughout an endodontic procedure does not lead to an increase in posttreatment symptoms.[21] Only initial clinical evidence exists either favoring the use of a patency file; however, experience suggests that this technique involves relatively little risk and provides some benefit as long as small files are used carefully.

The file that has reached the apical foramen has already formed a so-called glide path. This term has been used in endodontics for more than a decade[337] and relates to securing an open pathway to the canal terminus that subsequent engine-driven instruments can follow. The presence of a glide path is essential for the predictable use of rotary instruments since it minimizes the so-called screw-in or threading-in effect[173] and risk of torsional failure. It has been considered that taper lock may occur if the canal cross section is smaller than the tip of the instrument;[337] in that regard, a glide path reduces the contact area between subsequent shaping instruments and the canal wall, consequently the torsional loads induced by shaping rotary instruments are lower.[52]

It is advocated to explore and shape a root canal at least to a #15 size instrument before the use of a rotary NiTi instrument to full WL[442] to create a glide path for the safe advancement of the rotary shaping instrument tip.[321] The presence of an appropriate glide path is indicated by the fact that a straight size #15 K-file can passively and smoothly travel to WL with long in-and-out movements.

Classically, a glide path was created and secured with K-file sizes #10 and #15 used in watch-winding or balanced-force motion. However, recently NiTi rotary instruments with small tip diameter have been designed

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specifically to simplify the process after a size #10 K-file has reached WL.

Various small engine-driven instruments were introduced that are aimed at simplifying the glide path process—for example, Pathfiles (Dentsply Sirona),[47] Scout RaCe (FKG),[298] and G-file (MicroMega).[101] Copious irrigation and frequent recapitulations with a smaller file to WL may be required, and in some instances of highly mineralized canals, clinicians must devise strategies using small crown-down and/or step-back sequences.

The creation of a rotary glide path has demonstrated better preservation of the canal anatomy with fewer shaping aberrations[11,47] and has shown a less incidence of postoperative pain.[320] However, some studies have also reported acceptable performance of manual instruments for glide path preparation.[101,303]

Working length determination

Devices

Radiographs, tactile sensation, the presence of moisture on paper points, and knowledge of root morphology have been used to determine the length of root canal systems. Following earlier experiments by Custer,[99] subsequently Sunada[439] developed the first commercialized electronic apex locator, suggesting that the apical foramen could be localized using a direct electric current. Currently the electronic apex locator is considered an accurate tool for determining WL.[135] One study reported that the use of electronic apex locators in a dental student clinic resulted in a higher quality of obturation length control and an overall reduction in the number of radiographs taken.[135] However, these devices must not be considered flawless, because several variables are known to affect their accuracy. For example, immature roots can present problems.[208] Once roots mature (i.e., form a narrow apical foramen) and instruments are able to contact the canal walls, an electronic apex locator's accuracy greatly improves. Some investigators have found no statistical difference between roots with vital and necrotic tissue.[157,275] Because apical root resorption is prevalent in necrotic cases with long-standing apical lesions,[473] it may be concluded that apical resorption does not have a significant effect on the accuracy of electronic apex locators.

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Recently some clinicians have advocated the use of the electronically determined WL in lieu of WL estimations using the placement of a file in the canal and a radiograph. However, combined use of both radiographic and electronic techniques has been shown to result in greater accuracy.[120] Furthermore, radiographs may add essential anatomic information that could be missed if electronic apex locators are used exclusively.

The first two generations of electronic apex locators were sensitive to the contents of the canal and irrigants used during treatment. The development of an algorithm called the *ratio measurement method* distinguished the third generation of apex locators.[230] To arrive at this method, the impedance of the canal was measured with two current sources of different frequencies, and a quotient was determined using the electrical potentials proportional to each impedance.[230] This study found that electrolytes did not have a significant effect on the accuracy of the unit. Some third-generation apex locators are the Endex Plus, or Apit (Osada, Los Angeles, CA, USA); the Root ZX (J. Morita, Kyoto, Japan);[135,242] and the Neosono Ultima EZ (Satelec, Mount Laurel, NJ, USA). The Endex Plus device uses 1 and 5 kHz and provides apex location based on subtraction. The Root ZX emits currents at frequencies of 8 and 0.4 kHz and provides apex location based on the resulting quotient. In addition to latest model apex locators, manufacturers have developed smaller models (Fig. 8.36, *B*).

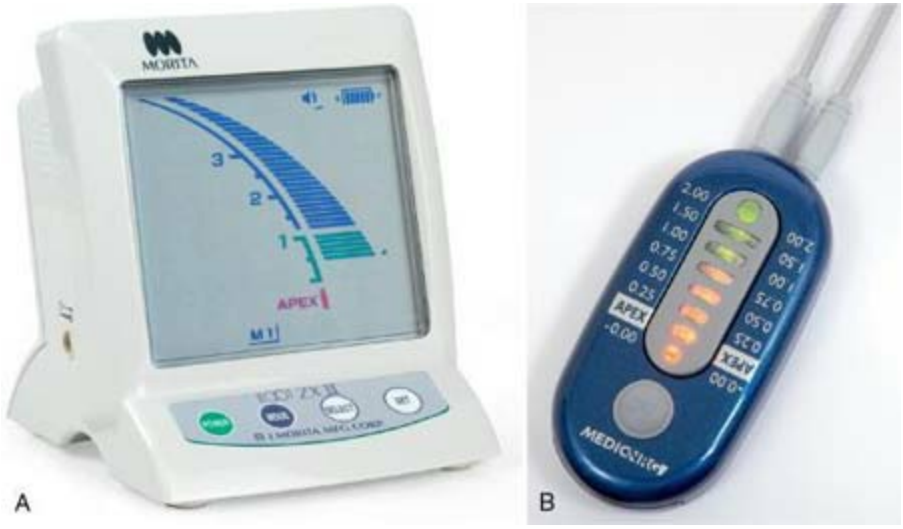

FIG. 8.36 Root ZX apex locator with lip clip and file holder.

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Set of two images show a root ZX apex locator and a hand-piece.

Source: (**A,** Courtesy J. Morita USA, Irvine, CA, USA). NRG miniature electronic apex locator. (**B,** Courtesy MedicNRG, Kibutz Afikim, Israel.)

Apex locators are generally safe to use; however, manufacturers' instructions state that they should not be used on patients with pacemakers without consulting the patient's cardiologist. However, when connected directly to cardiac pacemakers in vitro, electronic apex locators did not interfere with the function of the pacemaker[145] and did not interfere with the functioning of any of the cardiac devices tested in a clinical study under ECG monitoring.[487]

Strategies

Anatomic studies and clinical experience suggest that typically teeth are 19 to 25 mm long. Most clinical crowns are approximately 10 mm long, and most roots range from 9 to 15 mm in length. Roots, therefore, can be divided into thirds that are 3 to 5 mm long. An important issue in root canal treatment is the apical endpoint of the prepared shape in relation to the apical anatomy. Traditional treatment has held that canal preparation and subsequent obturation should terminate at the *apical constriction*, the narrowest diameter of the canal (Fig. 8.37). This point is believed to coincide with the cementodentinal junction (CDJ; see Chapter 9) and is based on histologic sections and ground specimens. However, the position and anatomy of the CDJ varies considerably from tooth to tooth, from root to root, and from wall to wall in each canal. Moreover, the CDJ cannot be located precisely on radiographs. For this reason, some have advocated terminating the preparation in necrotic cases at 0.5 to 1 mm short of the radiographic apex and 1 to 2 mm short[187,357,492] in cases involving irreversible pulpitis. Although there is no definitive validation for this strategy at present,[379] well-controlled follow-up studies seem to support it.[297,419]

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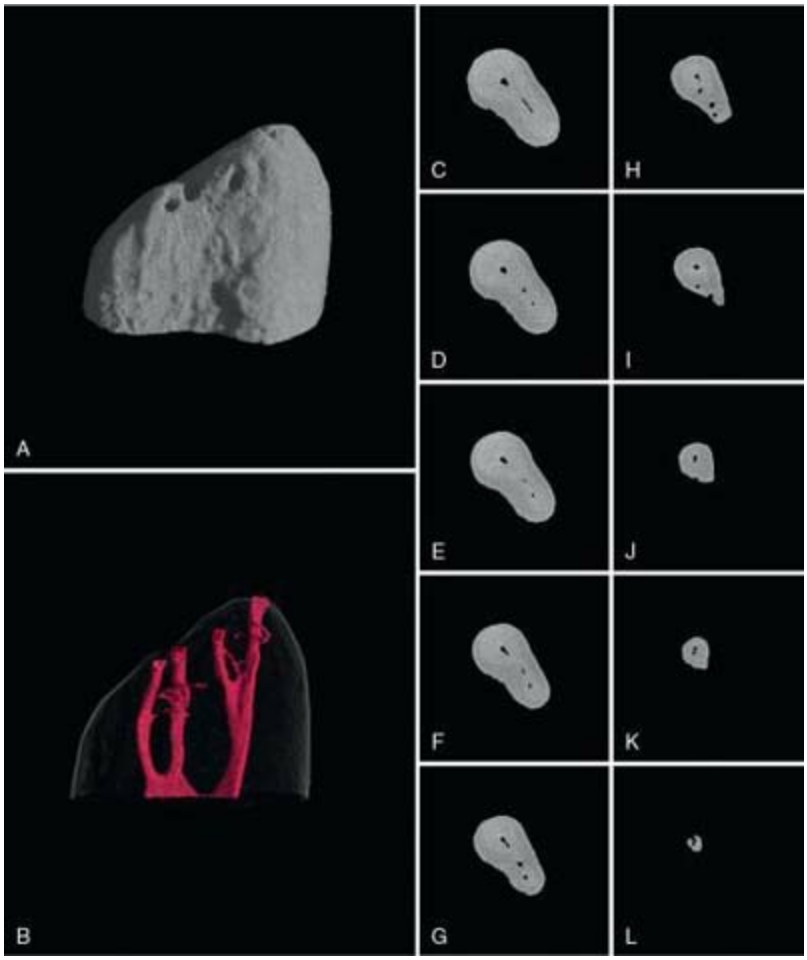

FIG. 8.37 Microcomputed tomographic scan of anatomy of the apical 5 mm of a mesiobuccal root (8 µm resolution). **A–B**, Three-dimensional reconstruction of outer contour and root canal systems. **C-L,** Cross sections 0.5 mm apart.

A) Scan shows a conical root with a textured surface.

B) Scan shows a conical root with two branched canals.

C) Cross-section of coronal canal shows a black dot in the center and a slanting line under it.

D) Cross-section of coronal canal shows three black dots across the surface.

E) Cross-section of coronal canal shows three black slightly elongated dots across the surface.

F) Cross-section of coronal canal shows three black larger elongated dots across the surface.

G) Cross-section of coronal canal shows three black irregular ruptures across the surface.

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H) Cross-section of canal shows four black irregular dots across the surface.

I) Cross-section of canal shows three larger black irregular dots with two small gray dots across the surface. Another dot is visible at the outline.

J) Cross-section of canal shows two larger black dots lying side by side with two small gray dots across the surface. Another dot is visible at the outline.

K) Cross-section of canal shows two larger black dots lying side by side to each other.

L) Cross-section of canal shows three larger black dots lying side by side to each other resulting in a rupture in the canal.

Working to shorter lengths may lead to the accumulation and retention of debris, which in turn may result in apical blockage (Fig. 8.38). If the path to the apex is blocked, working to short lengths may contribute to procedural errors such as apical perforations and fractured instruments. Such obstacles (which consist of collagen fibers, dentin mud, and most importantly, residual microbes) in apical canal areas are a major cause of persistent or recurrent apical periodontitis,[178,296,409] or posttreatment disease (also see Chapters 15 and 16).[138,490]

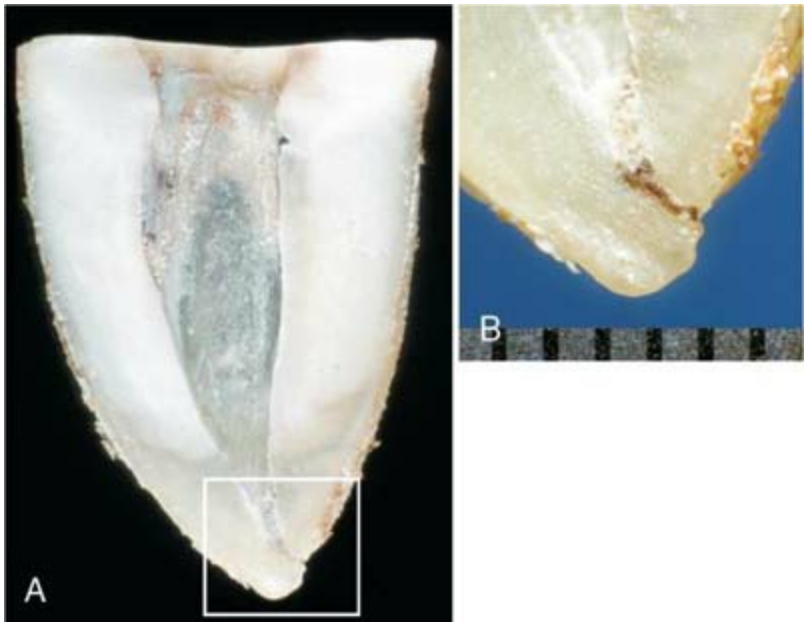

FIG. 8.38 Presence of dentin dust as a possible source of microbial irritation. Tooth #18 underwent root canal therapy. The clinician noted an apical blockage but was unable to bypass it. Unfortunately, intense

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pain persisted and at the patient's request, the tooth was extracted a week later. **A,** Mesial root of tooth #18; mesial dentin has been removed. **B,** Magnified view (×125) of rectangle in **A** shows an apical block (gradation of ruler is 0.5 mm).

A) Close-up of a cutaway tooth shows a canal with an inset marked on the root showing removed dentin dust.

B) Micrograph of an extracted tooth shows a blocked root canal with dentin dust.

Using an electronic apex locator has helped clinicians identify the position of apical foramina more accurately and allow safe canal shaping as close as 0.5 mm to the canal terminus.

Canal enlargement/preparation

Rationale

Like the position of the apical constriction, apical diameters are difficult to ascertain clinically.[240] Some clinicians have recommended gauging canal diameters by passing a series of fine files apically until one fits snugly. However, such an approach is likely to result in underestimation of the diameter.[486] This is a crucial point because the initial canal size is a major determinant for the desired final apical diameter, with the second relevant dimension being the size that allows deep irrigation needle penetration.

An ongoing debate exists between those who prefer smaller apical preparations combined with tapered shapes and those who favor larger apical preparations for better removal of infected dentin and to allow irrigation fluids access to the apical areas (Table 8.2). Both sides stress the importance of maintaining the original path of the canal during preparation; otherwise, bacteria in the apical one third of the root canal may not be reached by sufficient amounts of an antimicrobial agent.[289] Investigators demonstrated a higher percentage of bacterial elimination in single-root canal systems in situ by using a combination of significant enlargement of the apical third and NaOCl irrigation.[79] However, preparation errors (e.g., zips, canal transportation) may occur in such larger preparations specifically in curved canals, both with stainless steel and NiTi instruments (Fig. 8.39).

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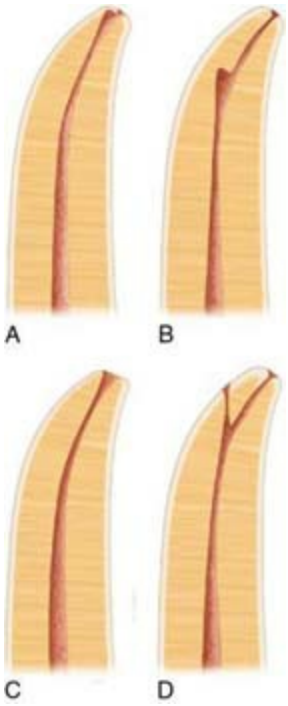

FIG. 8.39 Schematic diagrams showing the most common preparation errors. **A,** Apical zip. **B,** Ledge. **C,** Apical zip with perforation. **D,** Ledge with perforation.

A) A cutaway diagram of tooth shows a canal with upper broad and lower narrow canal, branching partially at the tip.

B) A cutaway diagram of tooth shows a canal with upper broad and lower narrow canal, branching partially at the tooth curve.

C) A cutaway diagram of tooth shows a canal with upper broad and lower narrow canal and a broader tip.

D) A cutaway diagram of tooth shows a canal with upper broad and lower narrow canal, branching completely at the tip. Tips are visibly broader.

Table 8.2

Characteristics of Wide and Narrow Apical Preparations

Root Canal Preparation	Benefits	Drawbacks
Narrow apical size	Minimal risk of canal transportation and extrusion of irrigants or filling material	Little removal of infected dentin Questionable rinsing effect

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	Can be combined with tapered preparation to counteract some drawbacks Less compaction of hard tissue debris in canal spaces	in apical areas during irrigation Possibly compromised disinfection during interappointment medication Not ideal for lateral compaction
Wide apical size	Removal of infected dentin Access of irrigants and medications to apical third of root canal	Risk of preparation errors and extrusion of irrigants and filling material Not ideal for thermoplastic obturation

Thorough disinfection of the apical part of a root canal is essential, because this area is most likely to harbor residual intraradicular bacteria.[297] Wider apical preparations remove potentially infected dentin, allowing the delivering needle and subsequently antimicrobial irrigants to penetrate the root canal more deeply.[91,130]

A study investigating rotary NiTi files of three tapers (.06,.08, and.10) with file tips in sizes #20, #30, and #40 showed that size #20 instruments left significantly more debris in the apical third compared with size #40 instruments.[464] On the other hand, a study in which half the samples were prepared to a size #25 file and the other half to a size #40 file found no statistically significant difference in bacterial growth after instrumentation, with no growth observed after 1 week of treatment with a $Ca(OH)_2$ dressing.[500] Step-down sequences with additional apical enlargement to ISO size #35 and a serial step-back technique with no apical enlargement were compared, using NaOCl and EDTA as irrigants. Here, no significant difference was detected in colony-forming units with or without apical enlargement.[95] These researchers concluded that dentin removal in the apical third might be unnecessary if a suitable coronal taper is achieved.

Despite the disagreement over the appropriate width of a preparation (see Table 8.2), these studies suggest that root canal preparations should be confined to the canal space, should be sufficiently wide, and should incorporate the original root canal cross sections (see Fig. 8.4).

Traditional cleaning and shaping strategies (e.g., the step-back technique)

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focused immediate preparation of the apical third of the root canal system, followed by various flaring techniques to facilitate obturation.[161,380,453] In an attempt to reach the canal terminus, the clinician first selected a small file, placed an appropriate curve on the instrument, and then tried to work the file to full length. If the terminus could not be reached, the file was removed and, after irrigation, either the same file or a smaller one was inserted. However, not infrequently, full length was not obtained, either because of blockage or because of coronal binding.

Coronal binding is caused by overhangs at the orifice level and when the canal is less tapered than an instrument, making it bind coronally. Moreover, a straight root often contains a curved canal, such as buccal and lingual curvatures that cannot be seen on radiographs.[98,343] In addition, passing a precurved negotiating file through a coronally tight canal will straighten the instrument.[425]

Various instrumentation sequences have been developed for hand and rotary instruments; these are discussed later in this chapter. However, the shape of the access cavity is the prerequisite that must be optimized before any canal preparation can take place (see Chapter 7).

Basic cleaning and shaping strategies for root canal preparation can be categorized as crown-down, step-back, apical widening, and hybrid techniques. In a crown-down approach, the clinician passively inserts a large instrument into the canal up to a depth that allows easy progress. The next smaller instrument is then used to progress deeper into the canal; the third instrument follows deeper again, and this process continues until the terminus is reached. Both hand and rotary instruments may be used in a crown-down manner. However, instrument sets with various tip diameters and tapers allow the use of either decreasing tapers or decreasing diameters for apical progress. Debate continues as to which of those strategies is superior for avoiding taper lock; currently no compelling evidence favors either of them.

In the step-back approach, WLs decrease in a stepwise manner with increasing instrument size. This prevents less flexible instruments from creating ledges in apical curves while producing a taper for ease of obturation.

As discussed previously, the aim of apical widening is to fully prepare apical canal areas for optimal irrigation efficacy and overall antimicrobial

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activity. Recently, apical enlargement has been broken down into three phases: pre-enlargement, apical enlargement, and apical finishing.[476]

Many rotary techniques require a crown-down approach to minimize torsional loads[52] and reduce the risk of instrument fracture. Used sequentially, the crown-down technique can help enlarge canals further. All basic techniques described so far may be combined into a hybrid technique to eliminate or reduce the shortcomings of individual instruments.

Provided adequate tools are used and the access cavity design is appropriate, excessive thinning of radicular structures can be avoided (see Fig. 8.5). Vertical root fractures and perforations are possible outcomes of excessive removal of radicular dentin in zones that have been termed "danger zones."[7] Overenthusiastic filing, for example, may lead to more procedural errors (see Figs. 8.22 and 8.39). On the other hand, ideal preparation forms without any preparation errors and with circular incorporation of the original canal cross sections may be achieved with suitable techniques (see Fig. 8.4).

Techniques

Standardized technique.

The standardized technique adopts the same WL definition for all instruments introduced into a root canal. It therefore relies on the inherent shape of the instruments to impart the final shape to the canal. Negotiation of fine canals is initiated with lubricated fine files in a so-called watch-winding movement. These files are advanced to WL and worked either in the same hand movement or with "quarter-turn-and-pull" until a next larger instrument may be used. Conceptually, the final shape should be predicted by the last instrument used. A single matching gutta-percha point may then be used for root canal filling. In reality, this concept is often violated: curved canals shaped with the standardized technique will be wider than the last used instrument,[16] exacerbated by the pulling portion of the hand movement. Moreover, adequate compaction of gutta-percha in such small a taper (~.02) is difficult or impossible (see Chapter 9).[15]

Step-back technique.

Realizing the importance of a shape larger than that produced with the

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standardized approach, Weine et al.[483] suggested the step-back technique, incorporating a stepwise reduction of the WL for larger files, typically in 1 mm or 0.5 mm steps, resulting in flared shapes with.05 and.10 taper, respectively. Incrementally reducing the WL when using larger and stiffer instruments also reduced the incidence of preparation errors, in particular in curved canals. This concept appeared to be clinically very effective.[293]

Although the step-back technique was primarily designed to avoid preparation errors in curved canals, it applies to the preparation of apparently[98,382] straight canals as well. Several modifications of the step-back technique have been described over the years, to explore and provide some enlargement prior to reaching the WL.

Step-down technique.

Other clinicians[156] described a different approach. They advocated shaping the coronal aspect of a root canal first before apical instrumentation commenced. This technique is intended to minimize or eliminate the amount of necrotic debris that could be extruded through the apical foramen during instrumentation;[129] moreover, by first flaring the coronal two thirds of the canal, apical instruments are unimpeded through most of their length. This in turn may facilitate greater control and less chance of zipping near the apical constriction.[246]

Crown-down technique.

Multiple modifications of the original step-down technique have been introduced, including the description of the crown-down technique.[133,274,378] The more typical step-down technique includes the use of a stainless steel K-file exploring the apical constriction and establishing WL. In contrast, a crown-down technique relies more on coronal flaring and then determination of the WL later in the procedure.

To ensure penetration during step-down, one may have to enlarge the coronal third of the canal with progressively smaller GG drills or with other rotary instruments. Irrigation should follow the use of each instrument and recapitulation after every other instrument. To properly enlarge the apical third and to round out ovoid shape and lateral canal orifices, a reverse order of instruments may be used starting with a size #20 (for example) and

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enlarging this region to a size #40 or #50 (for example). The tapered shape can be improved by stepping back up the canal with larger instruments, bearing in mind all the time the importance of irrigation and recapitulation.

The more typical crown-down, or double-flare technique,[133] consisted of an exploratory action with a small file—a crown-down portion with K-files of descending sizes and an apical enlargement to size #40 or similar. The original technique included stepping back in 1-mm increments with ascending file sizes and frequent recapitulations with a small K-file and copious irrigation. It is further emphasized that significant wall contact should be avoided in the crown-down phase to reduce hydrostatic pressure and the possibility of blockage. Several studies[377,378] demonstrated more centered preparations in teeth with curved root canals shaped with a modified double-flare technique and Flex-R files compared with shapes prepared with K-files and step-back technique. A double-flare technique was also suggested for ProFile rotary instruments.[387]

Balanced force technique.

Regarding hand movements, a general agreement exists that the so-called balanced force technique creates the least canal aberrations with K-files. This technique has been described as a series of rotational movements for Flex-R files,[362] but it can also be used for K-files and other hand instruments such as GT hand files. Many different explanations have been offered for the obvious and undisputed efficacy of the balanced force approach,[84,241,382] but general agreement exists that it provides excellent canal-centering ability, superior to other techniques with hand instruments.[29,65]

The balanced force technique involves three principle steps.[362] The first step (after passive insertion of an instrument into the canal) is a clockwise rotation of about 90 degrees to engage dentin (Fig. 8.40). In the second step, the instrument is held in the canal with adequate axial force and rotated counterclockwise to break loose the engaged dentin chips from the canal wall; this produces a characteristic clicking sound. Classically, in the third step, the file is removed with a clockwise rotation to be cleaned; however, because files used with the balanced force technique are not precurved, every linear outward stroke essentially is a filing stroke and may lead to some straightening of the canal path. Therefore, in many cases, the clinician may

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advance farther apically rather than withdrawing the file, depending on the grade of difficulty.

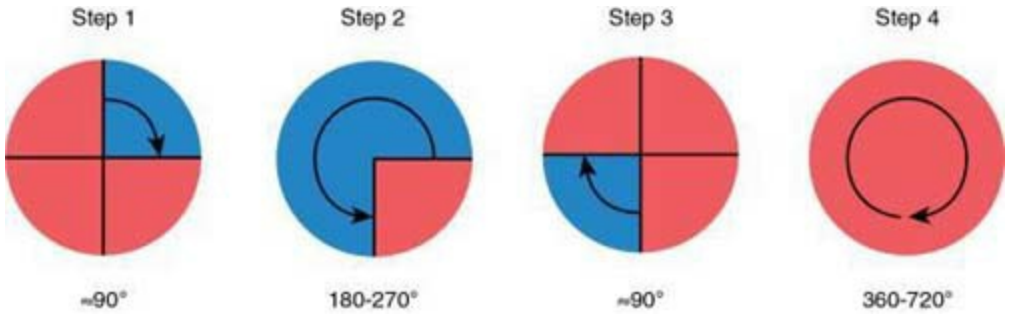

FIG. 8.40 Diagram of handle movements during balanced force hand preparation. STEP 1: After pressureless insertion of a Flex-R or NiTiFlex K-file, the instrument is rotated clockwise 90 degrees, using only light apical pressure. STEP 2: The instrument is rotated counterclockwise 180 to 270 degrees; sufficient apical pressure is used to keep the file at the same insertion depth during this step. Dentin shavings are removed with a characteristic clicking sound. STEP 3: This step is similar to step 1 and advances the instrument more apically. STEP 4: After two or three cycles, the file is loaded with dentin shavings and is removed from the canal with a prolonged clockwise rotation.

A) Diagram of a circle divided into four quarters shows blue colored first quarter with a clockwise arrow at 90 degrees and three red quarters.

B) Diagram of a circle divided into three merged quarters of blue with an anticlockwise arrow at 180 to 270 degrees and a second red quarter.

C) Diagram of a circle divided into four quarters shows blue colored third quarter with a clockwise arrow at 90 degrees and three red quarters.

D) Diagram of a red circle shows a clockwise arrow at 360 to 720 degrees.

Rotary instrumentation.

NiTi rotary instruments are invaluable devices in the preparation of root canals, although hand instruments may be able to enlarge some canals just as efficiently when used in appropriate sequences. After orifice modification, the access cavity and canals are flooded with irrigant, and a small file is advanced into the canal. Application of a lubricant when using hand instruments can help prevent apical blockage at this early stage. Once the WL

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has been established (aided by an electronic apex locator and radiographically verified), apical preparation begins to facilitate a glide path for subsequent rotary instrumentation (Fig. 8.41; also see earlier discussion).

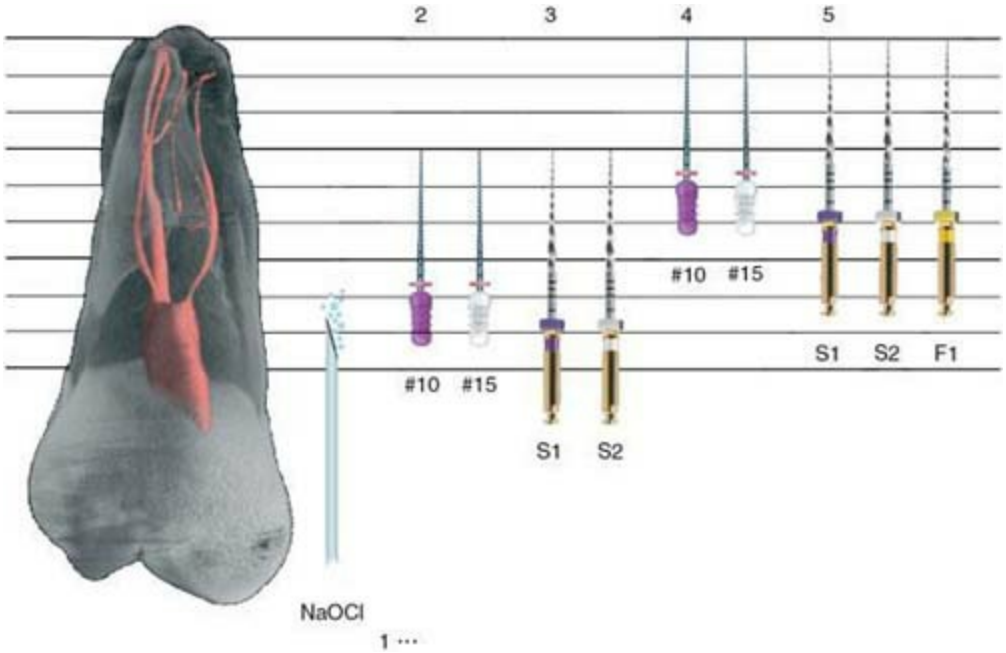

FIG. 8.41 Instrumentation of root canals with the single length technique, in this example using the ProTaper instruments. After irrigation and scouting (*1* and *2*), the coronal thirds are enlarged with shaping files *S1* and *S2*. Hand files then are used to determine the WL and to secure a glide path. Apical preparation is completed with S1 and S2. Finishing files are used to the desired apical width.

A cutaway diagram of coronal enlargement in a maxillary posterior tooth with a narrow canal that exits the pulp cavity at an angle. A range of orifice shapers show sodium hypochlorite angled shapers in number 10 and 15 under 2, S1 and S2 under 3, number 10 and number 15 under 4 and S1, S2 and F1 under 5.

Fig. 8.42 illustrates the development of two different shapes in the mesial root canals of a mandibular molar, clearly showing that substantial areas of the root canal surface in either case are not instrumented, even when apical size #50 or 0.09 taper are reached (see *red areas* in Fig. 8.42, *G* and *I*).

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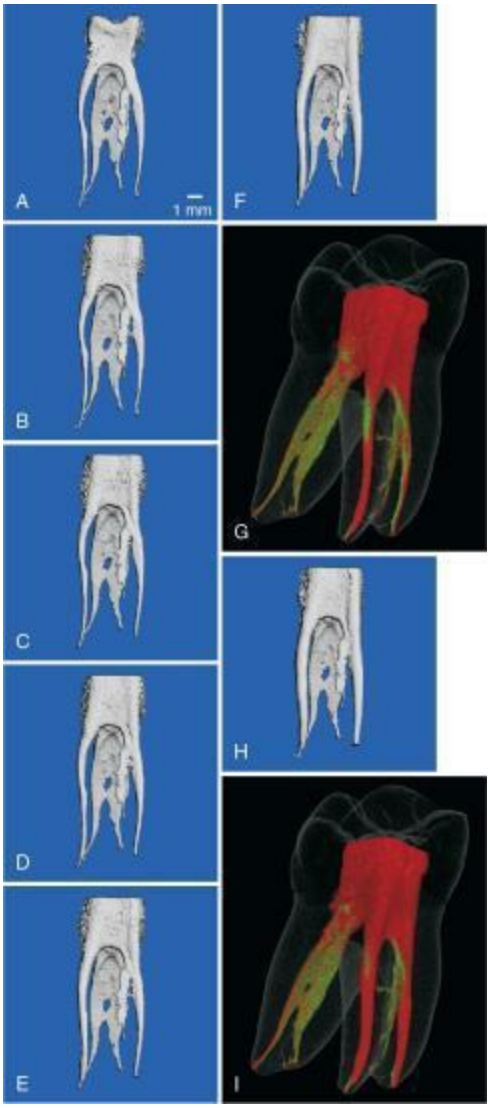

FIG. 8.42 Stepwise enlargement of mesial root canal systems in an extracted mandibular molar demonstrated with micro–computed tomography (μCT) reconstructions. The buccal canal *(left)* was prepared with a LightSpeed (LS) instrument, and the lingual canal *(right)* was shaped with a ProTaper (PT) instrument. **A,** Pretreatment view from the mesial aspect. Note the additional middle canal branching from the lingual canal into the coronal third. **B,** Initial preparation and opening of the orifices, aided by ultrasonically powered instruments. **C,** First step of root canal preparation, up to LightSpeed size #20 and ProTaper shaping file S1. **D,** Further enlargement to LS size #30 and PT shaping file S2. **E,** Apical preparation to LS size #40 and PT finishing file F1. **F,** Additional enlargement to LS size #50 and PT finishing file F2. **G,** Superimposed μCT reconstructions comparing the initial canal geometry *(in green)* with the shape reached after use of the instruments shown in **F**. **H,** Final shape after step-back with LS instruments and PT finishing file F3. **I,** Superimposed μCT reconstructions comparing initial geometry and final shape. Note the

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slight ledge in the buccal canal after LS preparation and some straightening in the lingual canal after PT preparation.

A) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole with three holes and a ruptured root outline.

B) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole with two smaller holes.

C) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole and two smaller distinct holes.

D) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole and three smaller holes lying linearly.

E) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole and a smaller hole over it.

F) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole and four smaller holes over it with a ruptured root outline.

G) Scan of a tooth shows a red crown and canals with pigments of green around the first, second canal and partial third canal.

H) Diagram of a modeled tooth shows a crown with one root canal depicting a large irregular hole with three holes over it with and a ruptured root outline.

I) Scan of a tooth shows a red crown and canals with pigments of green around the first canal and partial second canal with specks of pigment on third canal.

Specific nickel titanium (NiTi) instrumentation techniques

Crown-down.

This was the dominant approach for many years and is still being used, for example for ProFile and other instruments with standard.04 and.06 tapers. It must be noted that the manufacturers' instructions for these systems vary somewhat, and the instructions for GT rotary, RaCe, and the TF vary even more. Clinicians should always consult the manufacturers' instructions for details on working with any endodontic instrument.

Coronal preenlargement is suggested, the WL then is determined as described previously, and an open glide path is secured with K-files up to size #15 or #20, depending on the canal anatomy. If canal size permits, canal

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preparation begins with.06 taper instruments in descending tip diameters.[52] In more difficult small canals,.06 tapers are followed by.04-tapered instruments, also with descending tip diameters. Apical preparation is performed either with multiple shaping waves, as suggested for GT rotary files,[68] or in a step-back manner.[386] Recapitulation with a small hand file is recommended throughout the preparation.

Single length.

The approach for ProTaper and ProTaper Next instruments differs from that for earlier NiTi rotary files in that no traditional crown-down procedure is performed (see Fig. 8.44, later).

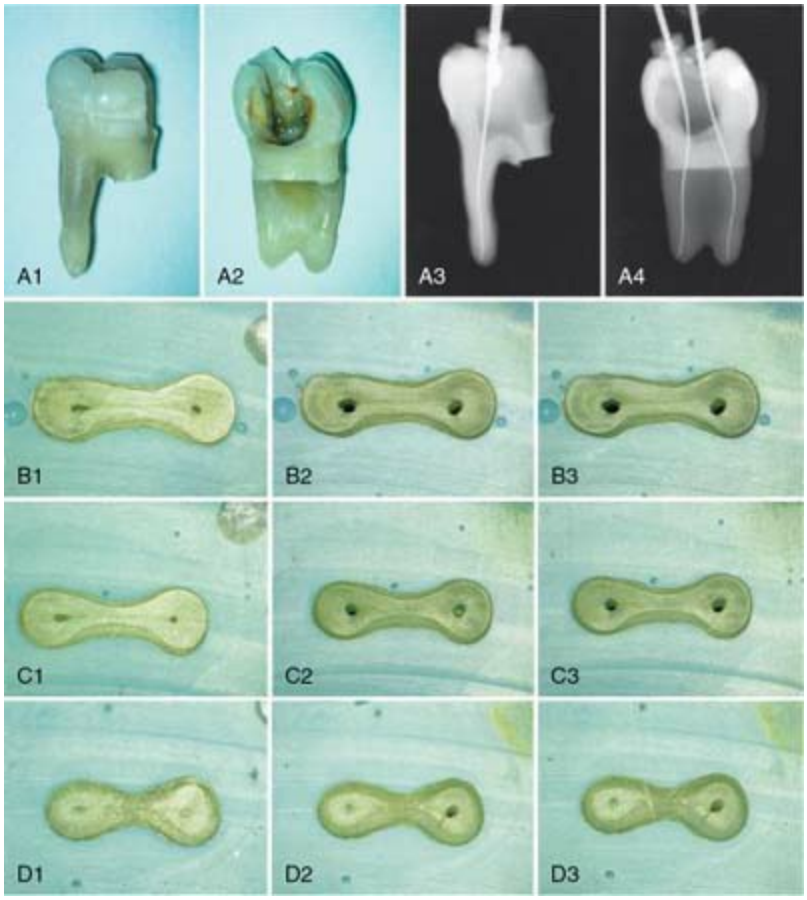

FIG. 8.44 Effect of a hybrid technique on root canal anatomy studied in a Bramante model. **A1–A4,** Both mesial canals of an extracted mandibular molar have been instrumented. Canal cross sections are shown before instrumentation **(B1–D1). B2–D2**, Cross sections after preenlargement with a ProTaper F3 file *(left canal)* and a size #45,.02

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taper instrument *(right canal)*. The final apical sizes were LightSpeed (LS) #50 and size #50,.02 taper in the left and the right canal, respectively.

A1) Lateral view of a tooth shows a crown, neck and root system with a missing right root.

A2) Lateral view of a tooth shows a decayed crown with a laterally missing root.

A3) Radiograph of a lateral tooth shows a radiopaque crown with a black smudge, radiolucent neck and a missing right canal. A radiopaque outline of a file inserted in left canal is visible.

B1) An image of horizontally sectioned tooth shows a lateral rupture on the left canal and a small circular rupture on right canal.

B2) An image of horizontally sectioned tooth shows two circular large ruptures in left and right canal.

B3) An image of horizontally sectioned tooth shows two circular large ruptures in left and right canal.

C1) An image of horizontally sectioned tooth shows a laterally larger rupture on the left canal and a small circular rupture on right canal.

C2) An image of horizontally sectioned tooth shows two circular large ruptures in left and right canal.

C3) An image of horizontally sectioned tooth shows two circular large ruptures in left and right canal.

D1) An image of horizontally sectioned tooth shows a laterally blurred rupture on the left canal and a small circular rupture on right canal with a white patch around it.

D2) An image of horizontally sectioned tooth shows circularly blurred ruptures in left canal and circular large rupture on right canal, with white patches around canals.

D3) An image of horizontally sectioned tooth shows two circular large ruptures in left and right canal, with white patches around it.

Source: (Courtesy Dr. S. Kuttler, Dr. M. Gerala, and Dr. R. Perez.)

First, size #10 and #15 hand files are passively inserted into the coronal two thirds of a root canal as pathfinding files, which confirm the presence of a smooth, reproducible glide path. This step is essential for ProTaper shaping

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instruments, because they are mostly side-cutting and have fine, rather fragile tips.

Shaping files S1 and S2 are then passively inserted into the scouted canal spaces, which have been filled with irrigant (preferably NaOCl). If necessary, the SX file can be used at this stage to relocate orifices or remove obstructing dentin. After each shaping file is used, the canals are reirrigated, and a size #10 file is used to recapitulate to break up debris and move it into solution. This process is repeated until the depth of the pathfinding #10 or #15 file is reached.

After irrigation, the apical third is fully negotiated and enlarged to at least a size #15 K-file, and WL is confirmed (see Fig. 8.44). Depending on the canal anatomy, the rest of the apical preparation can be done with engine-driven ProTaper shaping and finishing hand files. As an alternative, handles may be placed on these instruments' shanks, so that they can be used for a manual balanced force technique.

ProTapers S1 and S2 are then carried to full WL, still in a floating, brushing motion. WL should be confirmed after irrigation and recapitulation with a K-file, aided by an electronic apex locator and/or radiographs. Because of the progressive taper and more actively cutting flutes in the ProTaper design, interferences in the middle and coronal thirds are removed at this stage.

The preparation is finished with one or more of the ProTaper finishing files, used in a nonbrushing manner; because of their decreasing taper, these files will reach the WL passively. Recapitulation and irrigation conclude the procedures (see Fig. 8.41). Many other contemporary engine-driven techniques use the single-length concept—for example, WaveOne Gold, TRUShape, and XP-Shaper. However, the emphasis on Orifice Modification prior to deep instrument penetration allows these techniques to retain many of the benefits of the classic crown-down techniques, such as less change of WL during the procedure, deep and early access of irrigants, and less stress on fragile files.[327]

Hybrid techniques.

For many years, established clinical practice has suggested combining various NiTi preparation systems[79,476] to address certain shortcomings of

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most current instruments (Box 8.4). Although many combinations are possible, the most popular and useful ones involve coronal preenlargement followed by various apical preparation sequences. However, clinicians must keep in mind that anatomic variations in each canal must be addressed individually with specific instrument sequences. Most important, oval canals extend deep into the apical area,[442,482,491] and apical foramina may in fact be oval in most cases.[64] Naturally, a rotating file can produce a round canal at best (Fig. 8.43); therefore a strategy must be devised for adequately shaping oval canals without overly weakening radicular structure (compare Figs. 8.4 and 8.5).

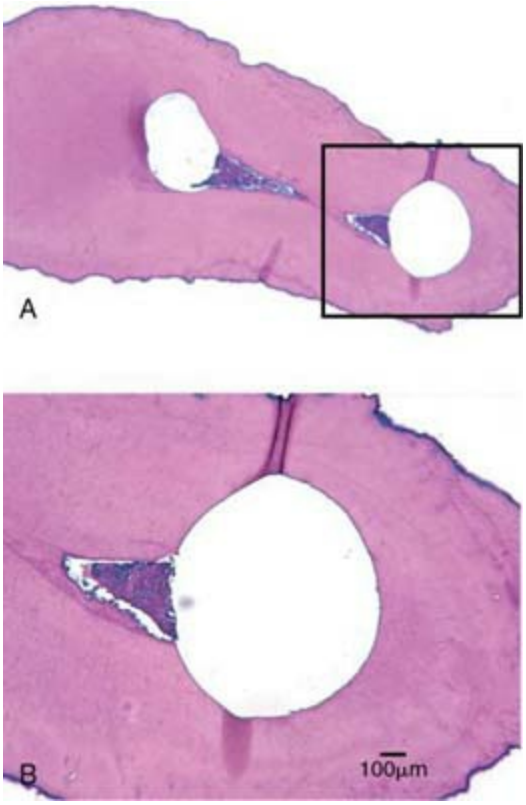

FIG. 8.43 Remaining potentially infected tissue in fins and isthmus configuration after preparation with rotary instruments. **A,** Cross section through a mesial root of a mandibular molar, middle to coronal third of the root. Both canals have been shaped; the left one is transported mesially (×10). **B,** Magnified view of rectangle in **A**. Note the presence of soft tissue in the isthmus area (×63).

A) An image of a horizontal section of tooth shows two holes with two triangular sections across the gap between the two. The right hole with the

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triangular section is enclosed in a square frame.

B) Magnified image of a horizontal section of tooth shows a holes with a triangular section across the left surface.

Source: (Courtesy Professor H. Messer.)

Box 8.4

Benefits of Using a Combination of Instruments for Endodontic Therapy

- Instruments can be used in a manner that promotes their individual strengths and avoids their weaknesses (most important).
- Hand instruments secure a patent glide path.
- Tapered rotary instruments efficiently enlarge coronal canal areas.
- Less tapered instruments allow additional apical enlargement.

Histologic slides (see Fig. 8.43) and microcomputed tomography reconstructions show critical areas that were not mechanically prepared despite the use of various individual rotary techniques. The aim of hybridizing NiTi rotary techniques, therefore, is to increase apical size using a fast and safe clinical procedure (Fig. 8.44).

The principle involves the use of a variety of instruments: for example, K-files for establishing patency, ProTaper instruments for body shaping and apical preenlargement, NiTi K-files or LightSpeed instruments for apical widening, and various instruments for final smoothing.[476]

Final apical enlargement

Conceptually, file sizes during canal preparation were termed “initial apical file,” “master apical file,” and “final file,” or IAF, MAF, and FF, respectively. The first file to bind, the IAF, is supposed to give clinicians a guide in determining the final size of their canal preparation. On the other hand, many current instrumentation techniques have, due to their design, predefined final sizes—for example, WaveOne Gold shaping aims at final sizes #25.07, #35.06, and #45.05 taper.

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Whichever apical size was conceptually aimed for, after achieving a tapered canal form, the apical size at that point should be verified by apical gauging. K-files or K-FlexOFiles of the same size are often recommended for this step. During gauging, files should be gently moved to WL and the binding point should be noted. Presence of an apical narrowing means that WL is reached without any shaping effort, with a size-corresponding K-file. However, the next larger K-file size will stay back by a small distance. Finally, maintenance of patency should be verified. If apical gauging stops short of WL, which suggests that a canal is undershaped, additional enlargement should be accomplished with any technique appropriate to the specific anatomy of the root canal. This often necessitates a hybrid technique.

Disinfection of the root canal system

Irrigation is defined as "to wash out a body cavity or wound with water or a medicated fluid" and aspiration as "the process of removing fluids or gases from the body with a suction device." Disinfectant, meanwhile, is defined as "an agent that destroys or inhibits the activity of microorganisms that cause disease."[1]

The objectives of irrigation in endodontics are mechanical, chemical, and biological. The mechanical and chemical objectives are as follows: (i) Flush out debris, (ii) lubricate the canal, (iii) dissolve organic and inorganic tissue, (iv) prevent the formation of a smear layer during instrumentation or dissolve it once it has formed, and (v) detach and disrupt biofilms.[38] Mechanical effectiveness will depend upon the ability of irrigation to generate optimum streaming forces within the entire root canal system, interact with the canal walls, and reach areas that cannot be prepared by mechanical instruments, including fins, isthmuses, and even lateral canals.[479] Chemical effectiveness depends upon the concentration of the antimicrobial irrigant, the area of contact, and the duration of interaction between irrigant and infected material.[58] Final efficiency of endodontic disinfection depends upon its chemical and mechanical effectiveness.[170]

The biological function of irrigants is related to their antimicrobial effects and their capacity to effectively eradicate or maximally reduce intracanal bacteria.

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In principle, irrigants should (i) have high efficacy against anaerobic and facultative microorganisms in their planktonic or biofilm states, (ii) inactivate endotoxin, (iii) be nontoxic when they come in contact with vital tissues, and not cause an anaphylactic reaction.[38]

Hydrodynamics of irrigation

Irrigation dynamics refers to how irrigants flow, penetrate, and readily exchange within the root canal walls as well as the forces produced by them. A better understanding of the fluid dynamics of different modes of irrigation contributes to achieving predictable disinfection of the root canal system. Hence, in endodontic disinfection, the process of delivery is as essential as the antibacterial characteristics of the irrigants.[58]

The effectiveness of root canal irrigation in terms of debris removal and eradication of bacteria depends on several factors that can be controlled by the operator: penetration depth of the needle, diameter of the root canal, inner and outer diameter of the needle, irrigation pressure, viscosity of the irrigant, velocity of the irrigant at the needle tip, and type and orientation of the needle bevel (Fig. 8.45).

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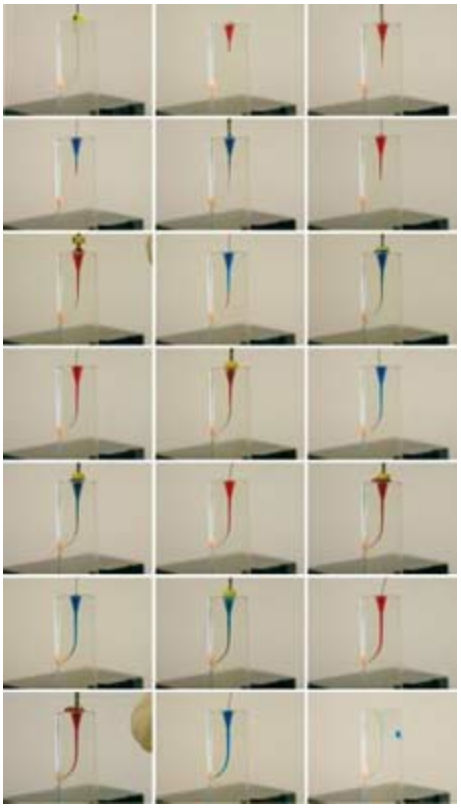

FIG. 8.45 Irrigation and movement of irrigants depends on canal shape. Sequential enlargement of a canal in clear plastic block was performed with a sequence of ProFile instruments in accordance with the manufacturer's recommendations. Alternating irrigation with blue and red fluid was done after each preparation step. Note the apical presence of irrigant after sufficient shape has been provided. Note the distribution of fluid immediately after irrigation with a 30-gauge needle.

A panel of twenty one glass samples with root shaped hollows depicts root shapes and sizes using different colored fluid fillings.

The size and length of the irrigation needle—to root canal dimensions—are of utmost importance for the effectiveness of irrigation. Apical root canal diameter and taper have an impact on needle penetration depth (see earlier in this chapter for details regarding apical preparation size; see Fig. 8.7 and Table 8.2).

The external needle diameter is of relevance for the depth of introduction into the root canal (Fig. 8.46) and rigidity of the needle body, an essential consideration for irrigation of curved canals. Common 27 gauge injection needles have an external diameter of 0.42 mm, but smaller stainless steel irrigation tips with external diameters of 0.32 mm (30 gauge) and even 31

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gauge or 0.26 mm (Navitip, Ultradent, South Jordan, UT, USA) are available. The Stropko Flexi-Tip (30 gauge) needle is fabricated from nickel-titanium to improve penetration into curved root canals. More recently, a flexible plastic irrigation tip was introduced (TruNatomy, Dentsply Sirona).

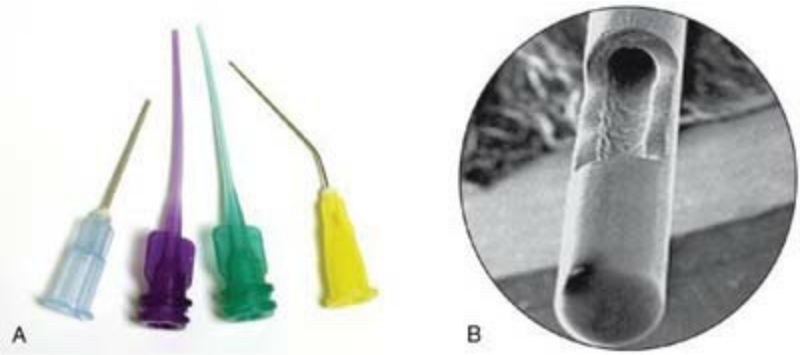

FIG. 8.46 **A,** Various types of needles for root canal irrigation. Shown are examples with open end and closed end, side vented. These are manufactured from plastic and stainless steel. **B,** SEM image of 30 gauge safety needle.

A) Close of a needles show two plastic open needles with a textured and plain needle.

B) Close-up of a cutaway needle with plain cylindrical tip exposing hollow of the needle.

Source: (Courtesy Dr. F. Tay.)

Apical pressure is the force applied perpendicular to the apical wall surface. Internal syringe diameter determines the pressure necessary for moving the syringe plunger. Speed of the plunger determines the velocity with which the irrigant is extruded. Fine needles require more pressure onto the plunger and extrude the irrigant with higher velocity than large needle sizes, which extrude significantly greater amounts of irrigants but for safety reasons cannot be introduced as deeply.

To improve the safety of irrigation and prevent extrusion of the irrigant through the apical foramen, some needle types release the solution via lateral openings and have a closed, safe-ended tip. The orientation of the bevel is crucial to produce a turbulence effect on the dentinal wall of the canal. Wall shear stress has a direct impact on the quality and efficacy of root canal wall

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débridement[61] and is one of the most critical fluid mechanics parameters. Side-vented and double side-vented needles led to maximum shear stress concentrated on the wall facing the outlet (the proximal outlet for the double side-vented needle).[60]

Flow is defined as the quantity of material passing a given area in a unit time period. The flow depends on many factors. Some of them are pressure: Pressure difference is needed to make flow happen. Flow occurs from higher pressure to lower pressure and the greater the difference, the greater the flow. Flow also changes with the diameter of tube or needle: the larger the needle, the more flow. Finally, the longer the length of the needle, the less the flow. In endodontics, anatomical constrains of the root canal make irrigation flow a challenge.

Irrigants

An optimal irrigant would have all of the characteristics considered beneficial in endodontics but none of the negative or harmful properties. Presently, no solution can be regarded as optimal. However, the combined use of selected irrigation products dramatically contributes to the successful treatment outcomes (Boxes 8.5 and 8.6).

Box 8.5

Benefits of Using Irrigants in Root Canal Treatment

- Removal of particulate debris and wetting of the canal walls
- Destruction of microorganisms
- Dissolution of organic debris
- Opening of dentinal tubules by removal of the smear layer
- Disinfection and cleaning of areas inaccessible to endodontic instruments

Box 8.6

Properties of an Ideal Irrigant for Root Canal Treatment

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An ideal irrigant should:

- Be an effective germicide and fungicide.
- Be nonirritating to the periapical tissues.
- Remain stable in solution.
- Have a prolonged antimicrobial effect.
- Be active in the presence of blood, serum, and protein derivates of tissue.
- Have low surface tension.
- Not interfere with repair of periapical tissues.
- Not stain tooth structure.
- Be capable of inactivation in a culture medium.
- Not induce a cell-mediated immune response.
- Be able to completely remove the smear layer, and be able to disinfect the underlying dentin and its tubules.
- Be nonantigenic, nontoxic, and noncarcinogenic to tissue cells surrounding the tooth.
- Have no adverse effects on the physical properties of exposed dentin.
- Have no adverse effects on the sealing ability of filling materials.
- Be convenient in its application.
- Be relatively inexpensive.

Sodium hypochlorite

NaOCl is the most commonly used irrigating solution[285] because of its antibacterial capacity and the ability to dissolve necrotic tissue, vital pulp tissue, and organic components of dentin and biofilms in a fast manner.[392]

NaOCl solution is frequently used as a disinfectant or a bleaching agent. It is the irrigant of choice in endodontics, owing to its efficacy against pathogenic organisms and pulp digestion, and satisfies most of the preferred characteristics stated earlier.[285]

History.

Hypochlorite was first produced in 1789 in France. Hypochlorite solution

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was used as a hospital antiseptic that was subsequently sold under the trade names “Eusol” and “Dakin’s solution.” NaOCl as a buffered 0.5% solution was recommended for the irrigation of wounds during World War I by Dakin.[103] In 1919 Coolidge[97] introduced NaOCl to endodontics as an intracanal irrigation solution.[504]

Mode of action.

When sodium hypochlorite contacts tissue proteins, nitrogen, formaldehyde, and acetaldehyde are formed. Peptide links are fragmented, and proteins disintegrate, permitting hydrogen in the amino groups (-NH-) to be replaced by chlorine (-NCl-) forming chloramines. This plays a vital role in the antimicrobial effectiveness. Necrotic tissue and pus are dissolved, and the antimicrobial agent reaches and cleans the infected areas.

Estrela[125] reported in 2002 that sodium hypochlorite exhibits a dynamic balance (Fig. 8.47):

1. Saponification reaction: Sodium hypochlorite acts as an organic and fat solvent that degrades fatty acids and transforms them into fatty acid salts (soap) and glycerol (alcohol), reducing the surface tension of the remaining solution.
2. Neutralization reaction: Sodium hypochlorite neutralizes amino acids by forming water and salt. With the exit of hydroxyl ions, the pH is reduced.
3. Hypochlorous acid formation: When chlorine dissolves in water, and it is in contact with organic matter, it forms hypochlorous acid. Hypochlorous acid is a weak acid with the chemical formula HClO that acts as an oxidizer. Hypochlorous acid ($HOCl^-$) and hypochlorite ions (OCl^-) lead to amino acid degradation and hydrolysis.
4. Solvent action: Sodium hypochlorite also acts as a solvent, releasing chlorine that combines with protein amino groups (NH) to form chloramines (chloramination reaction). Chloramines impede cell metabolism; chlorine is a strong oxidant and inhibits essential bacterial enzymes by irreversible oxidation of SH groups (sulphydryl group).[143]

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5. High pH: Sodium hypochlorite is a strong base (pH >11). The antimicrobial effectiveness of sodium hypochlorite, based on its high pH (hydroxyl ions action), is similar to the mechanism of action of calcium hydroxide. The high pH interferes in cytoplasmic membrane integrity due to irreversible enzymatic inhibition, biosynthetic alterations in cellular metabolism, and phospholipid degradation observed in lipidic peroxidation.[125]

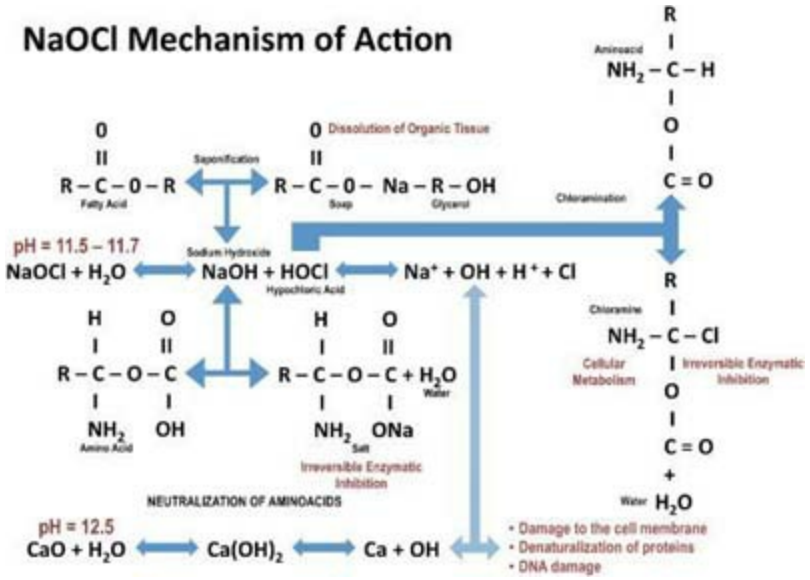

FIG. 8.47 Schematic diagram of the mechanism of action of NaOCl with the main interactions and properties highlighted. This diagram shows the chemical reaction of NaOCl when in contact with organic matter. The solution produces glycerol and soap (saponification reaction) and acts as a fat solvent degrading fatty acids. Moreover, hypochlorous acid is formed with a reduction of the pH and formation of chloramines (chloramination reaction). Damage to the cell membrane, denaturalization of proteins, and DNA damage may be observed.

The reaction are shown as follows:

Fatty acid on saponification leads to dissolution of organic tissue resulting in formation of soap and glycerol.

Sodium hypochlorite plus water (pH = 11.5 minus 11.7 gives sodium hydroxide and hypochloric acid which further give sodium plus hydroxide plus hydrogen plus chlorine.

Amino acid plus sodium hydroxide gives salt in an irreversible enzymatic inhibition reaction.

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Hypochloric acid, chloramine (cellular metabolism) and water on chlorination gives amino acid in an irreversible enzymatic inhibition.

Neutralization of amino acids: Calcium oxide plus water (pH = 12.5) yields calcium hydroxide which further gives calcium and hydroxide resulting in damage to the cell membrane, denaturalization of proteins and D N A damage.

Source: (Courtesy Dr. A. Manzur.)

Allergic reactions to sodium hypochlorite.

Although few reports have been published[127,180] on allergic reactions to NaOCl, real allergies to NaOCl are unlikely to occur, since both Na and Cl are essential elements in the physiology of the human body. It must be remembered that the hypochlorous acid (the active component of the sodium hypochlorite) is a chemical substance that is elaborated by neutrophils in the process of phagocytosis; it may create local tissue damage when it is produced in excess (liquefaction necrosis: purulent exudate) but does not cause allergic responses. Nevertheless, hypersensitivity and contact dermatitis may occur in rare situations. A recent case report[355] describes a severe chemical burn in an endodontist's eye caused by accidental contact with 3.5% NaOCl used as an irrigant during the root canal.

When hypersensitivity to NaOCl is suspected or confirmed, chlorhexidine (CHX) should not be used either because of its chlorine content. Individuals who genetically have more possibilities than the average population to generate allergies to multiple elements (allergies to food or latex) a skin test for both NaOCl and CHX may be indicated. The use of an alternative irrigant with high antimicrobial efficacy, such as iodine potassium iodide (IKI), should be considered, assuming there is no known allergy to that irrigant. Solutions such as alcohol or tap water are less effective against microorganisms and do not dissolve vital or necrotic tissue; however, $Ca(OH)_2$ could be used as a temporary medicament because it dissolves both vital and necrotic tissue.[17,188]

Temperature.

Increasing the temperature of low-concentration NaOCl solutions improves their immediate tissue-dissolution capacity (Fig. 8.48).[504] Furthermore,

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heated hypochlorite solutions remove organic debris from dentin shavings more efficiently. Bactericidal rates for NaOCl solutions, the capacity of human pulp dissolution, and increased efficacy were detailed in several studies.[418] There are various devices to preheat NaOCl syringes (Fig. 8.49); however, it was demonstrated that as soon as the irrigant touches the root canal system, the temperature reaches the body temperature.[508] Therefore, in situ heating of NaOCl is recommended by some authors. This can be done by activating ultrasonic or sonic tips to the NaOCl inside the root canal for a couple of minutes (see irrigation timings later). Macedo et al.[265] state that the efficacy of NaOCl on dentin is improved by refreshment, ultrasonic activation, and exposure time. In that investigation, a 10°C temperature rise during ultrasonic activation was insufficient to increase the reaction rate. However, there are no clinical studies available at this point to support the use of heated NaOCl.[38,94]

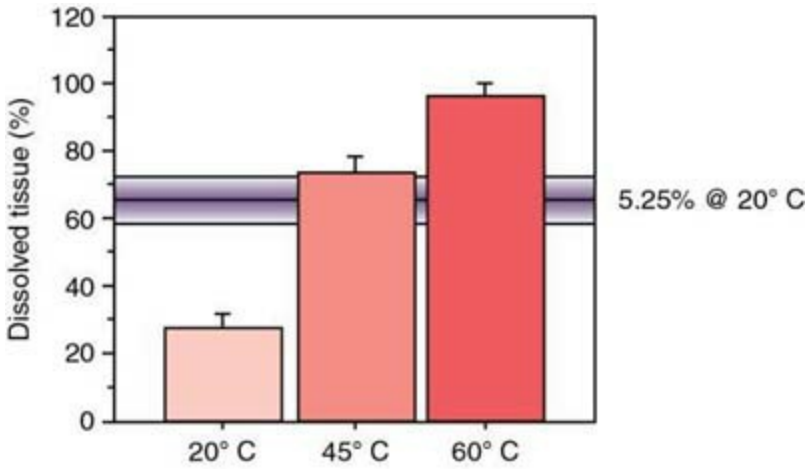

FIG. 8.48 Effect of heating on the ability of 0.5% sodium hypochlorite (NaOCl) to dissolve pulp tissue: NaOCl heated to 113°F (45°C) dissolved pulp tissue as well as the positive control (5.25% NaOCl) did. When the NaOCl was heated to 140°F (60°C), almost complete dissolution of tissue resulted.

Plot shows three bars representing degrees in Celsius versus dissolved tissue (in percentage) and increase (in percentage). The horizontal axis has three bars for 20 degree Celsius, 45 degree Celsius and 60 degree Celsius. The vertical ranges from 0 to 120 in increments of 20. The data is as follows:

20: 29, 45: 72 and 60: 95

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A state is determined at 5.25 percent at 20 degrees Celsius lies between 59 to 71 percent of dissolved tissue.

Source: (Modified from Sirtes G, Waltimo T, Schaetzle M, Zehnder M: The effects of temperature on sodium hypochlorite short-term stability, pulp dissolution capacity, and antimicrobial efficacy, *J Endod* 31:669–671, 2005.)

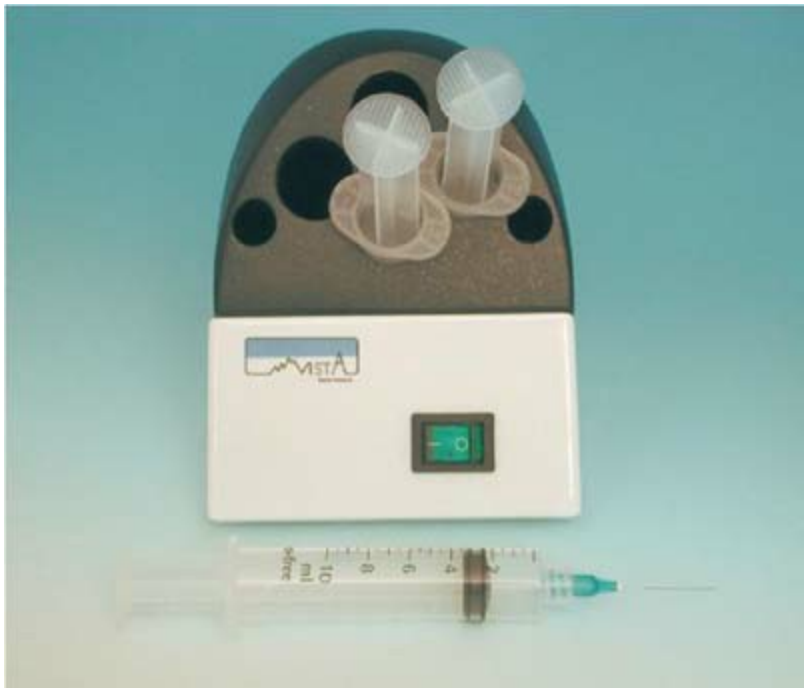

FIG. 8.49 Device for heating syringes filled with irrigation solution (e.g., sodium hypochlorite) before use.

A device for heating syringes show a power panel with two inserted syringes displaying the piston sleeve and another syringe lying laterally in front.

Source: (Courtesy Vista Dental Products, Racine, WI.)

Concentration.

NaOCl is used in concentrations between 0.5% and 6% for root canal irrigation. Controversy exists over recommended concentrations of sodium hypochlorite during root canal treatment. Several studies have tried to evaluate the influence of different concentrations of NaOCl on various aspects of the treatment such as antimicrobial efficacy, tissue dissolution,

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penetration into dentin, capacity to remove the smear layer, decalcification of dentin, microhardness, viability of stem cells in the apical region, viscosity, amount of debris extrusion, tooth surface strain, and postoperative pain. The results of these studies have been conflicting in nature. Some in vitro studies have shown that NaOCl in higher concentrations is more effective against *Enterococcus faecalis* and *Candida albicans*.[159,349,478] In contrast, clinical studies have indicated both low and high concentrations to be equally effective in reducing bacteria from the root canal system.[77,100] NaOCl in higher concentrations has a better tissue-dissolving ability.[186] However, in lower concentrations when used in high volumes, it can be equally effective.[290,413] Higher concentrations of NaOCl are more toxic than lower concentrations[430]; however, perhaps due to the anatomy of the root canal system, higher concentrations have successfully been used during root canal treatment, with a low incidence of problems. Higher concentrations cause more cytotoxicity, erosion of dentin, collagen degradation, decalcification, dentin deproteination, and tooth surface strain while reducing microhardness, elastic modulus, and flexural strength of dentin.[468] In the root canal environment, using higher NaOCl concentrations does not necessarily change outcomes, because a high volume of irrigation and frequent exchanges can compensate for the effects of the concentration.[149]

Time.

Retention time and the volume of irrigation are essential factors influencing the antibacterial activity of NaOCl.[149] There is conflicting evidence regarding the time course of the antibacterial effect of NaOCl.[38,176] In some articles hypochlorite kills the target microorganism in seconds, even at low concentrations, while other reports have published considerably longer times for the killing of the same species.[160] Such differences are likely a result of several factors such as irrigant concentration and presence of organic matter (inflammatory exudates, hard and soft tissue remnants and microbial biomass).

Several studies showed[174,291] that the presence of dentin caused marked delays in the killing of *E. faecalis* by 1% NaOCl. Many of the earlier studies were performed in the presence of an unknown amount of organic matter. When such confounding factors are eliminated, it has been shown that NaOCl

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kills the target microorganisms rapidly, even at low concentrations of less than 0.1%.[176,471] However, in vivo, the presence of organic matter consumes NaOCl and weakens its effect. Therefore continuous replenishing of irrigation solution and allowing sufficient contact time are important factors for the effectiveness of NaOCl.[176]

The chlorine ion, which is responsible for the dissolving and antibacterial capacity of NaOCl, is unstable and consumed rapidly (probably within the first 2 minutes) during the first phase of tissue dissolution,[290] which provides another reason for continuous replenishment. This should especially be considered because rotary root canal preparation techniques have expedited the shaping process. The optimal time that a hypochlorite irrigant at a given concentration needs to remain in the canal system is an issue yet to be resolved.[504] Hypochlorite should be used throughout the instrumentation, and for 1 to 2 minutes after completing the instrumentation.[177]

Toxicity.

If NaOCl is used without caution, it is very toxic and destructive to intraoral soft tissues, the periradicular vasculature, and cancellous bone where it can elicit severe inflammatory responses and degradation of organic components. Because of its high pH and strong oxidizing capacity, complete lysis of erythrocytes can occur at low concentration.

Even though the incident of NaOCl accident is reported as low, the actual frequency of such accidents is unknown, as many of them may not be reported, while minor extrusion incidents might even remain undetected due to the absence of severe symptoms.

A survey conducted on 314 diplomates of the American Board of Endodontics indicated that only 132 members reported experiencing a NaOCl accident.[228] Significantly more women experienced sodium hypochlorite accidents compared with men. More maxillary teeth than mandibular teeth and more posterior than anterior teeth were involved. A diagnosis of pulp necrosis with radiographic findings of periradicular radiolucency was positively associated with such accidents. Most respondents reported that patient signs and symptoms entirely resolved within a month. The occurrence of an accident, by itself, did not adversely affect the endodontic prognosis of the involved tooth. Anatomic variations may contribute significantly to the

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occurrence of a sodium hypochlorite accident.[228]

It is essential to recognize the symptoms and act accordingly. Accidental extrusion of NaOCl into the tissue results in severe pain, edema of neighboring soft tissues, possible extension of edema over injured half side of face/upper lip, profuse bleeding from root canal, profuse interstitial bleeding with hemorrhage of skin and mucosa (ecchymosis), chlorine taste, and irritation of throat after injection into maxillary sinus; secondary infection or even reversible anesthesia or paresthesia is possible.

NaOCl is cytotoxic and a potent dissolver of organic matter. This is the reason why edema may be perceived as a protective tissue response when a hyperosmotic and cytotoxic liquid is extruded into the vicinity of periradicular tissues. The ecchymosis involves the dissolution of blood vessel walls and hemorrhage around the subcutaneous soft tissues. Also, it is a potent oxidant, and the mechanism of injury from undiluted NaOCl is primarily oxidation of proteins. Depending on the concentration employed for root canal irrigation, NaOCl may also be very hypertonic (2800 mOsm/kg for 3% to 6% solutions) concerning tissue fluids; this partially accounts for the rapid appearance of edema during a NaOCl accident.[510]

If NaOCl is inadvertently extruded through the apex, accidents may occur (Fig. 8.50, *A–C*). For the management of these lesions, it is recommended to inform the patient and to control pain with local anesthesia and analgesics and close follow-up in the hours and days after the accident. To gain rapid pain relief, anesthetic infiltration had been attempted. However, if swelling is present, infiltration anesthesia is contraindicated to avoid spreading of any existing infection; a nerve block should be used instead.[167] Very little information was provided regarding the use of vasoconstrictors and the location of the injection attempted for pain relief. Theoretically, vasoconstrictors might limit the diffusion of NaOCl, but this would likely increase the risk of promoting tissue necrosis, especially with highly concentrated solutions promoting local ischemia.[167]

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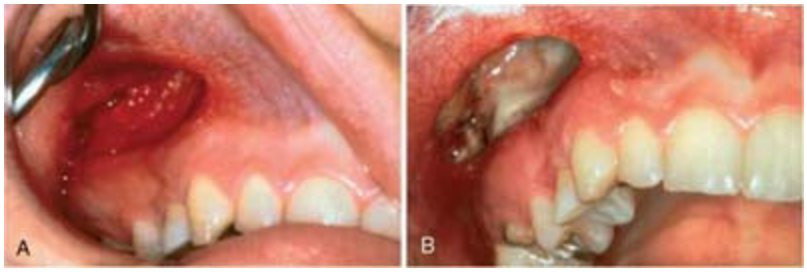

FIG. 8.50 Toxic effect of sodium hypochlorite on periradicular tissues. After root canal treatment of tooth #3, the patient reported pain. **A,** On a return visit, an abscess was diagnosed and incised. **B,** Osteonecrosis was evident after 3 weeks.

A) Close-up of an open mouth with a gauge exposes a lesion over lateral set of teeth.

B) Close-up of an open mouth shows a lesion over lateral set of teeth. The lesion exposes underlying teeth anatomy.

Some reports used saline to dilute the NaOCl or even CHX, but the latest should be avoided to prevent the formation of the precipitate (see later).

In general, postextrusion management is ambulatory using only oral medications. However, it was reported that about one third of patients (18/52) were hospitalized for monitoring and intravenous administration of drugs.[227] The application of extraoral cold compresses for reduction of swelling is also useful. After 1 day, warm compresses and frequent warm mouth rinses for stimulation of local systemic circulation should be used. Patients should be recalled daily to monitor recovery. The use of antibiotics is not obligatory and recommended only in cases of high risk or evidence of secondary infection. The administration of antihistamine is also not obligatory, and the use of corticosteroids is controversial. Further endodontic therapy with sterile saline or CHX as root canal irrigants and referral to hospital in case of worsening symptoms were suggested.[227]

Permanent sequelae could be divided into nerve lesions and scar tissues. Neurologic examination of the trigeminal and facial nerves should systematically be performed once anesthesia has dissipated. Tooth loss has not been reported as a direct result of NaOCl extrusion, but it may be involved. The latter is a real trauma for the patient, and it can lead to subsequent refusal to achieve endodontic treatment.[167]

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Chlorhexidine

History.

CHX was developed in the United Kingdom and first marketed as an antiseptic cream.[131] It has been used for general disinfection purposes and the treatment of skin, eye, and throat infections in both humans and animals.[131,257] It has been used as an irrigant and medicament in endodontics for more than a decade.[266,286,315]

Molecular structure.

The CHX molecule is a strong base with a pH between 5.5 and 7 that belongs to the polybisguanide group; it consists of two symmetric four-chlorophenyl rings and two bisguanide groups connected by a central hexamethylene chain. CHX digluconate salt is easily soluble in water and very stable (Fig. 8.51).[165]

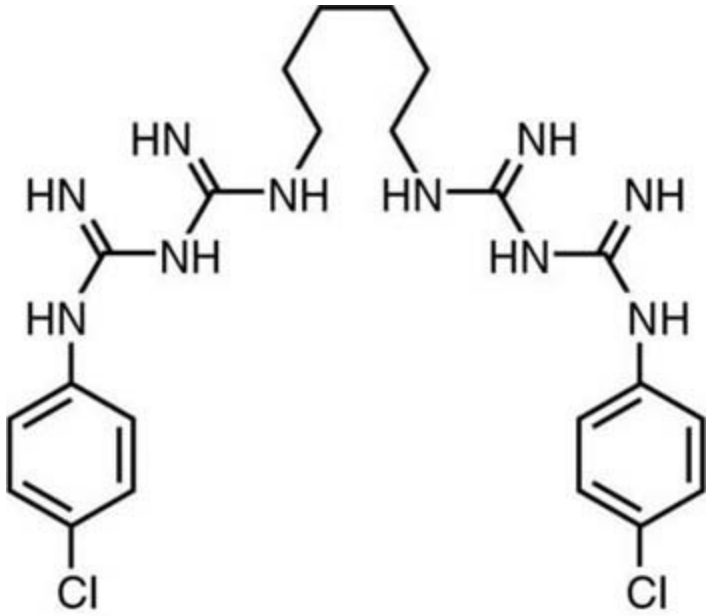

FIG. 8.51 Schematic drawing of the chlorhexidine molecule.

Molecular structure of chlorhexidine is shown.

Mode of action.

CHX, because of its cationic charges, is capable of electrostatically binding

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to the negatively charged surfaces of bacteria,[105] damaging the outer layers of the cell wall and rendering it permeable.[204] CHX is a wide-spectrum antimicrobial agent, active against gram-positive, gram-negative bacteria and yeasts.[115]

Depending on its concentration, CHX can have both bacteriostatic and bactericidal effects. At high concentrations, CHX acts as a detergent, and exerts its bactericidal effect by damaging the cell membrane and causes precipitation of the cytoplasm. At low concentrations, CHX is bacteriostatic, causing low molecular-weight substances (i.e., potassium and phosphorous) to leak out without the cell being permanently damaged.[39]

Substantivity.

Unlike NaOCl, CHX is capable of remaining in the dentin because of the cationic nature of its molecule. Its adsorption to dental hard tissues with gradual and prolonged release at therapeutic levels is usually called the residual effect or substantivity. This effect depends on the concentration of CHX and the contact time with the dentin; moreover, it has been detected in root canal dentin up to 120 days.[427] Komorowski et al.[233] showed that 5-minute application of CHX did not induce substantivity on dentin. Thus a short time inside the root canal can interfere with the adsorption of CHX on dentin.

At low concentrations of 0.005% to 0.01%, only a constant monolayer of CHX is adsorbed on the tooth surface, but at higher concentrations (0.02%) a multilayer of CHX is formed on the surface, providing a reservoir of CHX that can rapidly release the excess into the environment as the concentration of CHX in the surrounding environment decreases.[123] Time and concentration of CHX can influence the antibacterial substantivity, and the conclusions are inconsistent. Some studies demonstrated that 4% CHX has greater antibacterial substantivity than 0.2% after 5 minutes application.[286] Other studies stated that CHX should be left for more than 1 hour in the canal to be adsorbed by the dentin.[1] Komorowski et al.[233] suggested that a 5-minute application of CHX did not induce substantivity, so dentin should be treated with CHX for 7 days. However, when Paquette and Malkhassian[316] in their in vivo study medicated the canals with either liquid or gel forms of CHX for 1 week, neither of them could achieve complete disinfection.

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Therefore, residual antimicrobial efficacy of CHX in vivo remains to be demonstrated.

Cytotoxicity.

In the medical field, CHX is typically used at concentrations between 0.12% and 2.0%. According to Löe,[257] at these concentrations CHX has a low level of tissue toxicity, both locally and systemically. In another report, when 2% CHX was used as a subgingival irrigant, no apparent toxicity was noted on gingival tissues.[258,426] Moreover, CHX rinses have been reported to promote the healing of periodontal wounds.[24] Based on these reports, Jeansonne et al.[215] assumed that the periapical tissues would be as tolerant of CHX as gingival tissues. In two studies when CHX and NaOCl were injected into subcutaneous tissues of guinea pigs and rats, an inflammatory reaction developed; however, the toxic reaction from CHX was less than that of NaOCl.[306] Furthermore, when CHX was applied as a rinse in the extraction sites of third molars on the day of surgery and several days after, it was reported to reduce the incidence of alveolar osteitis.[80] Few reports of allergic and anaphylactic reactions to CHX exist in the literature.[146,305]

Conversely, some studies have reported unfavorable effects of CHX on tissues. Hidalgo and Dominguez[197] demonstrated that CHX is cytotoxic to some lines of cultured human skin fibroblasts. Recently the behavior of osteoblastic human alveolar bone cells in the presence of CHX and povidone-iodine (PI) has been investigated. It has been reported that CHX has a higher cytotoxicity profile than povidone iodine.[78] Faria et al.[132] also demonstrated that CHX injected in the hind paw of mice could induce severe toxic reactions. They reported that CHX induced apoptosis at lower concentrations and necrosis at higher concentrations when added to cultured L929 fibroblast cells.

A byproduct of combining CHX with NaOCl is the formation of toxic products such as parachloroaniline (PCA) that may harm tissues.[42] The toxicity level of CHX on periapical tissues when applied in the root canals, especially with other irrigants, merits further investigation.

Chlorhexidine as an endodontic irrigant.

CHX has been extensively studied as an endodontic irrigant and intracanal

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medication, both in vivo[33,255,268,316,494] and in vitro.[40,41,215,233,248,412,443,472] The antibacterial efficacy of CHX as an irrigant is concentration dependent. It has been demonstrated that 2% CHX has better antibacterial efficacy than 0.12% CHX in vitro.[41] When compared with the effectiveness of NaOCl, controversial results can be found. NaOCl has an obvious advantage over CHX with the dissolution capacity of organic matter that CHX lacks; therefore, even though in vitro studies suggest some advantages with the use of CHX, as soon as organic and dental tissue is added, NaOCl is clearly preferable.

When studied in vivo, investigators[359] reported that 2.5% NaOCl was significantly more effective than 0.2% CHX when the infected root canals were irrigated for 30 minutes with either of the solutions. In a controlled and randomized clinical trial, the efficacy of 2% CHX liquid was tested against saline using culture technique. Their results showed better disinfection of the root canals using CHX compared with saline as a final rinse.[503]

In a recent study, the antibacterial efficacy of 2% CHX gel was tested against 2.5% NaOCl in teeth with apical periodontitis, with the bacterial load assessed using real-time quantitative polymerase chain reaction (RTQ-PCR) and colony forming units (CFU). The bacterial reduction in the NaOCl group was significantly higher than the CHX group when measured by RTQ-PCR. Based on culture technique, bacterial growth was detected in 50% of the CHX-group cases compared with 25% in the NaOCl group.[472] On the other hand, another study based on this culture technique revealed no significant difference between the antibacterial efficacy of 2.5% NaOCl and 0.12% CHX liquid when used as irrigants during the treatment of infected canals.[414]

In a systematic review, Ng and colleagues[301] demonstrated that abstaining from using 2% CHX as an adjunct irrigant to NaOCl was associated with superior periapical healing. In summary, CHX lacks a tissue-dissolving property; therefore, NaOCl is still considered the primary irrigating solution in endodontics.

Interaction between CHX, NaOCL, and EDTA.

NaOCl and CHX when in contact, produce a change of color and a precipitate (Fig. 8.52). The reaction is dependent on the concentration of NaOCl. The higher the concentration of NaOCl, the larger the precipitate is if

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2% CHX is used.[42] Furthermore, concerns have been raised that the color change may have some clinical relevance because of staining, and the resulting precipitate might interfere with the seal of the root obturation. Basrani et al.[42] evaluated the chemical nature of this precipitate and reported the formation of 4-chloroaniline (PCA). Furthermore, a recent study[232] using a specific mass spectrometry analysis (TOFSIMS), demonstrated the penetration of PCA inside dentinal tubules. PCA is toxic in humans with short-term exposure, resulting in cyanosis, which is a manifestation of methemoglobin formation. The combination of NaOCl and CHX causes color changes and formation of a possibly toxic insoluble precipitate that may interfere with the seal of the root obturation. Alternatively, the canal can be dried using paper points before the final CHX rinse.[504]

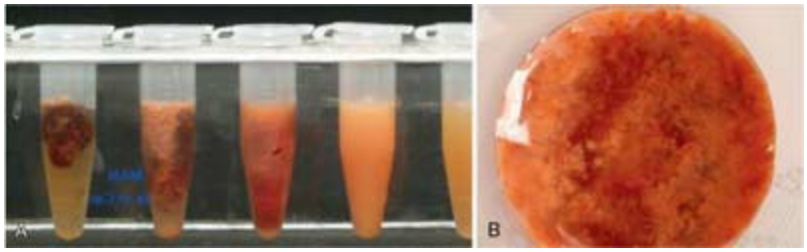

FIG. 8.52 Red precipitate forming after contact between NaOCl and chlorhexidine. **A,** When 2% chlorhexidine (CHX) is mixed with different concentration of NaOCl, a change of color and precipitate occurs. The higher the concentration of NaOCl, the larger is the precipitate formation. **B,** Detail of the interaction between 2% CHX and 5% NaOCl.

A) Five test-tubes with liquids show first test-tube with unmixed red precipitate at the top and yellow at lower, second test-tube with partially mixed red precipitate at the top and yellow at lower, third test-tube with almost mixed red and yellow precipitates, fourth test-tube with reddish yellow mixture and fifth test-tube with almost yellow mixture.

B) Close-up of an laterally filled test-tube with reddish yellow pigmented liquid.

The combination of CHX and EDTA produces a white precipitate, so a group of investigators[351] did a study to determine whether the precipitate involves the chemical degradation of CHX. The precipitate was produced and re-dissolved in a known amount of dilute trifluoroacetic acid. Based on the results, CHX was found to form a salt with EDTA rather than undergoing a

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chemical reaction.

Allergic reactions to chlorhexidine.

Allergic responses to CHX are rare and there are no reports of reactions following root canal irrigation with CHX.[209] The sensitization rate has been reported in several studies to be approximately 2%.[236] However, some allergic reactions such as anaphylaxis, contact dermatitis, and urticaria have been reported following direct contact to mucosal tissue or open wounds.[342,391,472] Patients with history of atypical reaction to any irrigant should undergo a basic immunologic evaluation before CHX or NaOCl are used. If any of these tests is positive, alternative substance should be use.[104]

Decalcifying agents.

Debris is defined as dentin chips or residual vital or necrotic pulp tissue attached to the root canal wall. Smear layer was defined by the American Association of Endodontists in 2003 as a surface film of debris retained on dentin or other surfaces after instrumentation with either rotary instruments or endodontic files; it consists of dentin particles, remnants of the vital or necrotic pulp tissue, bacterial components, and retained irrigants. While it has been viewed as an impediment to irrigant penetration into dentinal tubules (Fig. 8.53), there is still a controversy about the influence of smear layer on the outcome of endodontic treatment. Some researchers emphasize the importance of removing the smear layer to allow irrigants, medications, and sealers to penetrate dentinal tubules and improve disinfection. On the other hand, other researchers focused on keeping the smear layer as protection for bacterial invasion, apical and coronal microleakage, bacterial penetration of the tubules, and the adaptation of root canal materials. The majority of the conclusions on the smear layer are based on in vitro studies. A recent clinical study by Ng et al.[301] found that the use of EDTA significantly increased the odds of success of retreatment.

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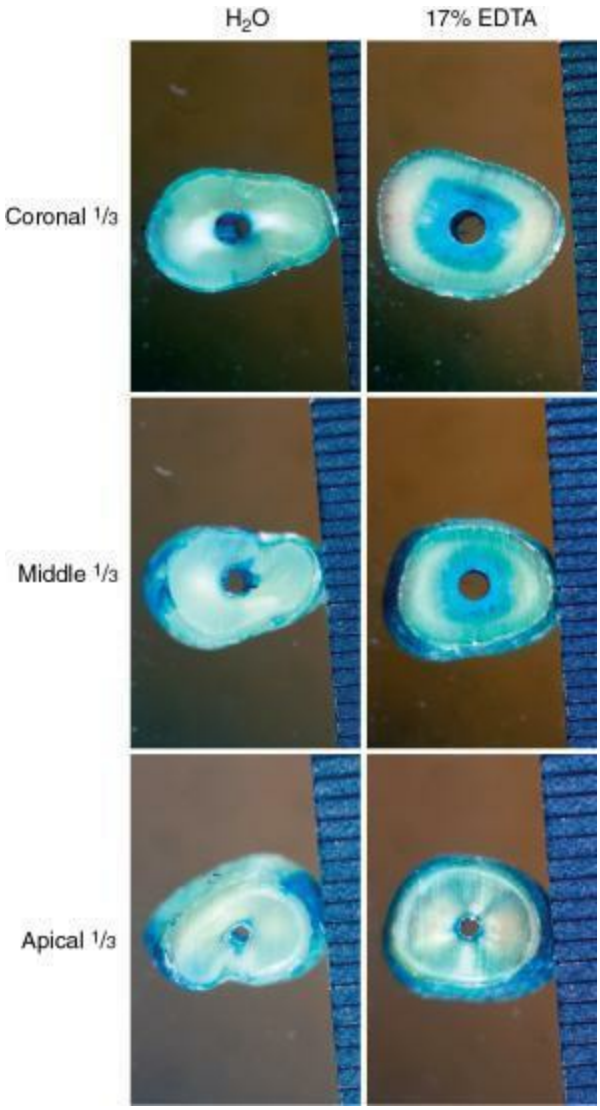

FIG. 8.53 Penetration of irrigants into dentinal tubules after root canal preparation with different dentin pretreatments. *Left column,* Irrigation with tap water and then with blue dye. *Right column,* Smear layer is removed with 17% EDTA, applied in high volume and with a 30-gauge needle, followed by irrigation with blue dye. Note the comparable diffusion of dye in the apical sections, whereas dye penetrated deeper into the dentin in the two coronal sections.

The images show the following data:

Coronal one-third: Water: A horizontal section of coronal one third shows a hole with an outline of blue on greenish white surface.

Coronal one-third: 17 percent EDTA: A horizontal section of coronal one third shows a hole with rings of blue, green and white colors.

Middle one-third: Water: A horizontal section of middle one third shows a

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hole with an outline of blue on greenish white surface.

Middle one-third: 17 percent EDTA: A horizontal section of middle one third shows a hole with rings of blue, green and white colors with outlines of green and blue.

Apical one-third: Water: A horizontal section of apical one third shows a hole with an outline of blue on greenish white surface.

Apical one-third: 17 percent EDTA: A horizontal section of apical one third shows a hole with sectorial outlines of green and white and outlines of white, blue and green.

Ethylenediaminetetraacetic acid

Ethylenediaminetetraacetic acid, widely abbreviated as EDTA, is a polycarboxylic amino acid, and a colorless, water-soluble solid EDTA is often suggested as an irrigation solution because it can chelate and remove the mineralized portion of the smear layer (Fig. 8.54). It is a polyaminocarboxylic acid with the formula $[CH_2N(CH_2CO_2H)_2]_2$. Its prominence as a chelating agent arises from its ability to sequester di- and tricationic metal ions such as Ca^{2+} and Fe^{3+}. After being bound by EDTA, metal ions remain in solution but exhibit diminished reactivity.[433]

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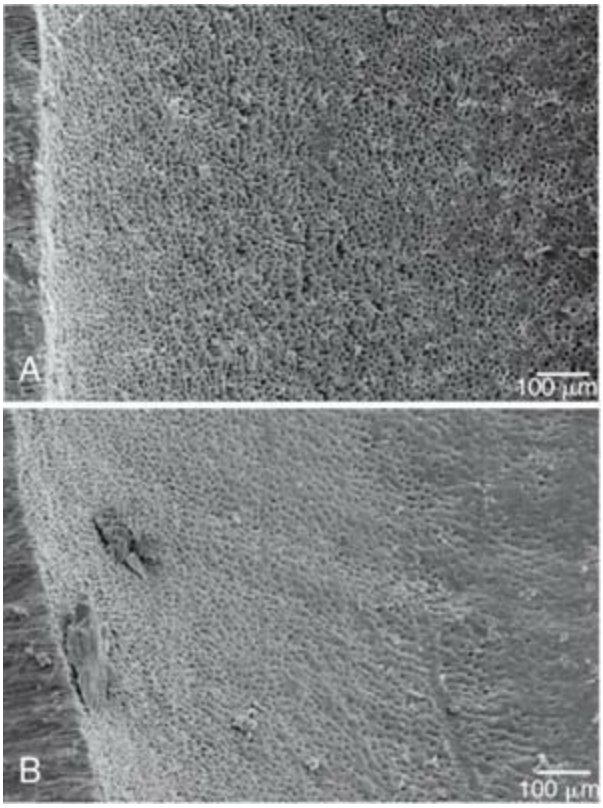

FIG. 8.54 Example of canals with minimal smear layer. **A,** Middle third after irrigation with 17% ethylenediaminetetraacetic acid (EDTA) and 2.5% sodium hypochlorite (NaOCl). **B,** Apical third with some particulate debris.

A) Micrograph of middle third with densely granuled smear layer.

B) Micrograph of apical third with granuled smear layer and large irregular debris on the surface.

History.

The compound was first described in 1935 by Ferdinand Munz, who prepared it from ethylenediamine and chloroacetic acid. Chelating agents were introduced into endodontics as an aid for the preparation of narrow and calcified root canals in 1957 by Nygaard-Østby.[206] Today, EDTA is mainly synthesized from ethylenediamine (1,2-diaminoethane), formaldehyde (methanal), and sodium cyanide.[206]

Mode of action.

On direct exposure for an extended time, EDTA extracts bacterial surface

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proteins by combining with metal ions from the cell envelope, which can eventually lead to bacterial death.[206] Chelators such as EDTA form a stable complex with calcium. When all available ions have been bound, equilibrium is formed and no further dissolution takes place; therefore, EDTA is self-limiting.[206]

Applications in endodontics.

EDTA alone usually cannot remove the smear layer effectively; a proteolytic component, such as NaOCl, must be added to remove the organic components of the smear layer.[158] For root canal preparation, EDTA has limited value alone as an irrigation fluid.[158] EDTA is generally used in a concentration of 17% and can remove the smear layers when in direct contact with the root canal wall for less than 1 minute. Even though EDTA has self-limited action, if it is left in the canal for longer or NaOCl is used after EDTA, erosion of dentin has been demonstrated (Fig. 8.55).

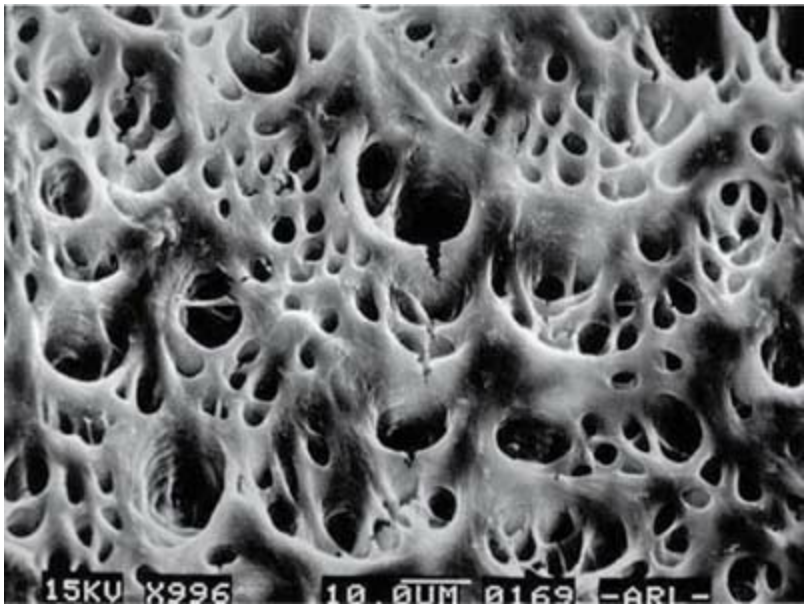

FIG. 8.55 Scanning electron microscope (SEM) image of root canal dentin exposed to 2 minutes of EDTA.

Micrograph of dentin shows thin walls with porous surface.

Although citric acid appears to be slightly more potent at similar concentration than EDTA, both agents show high efficiency in removing the

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smear layer. In addition to their cleaning ability, chelators may detach biofilms adhering to root canal walls.[168] This may explain why an EDTA irrigant proved to be highly superior to saline in reducing intracanal microbiota, even though its antiseptic capacity is relatively limited.[168] Antiseptics such as quaternary ammonium compounds (EDTAC) or tetracycline antibiotics (MTAD; see irrigants combination later) have been added to EDTA and citric acid irrigants, respectively, to increase their antimicrobial capacity. The clinical value of this, however, is questionable. EDTAC shows similar smear-removing efficacy as EDTA, but it is more caustic.

The effect of chelators in negotiating narrow, tortuous, calcified canals to establish patency depends on both canal width and the amount of active substance available since the demineralization process continues until all chelators have formed complexes with calcium.[206,506] Therefore, studies should be read with caution because one study showed demineralization up to a depth of 50 μm into dentin,[210] but other reports demonstrated significant erosion after irrigation with EDTA.[44]

A comparison of bacterial growth inhibition showed that the antibacterial effects of EDTA were stronger than citric acid and 0.5% NaOCl but weaker than 2.5% NaOCl and 0.2% CHX.[410] EDTA had a significantly better antimicrobial effect than saline solution. It exerts its strongest effect when used synergistically with NaOCl, although no disinfecting effect on colonized dentin could be demonstrated.[192]

Interaction of EDTA and NaOCL.

Investigators[164] studied the interactions of EDTA with NaOCl. They concluded that EDTA retained its calcium-complex ability when mixed with NaOCl, but EDTA caused NaOCl to lose its tissue-dissolving capacity, with virtually no free chlorine detected in the combinations. Clinically, this suggests that EDTA and NaOCl should be used separately. In an alternating irrigating regimen, copious amounts of NaOCl should be administered to wash out remnants of the EDTA. In modern endodontics, EDTA is used once the cleaning and shaping are completed for around 1 minute. It can be activated with ultrasonic activation for better penetration in dentinal tubules. It should be taken into consideration that raising the temperature of EDTA is

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not desirable, as chelators have a temperature range that they work their best. When EDTA is heated from 20 to 90 degrees, the calcium binding capacity decreases.[505]

HBPT and other phosphate bases tensides

HEBP (1-hydroxyethylidene-1, 1-bisphosphonate; also called etidronic acid) is a weak chelator.[504] It is a potential alternative to EDTA because it has no short-term reactivity with NaOCl. It can be used in combination with NaOCl without affecting its proteolytic or antimicrobial properties.[504] It is nontoxic[26,111] and it is used in medicine to treat bone diseases.[314]

Combination irrigants.

Irrigants with low surface tension are assumed to penetrate better in dentinal tubules and anatomic irregularities. Detergents (e.g., Tween 80) are added to irrigants to lower their surface tension. However, Boutsioukis and Kishen[58] did not find support for this rationale, perhaps because the effect of surface tension is important only at the interface between two immiscible fluids (e.g., between irrigant and an air bubble, but not between irrigant and dentinal fluid). Recent studies have also confirmed that surfactants do not enhance the ability of NaOCl to dissolve pulp tissue[221] or the efficacy of common chelators to remove calcium or smear layer.[58,111,506]

BioPure MTAD and Tetraclean.

Two other irrigants, based on a mixture of antibiotics, citric acid, and a detergent, are commercially available. They are capable of removing both the smear layer and organic tissue from the infected the root canal system.[454] MTAD (Fig. 8.56), introduced by Torabinejad and Johnson[455] at Loma Linda University in 2003, is an aqueous solution of 3% doxycycline, a broad-spectrum antibiotic; 4.25% citric acid, a demineralizing agent; and 0.5% polysorbate 80 detergent (Tween 80).[455] It is mixed from a liquid and powder before use. MTAD has been recommended in clinical practice as a final rinse after completion of conventional chemomechanical preparation.[44]

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FIG. 8.56 Container with BioPure MTAD.

A solution bottle for removing layers of smear and organic tissue from infected root canals with three syringes lying around.

Source: (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK, USA.)

Tetraclean (Ogna Laboratori Farmaceutici, Muggio, Italy) is a combination product similar to MTAD. The two irrigants differ in the concentration of antibiotics (doxycycline 150 mg/5 mL for MTAD and 50 mg/5 mL for Tetraclean) and the kind of detergent (Tween 80 for MTAD, polypropylene glycol for Tetraclean).

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Mode of action.

All of the tetracyclines are derivatives of the four-ringed nucleus that differ structurally regarding the chemical groups at 2, 5, 6, 7 positions. These derivatives exhibit different characteristics, such as absorption, protein binding, metabolism, excretion, and degree of activity against the susceptible organism.[191] Tetracyclines inhibit protein synthesis by reversibly binding to the 30S subunit of bacterial ribosome in susceptible bacteria. It is effective against *Aggregatibacter actinomycetemcomitans* (A.a), *Porphyromonas gingivalis*, and *Prevotella intermedia* and affects both gram-positive and gram-negative bacteria, with a more pronounced gram-negative effect. Tetracycline is a bacteriostatic antibiotic, but in high concentrations tetracycline may also be bactericidal. Doxycycline, citric acid, and Tween 80 together may have a synergistic effect on the disruption of the bacterial cell wall and the cytoplasmic membrane.

Smear layer removal.

In two studies, the efficacy of MTAD or EDTA in the removal of the smear layer was confirmed, but no significant difference between these two solutions was reported.[444,445]

Antibacterial efficacy.

Earlier independent in vitro research on MTAD showed its antimicrobial efficacy over conventional irrigants.[106,235] Torabinejad et al.[455] found that MTAD was effective in killing *E. faecalis* up to a 200× dilution. Shabahang and Torabinejad[396] showed that the combination of 1.3% NaOCl as a root canal irrigant and MTAD as a final rinse was significantly more effective against *E. faecalis* than the other regimens.[397] A study using extracted human teeth contaminated with saliva showed that MTAD was more effective than 5.25% NaOCl in disinfection of the teeth. In contrast to the previously mentioned studies, later research suggested less than the optimal antimicrobial activity of MTAD.[152,219,225] Krause et al.,[235] using bovine tooth sections, showed that 5.25% NaOCl was more effective than MTAD in disinfection of dentin discs inoculated with *E. faecalis*.[396,411]

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Clinical trials.

Malkhassian et al.[267] in a controlled clinical trial of 30 patients reported that the final rinse with MTAD did not reduce the bacterial counts in infected canals beyond levels achieved by chemomechanical preparation using NaOCl alone.

Protocol for use.

MTAD was developed as a final rinse to disinfect the root canal system and remove the smear layer. The effectiveness of MTAD to altogether remove the smear layer is enhanced when a low concentration of NaOCl (1.3%) is used as an intracanal irrigant before placing 1 mL of MTAD in a canal for 5 minutes and rinsing it with an additional 4 mL of MTAD as the final rinse.[396]

Combinations of irrigants

QMiX was introduced in 2011 as a combination product introduced for root canal irrigation (Fig. 8.57). It is recommended to be used at the end of instrumentation, after NaOCl irrigation. According to the patent,[175] QMix contains a CHX-analog, Triclosan (N-cetyl-N,N, N-trimethylammonium bromide), and EDTA as a decalcifying agent; it is intended as an antimicrobial irrigant as well as the removal of canal wall smear layers and debris.

FIG. 8.57 QMiX 2in1. Combination of CXH, EDTA, and detergent. QMix irrigating solution is a single solution used as a final rinse after bleach for one-step smear layer removal and disinfection

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Two solution bottles with a gloved hands holding a syringe dropping liquid into a measuring cap.

Source: (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK, USA).

Protocol.

QMiX is suggested as a final rinse. If sodium hypochlorite was used throughout the cleaning and shaping, saline could rinse out NaOCl remnants to prevent the formation of any precipitate.

Smear layer removal.

Stojic et al.[438] investigated the effectiveness of smear layer removal by QMiX using scanning electron microscopy. QMiX removed smear layer equally well as EDTA. Dai et al.[102] examined the ability of two pH versions of QMiX on the removal of canal wall smear layers and debris using an open canal design. Within the limitations of an open canal design, the two experimental QMiX versions are as useful as 17% EDTA in removing canal wall smear layers after the use of 5.25% NaOCl as the initial rinse.

Antibacterial efficacy and effect on biofilms.

Stojic et al.[438] also assessed the in vitro efficacy of QMiX against *E. faecalis* and mixed plaque bacteria, both in the planktonic phase and biofilms. QMiX and 1% NaOCl killed all planktonic *E. faecalis* and plaque bacteria in 5 seconds. QMiX and 2% NaOCl killed up to 12 times more biofilm bacteria than 1% NaOCl ($P < .01$) or 2% CHX ($P < .05$; $P < .001$). Wang et al.[480] compared the antibacterial effects of different disinfecting solutions on young and old *E. faecalis* biofilms in dentin canals using a novel dentin infection model and confocal laser scanning microscopy. Six percent NaOCl and QMiX were the most effective disinfecting solutions against the young biofilm, whereas, against the 3-week-old biofilm, 6% NaOCl was the most effective, followed by QMiX. Both were more effective than 2% NaOCl and 2% CHX. Morgental et al.[291] showed that QMiX was less effective than 6% NaOCl and similar to 1% NaOCl in bactericidal action. According to their in vitro study, it appears that the presence of dentin slurry has the potential to

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inhibit most current antimicrobials in the root canals system.

Moreover, Ordinola et al.[307] found that several endodontic irrigants containing antimicrobial compounds such as chlorhexidine (QMiX), cetrimide, maleic acid, iodine compounds, or antibiotics (MTAD) lacked an effective antibiofilm activity when the dentin was infected intraorally. The irrigant solutions 4% peracetic acid and 2.5% to 5.25% sodium hypochlorite decreased the number of live bacteria significantly in biofilms, providing also cleaner dentin surfaces ($P < .05$). They concluded that several chelating agents containing antimicrobials could not remove nor kill significantly biofilms developed on intraorally infected dentin, except for sodium hypochlorite and 4% peracetic acid. Dissolution ability is mandatory for an appropriate eradication of biofilms attached to dentin.

Clinical trials.

The efficacy and biocompatibility of QMiX were as yet only demonstrated via nonclinical in vitro and ex vivo studies. Further clinical research from independent investigators is needed to corroborate the findings.

Iodine potassium iodide.

IKI is a root canal disinfectant that is used in concentrations ranging from 2% to 5%. IKI kills a broad spectrum of microorganisms found in root canals but shows relatively low toxicity in experiments using tissue cultures.[430] Iodine acts as an oxidizing agent by reacting with free sulfhydryl groups of bacterial enzymes, cleaving disulphide bonds. *E. faecalis* often is associated with therapy-resistant periapical infections (see Chapter 16), and combinations of IKI and CHX may be able to kill $Ca(OH)_2$-resistant bacteria more efficiently. One study[416] evaluated the antibacterial activity of a combination of $Ca(OH)_2$ with IKI or CHX in infected bovine dentin blocks. Although $Ca(OH)_2$ alone was unable to destroy *E. faecalis* inside dentinal tubules, $Ca(OH)_2$ mixed with either IKI or CHX effectively disinfected dentin. Others[31] demonstrated that IKI was able to eliminate *E. faecalis* from bovine root dentin when used with 15-minute contact time. A distinct disadvantage of iodine is a possible allergic reaction in some patients. Although iodine is not generally considered an allergen, some patients are hypersensitive to this compound

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and may be considered to have an iodine “allergy.”

Intracanal medication

Intracanal medication is used when treatment cannot be completed in one appointment (see Chapters 5 and 15). The surviving intracanal bacteria after cleaning and shaping may increase between appointments.[76,477] The main objectives for the intracanal medication are restrict bacterial regrowth, supply continued disinfection, and create a physical barrier.

Calcium hydroxide

Calcium hydroxide is the most popular intracanal medication use. It was introduced by Hermann[194] in 1920. Although its use was well documented for its time, evidence of its efficacy in clinical endodontics is controversial. A series of articles[76,77] promoted the antibacterial efficacy of $Ca(OH)_2$ in human root canals. Subsequent studies substantiated these reports,[308,420] and the routine use of $Ca(OH)_2$ as an interappointment intracanal medicament became widespread.[373]

However, newer clinical studies and systematic reviews failed to show a clear benefit of $Ca(OH)_2$ to further eliminate bacteria from the root canal.[325,373] $Ca(OH)_2$ mostly is used as a slurry of $Ca(OH)_2$ in a water base; at body temperature, less than 0.2% of the $Ca(OH)_2$ is dissolved into Ca++ and OH ions. Because $Ca(OH)_2$ needs water to dissolve, water should be used as the vehicle for the $Ca(OH)_2$ paste. In contact with air, $Ca(OH)_2$ forms calcium carbonate ($CaCO_3$). However, this is a lengthy process and of little clinical significance. $Ca(OH)_2$ paste, with a significant amount of calcium carbonate, feels granular because the carbonate has a very low solubility.[367,420]

$Ca(OH)_2$ is a slow-acting antiseptic; direct-contact experiments in vitro show that a 24-hour contact period is required for complete killing of enterococci.[367,420] Another study of 42 patients found that NaOCl canal irrigation reduced the bacteria level by only 61.9%, but use of $Ca(OH)_2$ in the canals for 1 week resulted in a 92.5% reduction.[403] These researchers

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concluded that $Ca(OH)_2$ should be used in infected cases to more predictably obtain débridement and decontamination.

In addition to killing bacteria, $Ca(OH)_2$ has the useful ability to hydrolyze the lipid moiety of bacterial lipopolysaccharides (LPS), thereby inactivating the biologic activity of the lipopolysaccharide and reducing its effect.[368] This is a desirable effect because dead cell wall material remains after the bacteria have been killed and can continue to stimulate inflammatory responses in the periradicular tissue.

The main characteristics of $Ca(OH)_2$ include limited solubility, high pH, broad-spectrum antimicrobial agent, and antimicrobial action sustained for long periods.

Other uses of $Ca(OH)_2$.

Long-term calcium hydroxide treatment can be used to induce apexification of the immature tooth with pulpal necrosis before placing an obturation material such as gutta-percha in the root canal system.[137] Also, in cases where revascularization/guided pulpal repair is desired, $Ca(OH)_2$ may be used instead of antibiotic pastes (for more details, see later and Chapter 12).

Clinical protocol.

Calcium hydroxide should be in contact with the tissue to act. $Ca(OH)_2$ powder may be mixed with sterile water or saline and placed into the canal using a lentulo paste filler.[324] Alternatively, the mix may be applied from sterile, single-dose packages (e.g., Calasept [J.S. Dental, Ridgefield, CT, USA]; Calcijet [Centrix, Shelton, CT, USA]; and DT Temporary Dressing [Global Dental Products, North Bellmore, NY, USA]; Fig. 8.58). The mixture should be thick to carry as many $Ca(OH)_2$ particles as possible; however, it should not be overdried to retain enough moisture and to promote continued dissociation with a resulting high pH. For maximum effectiveness, the root canal should be filled homogeneously to the WL with a slurry, and a radiograph should be taken to confirm this.

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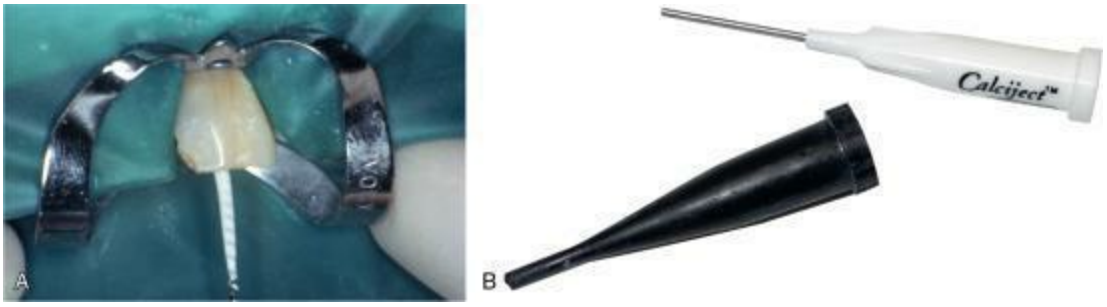

FIG. 8.58 **A,** Application of calcium hydroxide paste in the canal with a lentulo spiral. **B,** Calciject is a calcium hydroxide prefilled, easy-to-use, single-dose syringe system. Centrix NeedleTube cartridges can be used for direct syringe injection into the root canal.

A) Close-up of a rubber dam and clamp exposing a tooth with a vertical outline of lentulo spiral instrument.

B) Close-up of a single dose injection system shows an injection barrel, a plain tip and a cap.

Source: (*A,* Courtesy Dr. S. Friedman. *B,* Courtesy Centrix, Shelton, CT, USA.)

Accidents with $Ca(OH)_2$ placement have been reported and the dentist should be aware of them, and because it is a strong base that may cause irreversible injury to vital tissue that comes into contact with this substance, specifically nerve damage may occur. Therefore, care must be taken not to extrude $Ca(OH)_2$ through the apex, as this carries the risk of reaching the maxillary sinus or the mandibular canal. Calcium hydroxide can also cause blindness when it comes into contact with the eye. Clinicians should take adequate precautions to prevent this serious complication.[256]

Limitations of calcium hydroxide.

There are some concerns regarding the use of $Ca(OH)_2$. The handling and proper placement of $Ca(OH)_2$ present a challenge to the average clinician.[417] Moreover, the removal of $Ca(OH)_2$ is frequently incomplete, resulting in a residue covering 20% to 45% of the canal wall surfaces, even after copious irrigation with saline, NaOCl, or EDTA.[242] Residual $Ca(OH)_2$ can shorten the setting time of zinc oxide eugenol–based endodontic sealers.[271] Notably, it may interfere with the seal of the root filling and compromise the quality of

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treatment. An additional concern is that $Ca(OH)_2$ is not effective against several endodontic pathogens, including *E. faecalis* and *C. albicans*.[478] In fact, in vitro studies have shown that dentin can inactivate the antibacterial activity of $Ca(OH)_2$,[174,347] and one clinical study[325] demonstrated that the number of bacteria-positive canals increased after $Ca(OH)_2$ medication. Other studies have further indicated that $Ca(OH)_2$ could not predictably eliminate bacteria or that cultures changed from negative to positive after $Ca(OH)_2$ placement.[325,477]

When different studies report inconsistent results, a systematic review and meta-analysis technique can clarify conflicting research data and the current state of knowledge regarding specific issues. Therefore, based on the current best available evidence, $Ca(OH)_2$ has limited effectiveness in eliminating bacteria from human root canals when assessed by culture techniques. The quest for better antibacterial protocols and sampling techniques must continue to ensure that bacteria can be reliably eradicated before obturation.

Chlorhexidine

In vitro, CHX used as intracanal medicament has at least as good or even better antimicrobial efficacy than $Ca(OH)_2$.[411] Notably, 2% CHX was very effective in eliminating a biofilm of *E. faecalis*.[253] In vivo, it inhibits experimentally induced inflammatory external root resorption when applied for 4 weeks.[255] In infected root canals, it reduces bacteria as effectively as $Ca(OH)_2$ when applied for 1 week.[33] Unlike $Ca(OH)_2$, CHX has a substantive antimicrobial activity that, if imparted onto the root dentin, has the potential to prevent bacterial colonization of root canal walls for prolonged periods.[215,233] This effect depends on the concentration of CHX, but not on its mode of application, which may be as a liquid, gel, or controlled-release device.[40]

CHX as intracanal medication has been the focus of many in vitro[36,114,233,247,411] and in vivo studies.[33,255,268,316,509] The results of in vitro experiments were mostly in favor of CHX regardless of its mode of application. In vivo, variable results were reported; one of the reasons for this is that researchers[174] developed an experimental model using dentin powder

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particles to investigate the possible inactivation of some antibacterial medicaments when they come in contact with dentin. They showed that dentin powder had inhibitory effects on all medicaments tested. The effect was dependent on the concentration of the medicament and the duration of contact. The effect of $Ca(OH)_2$ was abolished by the presence of dentin powder. The effect of 0.05% CHX and 1% NaOCl was reduced but not eliminated by the presence of dentin.

In vitro and animal studies suggest that CHX has the potential to replace $Ca(OH)_2$ as an intracanal medicament, but the listed limitations of the in vitro experiments make the conclusions obtained challenging to extrapolate to actual clinical situations. In vivo, human studies are therefore required to test the efficacy of CHX as an intracanal medication.

Compared with the number of in vitro studies, very few clinical studies have been conducted to assess the effectiveness of CHX as an intracanal medication. An in vivo investigation[33] assessed the antibacterial efficacy of three different intracanal medications: camphorated paramonochlorophenol, $Ca(OH)_2$, and 0.12% CHX liquid by applying them for 1 week in single-rooted teeth of patients. Using a culture method, it was reported that the proportions of positive cultures were not significantly different among the tested medications, but they were slightly lower in teeth medicated with CHX (0.12%) liquid than those medicated with camphorated paramonochlorophenol or $Ca(OH)_2$.[33]

Another in vivo study evaluated the antibacterial effectiveness of 2% CHX liquid as an intracanal medication in teeth with apical periodontitis.[316] The results showed a moderate increase in bacterial counts during a medication period of 7 to 14 days that was similar to outcomes seen and reported for $Ca(OH)_2$ by Peters et al.[325] It was speculated that the CHX liquid may have partially escaped from the apical foramen and that the higher-viscosity gel form might have been better suited as an intracanal medication.[316]

However, a different study[268] demonstrated that intracanal medication with $Ca(OH)_2$, 2% CHX gel, or a mixture of $Ca(OH)_2$/CHX applied for 7 days did not reduce the bacterial concentration beyond what was achieved after chemomechanical preparation using 1% NaOCl. The results were not significantly different among the medication groups. Similar results were

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found by other investigators,[267] when after a randomized controlled trial of 30 patients, they concluded that a final rinse with MTAD (a *m*ixture of *t*etracycline, *a*cid, and *d*etergent) and intracanal application of 2% CHX gel did not reduce bacterial counts beyond levels achieved by chemomechanical preparation using NaOCl.

Chlorhexidine mixed with calcium hydroxide.

During the last few years, researchers have studied the combination of $Ca(OH)_2$ and CHX, with the concept that their antimicrobial properties interact in a synergistic fashion that enhances their efficacy. The high pH of $Ca(OH)_2$ was unaffected when combined with CHX.[42] However, the results have not been conclusive. Some in vitro studies had reported an improved antibacterial action when both agents were combined,[41,128,504] while other studies reported contradictory results.[181]

Recent animal studies have evaluated the tissue reactions to the mix of $Ca(OH)_2$/CHX, showing that the combination exerts excellent antimicrobial properties[421] and improves healing of the periapical tissues. In vivo studies have shown that the mix is at least as good as both agents applied separately in necrotic teeth with apical periodontitis,[268] as well as in previously treated cases with persistent apical periodontitis. A more recent study utilizing a CHX-based protocol of 0.12% CHX as an irrigant followed by a 7-day intracanal medication of $Ca(OH)_2$/0.12% CHX has shown promising results. The authors concluded that chemomechanical canal shaping using 0.12% CHX solution as an irrigant significantly reduced the number of intracanal bacteria, but it failed to render the canals bacteria free. Further intracanal medication with a $Ca(OH)_2$/CHX paste significantly improved the results by reducing the number of bacteria.[408] Therefore, it seems that the usefulness of mixing $Ca(OH)_2$ with CHX remains controversial.

Phenolic preparations

Phenol (C_6H_5OH) and phenolic preparation used to be very commonly used intracanal medicament in endodontics. It was thought that because of their volatile properties, it could penetrate dentinal tubules and anatomical irregularities. However, it was later demonstrated that these compounds have

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a short life and their volatility can diffuse through the temporary fillings and also through the periapical tissue causing toxicity. Despite the severe toxicity of phenolic preparations, derivatives of phenol, such as paramonochlorophenol (C_6H_4OHCl), thymol ($C_6H_3OHCH_3C_3H_7$), and cresol ($C_6H_4OHCH_3$), remain available. Currently, $Ca(OH)_2$ or no medication is preferred.[147] Phenol is a nonspecific protoplasm poison that has an optimal antibacterial effect at 1% to 2%. Many dental preparations use much too high a concentration of phenol (e.g., in the range of 30%).[147] At such a concentration, the antimicrobial effect in vivo is lower than optimal and of short duration.[279] Derivatives of phenol are stronger antiseptics and more toxic than phenol. Phenolic compounds are available as camphorated solutions.[430] Camphoration results in a less toxic phenolic compound because it slows the release of toxins to the surrounding tissues. Studies in vitro have shown that phenol and phenol derivatives are highly toxic to mammalian cells, and their antimicrobial effectiveness does not sufficiently balance their toxicity.[430] Phenols are ineffective antiseptics under clinical conditions.[76]

Formaldehyde

Formaldehyde, used as formocresol, is highly toxic, mutagenic, and carcinogenic; however, historically it has been used extensively in endodontic therapy.[249] These formulations are still being recommended in pediatric dentistry when treating deciduous teeth. The formaldehyde component of formocresol may vary substantially between 19% and 37%. Tricresol formalin, another formaldehyde preparation, contains 10% tricresol and 90% formaldehyde.[249] All of these preparations have formaldehyde content well above the 10% generally used for fixation of pathologic specimens. Formaldehyde is volatile and releases antimicrobial vapors when applied to a cotton pellet for pulp chamber disinfection. All formaldehyde preparations are potent toxins with antimicrobial effectiveness much lower than their toxicity.[431,432] There is no clinical reason to use formocresol as an antimicrobial agent for endodontic treatment, based on what is known at this time. The alternatives are better antiseptics with significantly lower toxicity.[431,432]

Halogens

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Chlorinated solutions have been used for many years to irrigate root canals. They are also used as intracanal dressings in the form of chloramine-T, an N-chloro-tosylamide sodium salt. Iodine, in the form of IKI, is a very effective antiseptic solution with low tissue toxicity. IKI is an effective disinfectant for infected dentin and can kill bacteria in infected dentin in 5 minutes in vitro.[367] IKI releases vapors with a durable antimicrobial effect. The solution can be prepared by mixing 2 g of iodine in 4 g of potassium iodide; this mixture then is dissolved in 94 mL of distilled water. Tincture of iodine (5%) has proved to be one of the few reliable agents for disinfection of rubber dam and tooth surfaces during the preparation of an aseptic endodontic work-field.[288]

Steroids

Steroids have been used locally, within the root canal system to reduce pain and inflammation. Ledermix (Lederle Pharmaceuticals, Wolfratshausen, Germany) is a commercially available product that was developed about 1960 by Prof. André Schroeder.[390] The active ingredients are the potent anti-inflammatory corticoid triamcinolone acetonide in combination with the broad-spectrum antibiotic demeclocycline. It is an intracanal medicament paste popularly used in some countries. Ledermix paste has been advocated as an initial dressing, mainly if the patient presents with endodontic symptoms.[390] Ledermix paste contains triamcinolone acetonide as an anti-inflammatory agent, at a concentration of 1%.[229] The clinical effect is a rapid relief of pain associated with acute inflammatory conditions of the pulp and periodontium.

Ledermix paste is a nonsetting, water-soluble paste material for use as root canal medicament or as a direct or indirect pulp-capping agent. The mechanisms of action of this substance are based on inhibition of the ribosomal protein synthesis in the bacteria. The release and dentin diffusion characteristics of triamcinolone from Ledermix paste, when used as a root canal medicament, have been investigated under different conditions.[5,6] Collectively, these studies show that triamcinolone is released from Ledermix paste in the root canal and can reach the systemic circulation via diffusion through dentinal tubules, lateral canals, and the apical foramen. Also, because of its root resorption inhibition property, it was tested for replanted teeth in

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dogs. The results showed that the groups treated with Ledermix, triamcinolone, and demeclocycline had significantly more favorable healing and more remaining root structure than the group filled with gutta-percha and sealer (positive control).[85] The use of the steroid triamcinolone in combination with a zinc oxide eugenol paste has recently been popularized with the introduction of Odontopaste (Australian Dental Manufacturing, Brisbane, Australia); however, data regarding the clinical efficacy of this product are limited.

Triple-antibiotic paste

Antibiotic combinations have been studied over the past few years as a regimen during regenerative endodontic strategies. The literature is inconsistent on the effectiveness of double and triple antibiotic pastes (DAP and TAP), respectively, against mono- and multispecies biofilms. The triple-antibiotic regimen, composed of metronidazole, ciprofloxacin, and minocycline, was first tested[374] for its effectiveness against *Escherichia coli*–infected dentin in vitro. The same research group also tested its bactericidal efficacy against microbes from carious dentin and infected pulp. They found that the mixture of antibiotics is sufficiently potent to eradicate the bacteria.[448] The clinical effectiveness of the triple-antibiotic paste in the disinfection of immature teeth with apical periodontitis has been reported.[488] One potential concern of using an intracanal antibiotic paste is that it may cause bacterial resistance. In addition, intracanal use of minocycline can cause tooth discoloration, creating potential cosmetic complications. For this reason, a dual paste (metronidazole, ciprofloxacin) and, alternatively, abandonment of this protocol in favor of $Ca(OH)_2$ have been considered.[245] Another reason for such a change could be the reported high toxicity to stem cells of paste prepared from antibiotic powder,[364] and possible allergic reactions in patients, remain important concerns with this category of medicaments. With the current available evidence, it remains unclear if intracanal medicaments are effective against multispecies biofilms.[299]

Lubricants

In root canal treatment, lubricants are mostly used to emulsify and keep in suspension debris produced by mechanical instrumentation. Although

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irrigation solutions serve as lubricants for hand instrumentation, particular gel-type substances are also marketed. Two of these are wax-based RC-Prep, which contains EDTA and urea peroxide, and glycol-based Glyde. Another purported function of lubricants is to facilitate the mechanical action of endodontic hand or rotary files. A study evaluating the effects of lubrication on cutting efficiency found that tap water and 2.5% NaOCl solutions increased cutting efficiency compared with dry conditions.[501] The authors of this study cited the ability of an irrigant to remove debris as the factor for the increased efficiency. Similarly, a reduction of torque scores was found when canals in normed dentin disks were prepared with ProFile and ProTaper instruments under irrigation, but the use of a gel-type lubricant did not, resulting in similar torques as in dry, nonlubricated canals.[55,330]

In summary, irrigation is an essential step in root canal treatment to ensure disinfection. NaOCl is the irrigant of choice because of its tissue-dissolving and disinfecting properties. EDTA or other chelators should be used at the end of a procedure to remove the smear layer, followed by a final flush with NaOCl for 1 minute for maximum cleaning efficiency and to minimize dentin erosion. This strategy also minimizes the inactivation of NaOCl by chemical interactions.

Disinfection devices and techniques

Syringe delivery

Application of an irrigant into a canal using a syringe and needle allows exact placement, replenishing of existing fluid, rinsing out of larger debris particles, as well as allowing direct contact to microorganisms in areas close to the needle tip. In passive syringe irrigation, the actual exchange of irrigant is restricted to 1 to 1.5 mm apical to the needle tip, with fluid dynamics taking place near the needle outlet.[59,504] Volume and speed of fluid flow are proportional to the cleansing efficiency inside a root canal.[59] Therefore, both the diameter and position of the needle outlet determine successful chemomechanical débridement; placement close to WL is required to guarantee fluid exchange at the apical portion of the canal, but close control of insertion depth is required to avoid extrusion.[59,201] Therefore, the choice of an appropriate irrigating needle is important. Although larger-gauge needles

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allow a quicker and larger amount of fluid exchange, the wider diameter does not allow cleaning of the apical and narrower areas of the root canal system (see the discussion of irrigation dynamics earlier in this chapter; see also Fig. 8.7). Excess pressure or binding of needles into canals during irrigation with no possibility of backflow of the irrigant should be avoided under all circumstances[205] to prevent extrusion into periapical spaces. In immature teeth with wide, apical foramina or when the apical constriction no longer exists, special care must be taken to prevent irrigation extrusion and potential accidents.[100]

There are different sizes and types of irrigation needles. The size of the irrigation needle[91] should be chosen depending on the canal size and taper.[79,281] Most root canals that have not been instrumented are too narrow to be reached effectively by disinfectants, even when appropriate irrigation needles are used (see Figs. 8.7 and 8.45). Therefore, effective cleaning of the root canal must include intermittent agitation of the canal content with a small endodontic instrument[276,466] to prevent debris from accumulating in general (see Fig. 8.43) and specifically at the apical portion of the root canal.

Preparation size[281] and to some extent taper[95] determine how closely a needle can be placed to the final apical millimeters of a root canal. Open-ended needles are recommended over the end open needles to prevent extrusion of the irrigant. Some needles and suction tips may be attached to the air/water syringe to increase both the speed of irrigant flow and the volume of irrigant. Examples include the Stropko Irrigator (Vista Dental Products), an adapter that connects to the air/water syringe and accepts standard Luer-lock needle tips for irrigant removal and application as well as air-drying.

Manually activated irrigation

Liquid placed inside the root canal more effectively reaches crevices and mechanically untouched areas if it is agitated inside the root canal. Corono-apical movements of the irrigation needle,[205] stirring movements with small endodontic instruments,[276,467] and manual push-pull movements using a fitted master gutta-percha cone have been recommended.[202]

Other than conventional irrigation, additional techniques for endodontic disinfection have been proposed and tested, including laser systems and

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gaseous ozone. Specific devices for endodontic irrigation and/or disinfection have been introduced, among which are the EndoActivator System (Dentsply Sirona), passive ultrasonic irrigation (PUI), the EndoVac (Discus, Culver City, CA, USA), the Safety-Irrigator (Vista Dental Products, Racine, WI, USA), the Self-adjusting File (see earlier), photoactivation disinfection, ozone generators, and others. These devices use pressure, vacuum, oscillation, and a combination of suction.

Sonically activated irrigation

The EndoActivator System uses safe, noncutting polymer tips in an easy-to-use subsonic handpiece to quickly and vigorously agitate irrigant solutions during endodontic therapy (Fig. 8.59).

FIG. 8.59 The EndoActivator, a sonic frequency system.

Close-up of endoactivator handpiece with an activator tip fixed in the front with three microholes.

Source: (Courtesy Dentsply Tulsa Dental Specialties, Tulsa, OK, USA.)

In a study,[116] the safety of various intracanal irrigation systems was analyzed by measuring the apical extrusion of irrigant. The authors concluded that EndoActivator had a minimal statistically insignificant amount of irrigant extruded out of the apex in comparison with manual, ultrasonic, and Rinsendo (Dürr Dental, Bietigheim-Bissingen, Germany) groups.[116]

When cleanliness of the root canal walls was analyzed,[366] investigators suggested that both passive sonic or ultrasonic irrigation rendered root canals significantly cleaner than manual preparation in comparison with manual syringe irrigation.[74,465] When comparing sonic with ultrasonic irrigation, the results can be controversial. The majority of the studies favors ultrasonic irrigation.[217,366] The difference lies in the oscillating movements: sonic

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devices range between 1500 and 6000 Hz, and ultrasonic equipment requires vibrations greater than 20,000 Hz.[217,262] If the sonic device is left in the canal for extended periods, cleaning is improved. Sonic or ultrasonic irrigation may be carried out with activated smooth wires or plastic inserts, endodontic instruments, or activated irrigation needles. Examples include EndoSonor (Dentsply Maillefer) and EndoSoft ESI (EMS, Nyon, Switzerland) inserts, IrriSafe (Satelec, Acteon, Merignac, France), the EndoActivator System, and the Vibringe sonic syringe (Cavex, Haarlem, Netherlands). Inadvertent cavitation of root canal walls has not been observed with sonic activation of instruments.[276]

Ultrasonic-assisted irrigation

Ultrasonic devices were first introduced in endodontics by Richman.[356] The files are driven to oscillate at ultrasonic frequencies of 25 to 30 kHz to mechanically prepare the root canal walls.[475] Recently, it has been shown that ultrasonically driven files are useful to activate the irrigation liquids inside the root canal system by inducing acoustic streaming and cavitation.

Two types of ultrasonic irrigation have been described in the literature[10,485]: one in which irrigation is combined with simultaneous ultrasonic instrumentation (UI) and another without simultaneous instrumentation, called *passive ultrasonic instrumentation* (PUI). During UI, the file is intentionally brought into contact with the root canal wall. However, because of the complex canal anatomy, UI never contacts the entire walls, and it may result in the uncontrolled cutting of the root canal walls without effective disinfection.[10] In a study by Macedo[265] instrument oscillation frequency, ultrasonic power, and file taper determined occurrence and extent of cavitation. Some degree of cavitation occurred between the file and canal surface and reached lateral canals and isthmuses.

PUI was first described by Weller et al.[485] The term “passive” is related to the noncutting action of the ultrasonically activated file.[10] PUI relies on the transmission of acoustic energy from an oscillating file or smooth wire to an irrigant in the root canal.

PUI should be introduced in the canal once the root canal system has a final apical size and taper. A fresh solution of irrigant should be introduced, and a small file or smooth wire (e.g., size #15) is ultrasonically activated.

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Since the root canal has already been shaped, the file or wire can move more freely,[467] and the irrigant can penetrate the apical part of the root canal system,[237] with the cleaning effect being more significant.[261] Using this noncutting approach, the potential to create aberrant shapes within the root canal is reduced to a minimum. A file larger than a #15 or #20 requires a wide root canal to reduce oscillation dampening by wall contact.

Ultrasonic activation of the irrigant seems to improve débridement of the root canal system in vitro; the results in vivo present some controversy. Therefore objective guidelines regarding their risks and benefits have not been ascertained.[251] Ultrasonic activation of the irrigant can be intermittent or continue. The ProUltra PiezoFlow (Dentsply Sirona) has been introduced to irrigate and activate the liquids at the same time. The device consists mainly of an ultrasonically energized needle connected to a reservoir of sodium hypochlorite (NaOCl). This continuous ultrasonic irrigation (CUI) system allows simultaneous continuous irrigant delivery and ultrasonic activation; unlike PUI, it does not require the intermittent replenishment of irrigant between ultrasonic file activations. Research shows better elimination of debris and better penetration of irrigant into dentinal tubules.[82,218]

Negative apical pressure irrigation

Another approach to afford better access to irrigation solution to the apical portion of the canal is the so-called negative-pressure irrigation (Fig. 8.60). Here, irrigant is delivered into the access chamber, and a very fine needle connected to the dental unit's suction device is placed into the root canal. Excess irrigant from the access cavity is then transported apically and ultimately removed via suction. First, a macrocannula, equivalent to an ISO size #55,.02 taper instrument, removes coronal debris. Subsequently, a microcannula, equivalent to a size #32,.02 taper, removes particles lodged close to WL. Such a system is commercially available (EndoVac, Discus Dental) and may prove a valuable adjunct in canal disinfection.[302] One of the main characteristics of the system is safety. Several studies demonstrated that EndoVac use is unlikely to extrude irrigation solution through the apex. On the other hand, because the irrigation is deposited in the coronal area, the irrigant flow in the apical last millimeters of the canal is very passive, and some concerns were expressed that flow is laminar and passive in the apical

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region. In a recent study, the apical negative pressure mode of irrigation generated the lowest wall shear stress.[86]

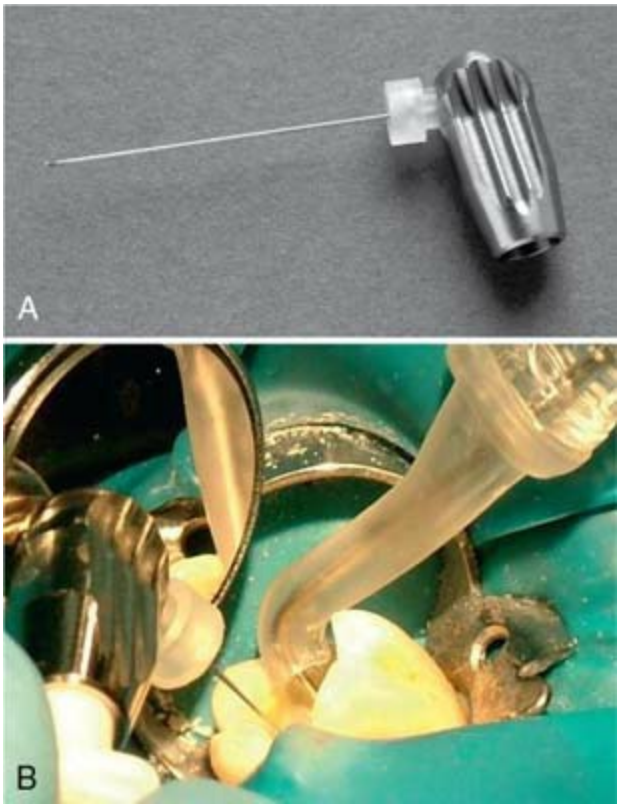

FIG. 8.60 The EndoVac system. **A,** Magnification of the closed-ended microcannula (**A**). **B,** Clinical view of the EndoVac system combined with the Safety-Irrigator.

A) Close-up of a hypodermic needle with a spherically sealed head.

B) Close-up of a rubber dam with clamp exposes a treatable tooth with a working hypodermic needle and a safety irrigator instrument.

Source: (*A,* Courtesy Discus Dental LLC, Culver City, CA. *B,* Courtesy Dr. A. Azarpazhooh.)

Another device that makes use of pressure-suction technology is the RinsEndo system (Dürr Dental, Bietigheim-Bissingen, Germany). It aspirates the delivered rinsing solution into an irrigation needle that is placed close to WL and at the same time activates the needle with oscillations of 1.6 Hz amplitude.[63]

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Safety-Irrigator

The Safety-Irrigator (Vista Dental Products) is an irrigation/evacuation system that apically delivers the irrigant under positive pressure through a thin needle containing a lateral opening and evacuates the solution through a large needle at the root canal orifice (Fig. 8.61). The Safety-Irrigator features a large coronal evacuation tube, enabling the user to irrigate and evacuate simultaneously safely. It fits any standard Luer-lock syringe. Designed to limit the risk of NaOCl accidents, this "negative-pressure" irrigation device comes fully assembled and fitted with side-vented irrigating needle for added safety. This system was tested in vitro to evaluate the removal of dentin debris from artificially made grooves in standardized root canals and showed that there was no significant difference between the manual dynamic activation (MDA) with a nontapered gutta-percha cone, the Safety Irrigator, and the apical negative pressure irrigation. These techniques produced better cleaning efficacy than syringe irrigation (P <.005) but significantly worse than MDA with a tapered cone (P <.05). CUI was significantly better than all the other techniques tested in this study (P <.001).[218]

FIG. 8.61 The Safety-Irrigator.

Close-up of a hypodermic curved needle with a spherically sealed end and twelve microholes radially arranged in the last 0.7 millimeters.

Source: (Courtesy Vista Dental Products, Racine, WI, USA.)

GentleWave system

The GentleWave (GW) system (Sonendo Inc, Laguna Hills, CA) was introduced in the United States in 2016. The device is composed of a console,

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a so-called procedure instrument, which is a single-use tip attached to a handpiece (for either molars or anterior and premolar treatment), and a central unit that contains three individual irrigation solution containers, one waste canister, a degassing system, and a pressure generator (Fig. 8.62). The system delivers an energized flow of irrigation solutions from the central unit to the procedural instrument. According to the manufacturer, the fluid stream entering the tooth creates a shear force, which in turn causes hydrodynamic cavitation. The implosion of micro-bubbles then creates an acoustic field of broadband frequencies, which travels through the fluid into the root canal system. The programmed irrigation regimen begins with 3% NaOCl followed by 8% EDTA, with a rinse of distilled water in between and at completion. The fluid within the root canal space is continuously collected and removed from the chamber through a five-point vented suction system built in the sealing lid of the procedural instrument.[179] A sealed environment is needed between the tooth and the GW Procedure Instrument to allow for constant refreshing of degassed procedure fluids and simultaneous evacuation of debris. The system operates within an air-free, sealed root canal system, filled only with procedure fluid and appears to induce a vertical flow within the root canal system with a slight negative pressure at the apices, which reduces the potential for extrusion. The system applies advanced fluid dynamics, acoustics, and tissue dissolution chemistry to remove tissue and debris from the entire root canal system simultaneously.[179]

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FIG. 8.62 The GentleWave system (Sonendo, Irvine, CA, USA). The system uses multisonic energy to develop a broad section of waves within the irrigation solution to clean inside the roots canal system. It has two main components: a hand piece and a console (shown in figure).

Close-up of a safety irrigator shows a console and a handpiece connected to it.

The studies investigating cleaning and disinfection of the root canal system concluded that GW irrigation results in a faster dissolution rate than other systems[179] and that can clean canals efficiently with minimal instrumentation and can reduce residual debris[287] and promote better penetration into dentinal tubules compared with needle irrigation.[83]

The latter study,[83] using MicroCT, further concluded that GW irrigation was effective in the removal of accumulated dentin from the isthmus of mesial roots in mandibular molars. GentleWave irrigation was superior to needle irrigation but statistically similar to continuous ultrasonically powered irrigation (CI), with debris reductions for canals and isthmuses, respectively, of 96.4% and 97.9% for GW and 91.2% and 93.5% for CI.

As of the writing of this chapter, only three in vivo studies are published,

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all by the same group. The first two reports[405,406] evaluated the 6- and 12-month healing outcome after endodontic treatment using GW. The cumulative success was 97% at 12 months, with 92% and 5% of the necrotic pulp cases classified as healed and healing, respectively. However, in these studies, only 23% of the total sample had preoperative signs of asymptomatic apical periodontitis, which is a well-established outcome predictor.

The subsequent report[404] addressed this concern by assessing the 12-month healing outcome in teeth with a score greater than 3 in the periapical index (PAI). A reduction in PAI scores occurred in 98% of the teeth that received treatment with GW, with 82% and 16% of the teeth classified as healed and healing, respectively. However, despite initial encouraging data, more independent studies on GW are essential to fully describe the clinical applicability of this technique.

Laser-activated irrigation

Lasers are widely used in dentistry and include a diode, Nd:YAG, erbium and CO_2, which produces radiation in both the near and far infrared electromagnet spectrum.[166] Laser devices have been proposed to improve the efficacy of irrigants.[166] Lasers have been studied for their ability to clean and effectively disinfect root canals. The Er:YAG laser wavelength (2940 nm) has the highest absorption in water and high affinity to hydroxyapatite, which makes it suitable for use in root canal treatment.[96]

Laser energy may be used to activate irrigant solutions in different ways, for example at a molecular level, as in photo-activated disinfection (PAD) or at a bulk flow level as in laser-activated irrigation (LAI). Several studies in vivo and ex vivo indicated that laser activated irrigation is promising in removing smear layer[150] and dentin debris[108] in less time than PUI. The mechanism of action[51] is based on the generation of a secondary cavitation effect with expansion and the successive implosion of fluids.[51]

These results are in agreement with data related to a new erbium laser technique that used photon-induced photoacoustic streaming (PIPS) of irrigants. In that technique, the laser tip is placed into the coronal access opening of the pulp chamber only and is kept stationary without advancing into the orifice of the canal.[118] The use of a tapered and stripped tip with specific minimally ablative laser settings is required: low energy (20 mJ),

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pulse repetition rate (15 Hz), and short pulse duration (50 μs). The difference in laser penetration and bacterial killing is attributed to the difference in the degree of absorption of different wavelengths of light within the dentin. Bergmans et al.[45] concluded in their in vivo study that the Nd:YAG laser irradiation is not an alternative, but a possible supplement to existing protocols for canal disinfection as the properties of laser light may allow a bactericidal effect beyond 1 mm of dentin. Endodontic pathogens that grow as biofilms, however, are challenging to eradicate even upon direct laser exposure.[58]

Photoactivated disinfection

Photodynamic therapy (PDT) or light-activated therapy (LAT) may have endodontic applications because of its antimicrobial effectiveness.[185] In principle, antimicrobial photodynamic therapy (APDT) is a two-step procedure that involves the introduction of a photosensitizer (step 1: photosensitization of the infected tissue) followed by light illumination (step 2: irradiation of the photosensitized tissue) of the sensitized tissue, which would generate a toxic photochemistry on the target cell, leading to cell lysis. Each of these elements used independently will not have any action, but together they have a synergism effect of producing antibacterial action. Indeed, in vitro experiments showed promising results as an adjunct disinfected device. Shrestha and Kishen[400] concluded that the tissue inhibitors existing within the root canal affected the antibacterial activity of PDT at varying degrees and further research is required to enhance their antimicrobial efficacy in an endodontic environment. Photosensitizers combined with nanoparticles such as Rose Bengal may have a promising antibacterial efficacy.[401]

Antibacterial nanoparticles

The concept of nanotechnology was first discussed in 1959 by Richard Feynman; the topic has recently been reviewed by Shrestha and Kishen.[402] Nanotechnology is the science conducted at the nanoscale, using particles about 1 to 100 nm, and it can be used in chemistry, biology, physics, materials science, and engineering. The particles can be classified depending on their shape as atom clusters, grains, fibers, films, nanoholes, and

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composites from these combinations. Nanomaterial properties vary majorly from other materials due to the increase in surface area per unit mass.

In dentistry, nanotechnology is currently investigated in the following fields: local nanoanaesthesia, hypersensitivity cure, tooth repositioning, nanorobotic dentifrice, dental durability, cosmetics, nanodiagnostics, and gene therapy.[49] More specifically, the research on nanomaterials is focusing on nanocomposites, nanosolution, nanoadhesives, and impression materials.

Nanoparticles in endodontics are being developed for their efficient disinfection of root canals, due to the broad-spectrum antibacterial activity. The nanoparticles evaluated in endodontics include Chitosan (CS-np), zinc oxide (ZnO-np), and silver (Ag-np) nanoparticles.[273]

CS-np have been widely studied and appear to be effective against monospecies *(E. faecalis)* and multispecies biofilms, even in the presence of tissue inhibitors, and it has been attributed to their ability to disrupt the cell wall.[402] Silver nanoparticles are also being evaluated for use as root canal disinfecting agents. In gel and liquid form, such nanoparticles have been shown to be able to kill and disrupt *E. faecalis* biofilm.

Biactive glass (SiO_2-Na_2O-CaO-P_2O_5) is another revolutionary introduction in the field of nanotechnology, and it has been suggested for root canal disinfection. The antimicrobial effect of bioactive glass is due to its ability to maintain an alkaline environment over a period of time.[273] When used in root canals, bioactive glass was found to kill bacteria, but the mechanism of action was not pH related and dentin did not seem to alter its effect.[507]

It is recently being researched for its use for endodontic regeneration in which diseased or necrotic pulp tissues are removed and replaced with healthy pulp tissues to revitalize teeth. It was reported the first use of nanostructured and functionalized multilayered films was as a new active biomaterial for endodontic regeneration.

HealOzone.

Ozone, in the gaseous or aqueous phase, is a powerful and reliable antimicrobial agent against bacteria, fungi, protozoa, and viruses. The mechanism of action is based on the destruction of cell walls and cytoplasmic membranes of bacteria and fungi by the oxidant potential of the ozone.

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During this process, ozone attacks glycoproteins, glycolipids, and other amino acids and inhibits the enzymatic control system of the cell with an increase of the membrane permeability leading to immediate functional cessation. The ozone molecules then readily enter the cell and effectively kills microorganisms.[272]

It is applied to oral tissues in the following forms: ozonated water, ozonated olive oil, and oxygen/ozone gas. In vitro studies showed promising results on their antibacterial effect, clinical results demonstrated the opposite.[126,193,294,295] Ozone gas is currently used clinically for endodontic treatment; however, the results from in vivo studies are inconclusive.[193,294,295] This inconsistency is attributed to the lack of information about the maximum duration of application and concentration that should be used.[27,226] The hazards of ozone, when used in endodontics, have not been investigated. Care should be taken concerning the patient's and the dentist's exposure to the gas.

Water preparations.

Super-oxidized water,[171] also called electrochemically activated water[270,422] or potential oxidative water,[189,394] is effectively saline that has been electrolyzed to form superoxidized water, hypochlorous acid, and free chlorine radicals. It is commercially available as Sterilox (Sterilox Technologies, Radnor, PA, USA). This solution is nontoxic to biologic tissues, yet able to kill microorganisms. The solution is generated by electrolyzing saline solution—a process no different from that used in the commercial production of NaOCl.[136] The difference, however, is that the solution accumulating at the anode is harvested as the anolyte and at the cathode as the catholyte. These solutions display properties that are dependent upon the strength of the first saline solution, the applied potential difference, and the rate of generation. The technology that allows harvesting of the respective solutions resides in the design of the anode and the cathode and originates either in Russia (electrochemically activated water) or Japan (potential oxidative water).[269] Although the solutions bear different names, the principles in the manufacturing process appear to be similar.

The use of superoxidized water is sparsely described in the endodontic literature but shows early promise. The solutions from both technologies have

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been tested for their ability to débride root canals,[189] remove the smear layer,[394,422] and kill bacteria[200] and bacterial spores.[259] Results are generally favorable, while also showing biocompatibility with vital systems.[211]

Anolyte and catholyte solutions generated from one such technology (Radical Waters Halfway Johannesburg, South Africa) have shown promise as antibacterial agents against laboratory-grown, single-species biofilm models.[151] Such solutions have been recommended as suitable for removing biofilms in dental unit water lines[269] and have even been marketed for this purpose. Cautious clinicians may prefer to wait for more studies to demonstrate safety and efficacy under normal clinical setting conditions before adopting newer, less tested irrigating solutions.

Criteria to evaluate cleaning and shaping[4]

Well-shaped canals

The main aims of canal shaping are to directly remove tissues and microbial irritants and to provide sufficient geometric space for subsequent obturation (see Table 8.2). In order to achieve these goals, the prepared canal should include the original canal (see *red areas* in Fig. 8.4); there should be an apical narrowing and the canal should be tapered. These concepts have been popularized by Schilder[386] and are still maintained today.[3,124]

Therefore, a well-shaped canal is defined more specifically by the absence of procedural errors (see later) and the achievement of disinfection; more recently, another element was added to this equation—the retention of as much tooth structure as feasible.[154]

The fact that a canal is adequately shaped can often be determined by a clinician from radiographs and clinical experience—for example, when fitting a cone. At this time, the feel would be of tug-back, a slight resistance to pull; a radiograph should demonstrate a symmetrical canal shape lateral of the cone, the presence of an intact apical narrowing, and no thinned out radicular wall sections.

Using magnification, clinicians should inspect the canal orifice and the coronal third of each shaped canal for clean canal walls.[319] Immediately after irrigation with sodium hypochlorite, an absence of visible turbidity and

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effervescence should be noted. If present, these phenomena along with visible deposits on the canal walls are indicative of organic matter still in suspension or adherent to the radicular walls.

Signs of mishaps

Instrument fracture

Most reports suggest that manual endodontic file fracture or rotary instrument fracture occur at a rate of approximately 1% to 6% and 0.4% to 5%, respectively.[434,463] Such fractures are untoward events and perceived as such by clinicians.[50] Evidently retained instrument fragments limit access of disinfecting irrigants to the root canal system, possibly impeding sufficient elimination of microorganisms.[178] However, the current clinical evidence does not suggest that the presence of a retained instrument must result in a significant higher rate of failing root canal treatments when done by specialists.[434]

In general, instruments used in rotary motion break in two distinct modes —torsional and flexural.[329,375,462] Torsional fracture occurs when an instrument tip is locked in a canal while the shank continues to rotate, thereby exerting enough torque to fracture the tip. This also may occur when instrument rotation is sufficiently slowed in relation to the cross-sectional diameter. In contrast, flexural fracture occurs when the cyclic loading leads to metal fatigue. This problem precludes the manufacture of continuously rotating stainless steel endodontic instruments, because steel develops fatal fatigue after only a few cycles.[393] NiTi instruments can withstand several hundred flexural cycles before they fracture,[250,348,462] but they still can fracture in the endodontic setting after a low (i.e., below 10,000) number of cycles.[87]

Repeated loading and cyclic fatigue tests for endodontic instruments are not described in pertinent norms. Initially, rotary instruments such as GG burs and Peeso reamers were tested with a superimposed bending deflection.[62] In another study, stainless steel and NiTi hand files were rotated to failure in steel tubes with an acute 90-degree bend and an unspecified radius.[393] Under these conditions, size #40 stainless steel instruments fractured after fewer than 20 rotations, whereas various NiTi files of the same

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size withstood up to 450 rotations.

Cyclic fatigue was also evaluated for ProFile.06 taper instruments using a similar device.[498] The number of rotations to failure for unused control instruments ranged from 1260 (size #15 files) to 900 (size #40 files). These scores did not change when the instruments were tested under simulated clinical conditions such as repeated sterilization and contact with 2.5% NaOCl. Subsequently, control instruments were compared with a group of instruments used in the clinical setting in five molar cases;[497] again, no significant differences were found in resistance to cyclic fatigue.

One study[183] used a different testing method involving tempered metal cylinders with radii of 5 and 10 mm that produced a 90-degree curve. They reported fatigue fractures for size #15,.04 taper ProFile instruments after about 2800 cycles with the 10 mm cylinders. In size #40,.04 taper ProFile instruments, fractures occurred after about 500 cycles with the 5-mm cylinders. In comparison, size #15,.06 taper ProFile instruments also failed after about 2800 revolutions with the 10-mm cylinders, but failure occurred in size #40,.06 taper ProFile specimens after only 223 cycles with the 5-mm cylinders.

Rotary NiTi instruments with larger tapers and sizes consistently fractured after fewer rotations,[345] and although the radius of the curves was halved, fatigue-life was reduced by 400%. Another investigation[183] reported similar results for selected HERO instruments, and their findings were confirmed by other tests on GT rotary instruments. Size #20,.06 taper GT files failed after 530 rotations in a 90-degree curve with a 5-mm radius; size #20,.12 taper GT files failed after 56 rotations under the same conditions.[334]

Reuse of rotary instruments depends on safety, specifically on assessment of fatigue and also the potential to properly clean NiTi surfaces.[34,304,311,398,424,428,458] Specific instruments perform differently in this regard, since fatigue depends more on the amount of metal in cross section at the point of stress concentration[163,457] than on the specifics of instrument design.[88]

On the other hand, manufacturers constantly claim that their instrument has been equipped with design elements that render it more fatigue resistant. For example, LightSpeed LSX is manufactured without a milling process. However, no data have been published regarding its fatigue resistance. GTX

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is manufactured from a novel NiTi alloy, M-Wire, to increase its fatigue resistance.[220] However, investigators[234] could not confirm these findings. Similarly, another study[243] did not find the TF, which is not milled and hence believed to be fatigue resistant,[143] to perform better than conventionally manufactured ProFile rotaries. Another feature, electropolishing (as discussed earlier), does not appear to confer a significantly increased fatigue resistance to EndoSequence[243,353] and RaCe.[457,458,495] One possible reason for these variable outcomes are the different testing environments used in vitro[90]; clinically, even greater variability is to be expected.

Recently, cyclic fatigue tests are sometimes conducted under more directly clinically applicable conditions, such as varying ambient temperature or introducing dynamic movement. For example, while the latest martensitic alloys we showed to have significantly increased fatigue resistance at room temperature, this benefit was greatly reduced at body temperature.[22,109]

Attempts have been made to use tests according to norms and specifications described for stainless steel hand instruments such as K-files and Hedström files,[2] since no comparable norms exist for instruments used in continuous rotary motion. Consequently, a number of models have been devised to assess specific properties of NiTi rotary instruments, including torque at failure, resistance against cyclic fatigue, and others (Fig. 8.63). These systems may simultaneously assess torque at failure, working torque axial force, and cyclic fatigue.

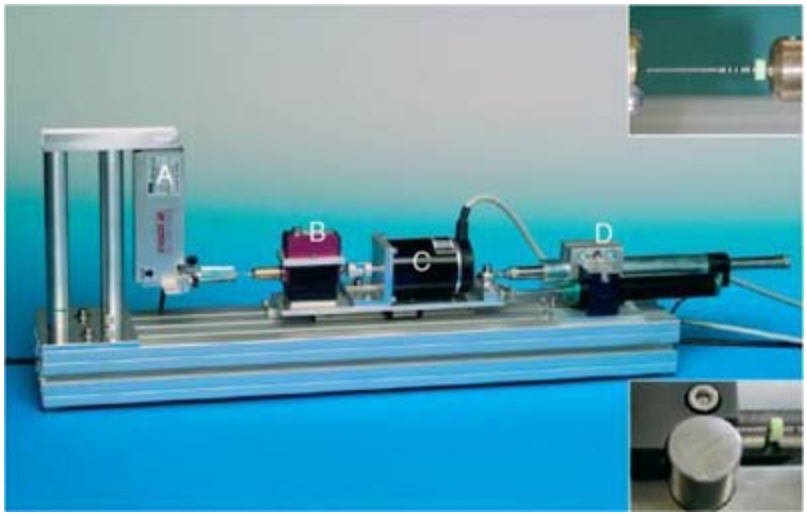

FIG. 8.63 Testing platform for analysis of various factors during

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simulated canal preparation with rotary endodontic instruments. Labeled components are a force transducer *(A)*, a torque sensor *(B)*, a direct-drive motor *(C)*, and an automated feed device *(D)*. For specific tests, a cyclic fatigue phantom or a brass mount compliant with ISO No. 3630-1 *(inserts)* may be attached.

Four different nickel titanium rotary instruments kept on a platform are labeled from A through D (left to right).

According to the norms mentioned previously, torque at failure is recorded with the apical 3 mm of the instrument firmly held in the testing device while the instrument's handle is rotated. A wide variety of rotary NiTi endodontic instruments have been tested in this way. For example, ProFile NiTi rotary files in ISO sizes #25, #30, and #35 (.04 taper) fractured at 0.78, 1.06, and 1.47 Ncm, respectively.[441]

When analyzing clinical factors involved in instrument fracture, one must consider both torsional load and cyclic fatigue.[375] However, these are not separate entities, especially in curved canals.[57] Working an instrument with high torque may lower resistance to cyclic fatigue.[141] Conversely, cyclic prestressing has been shown to reduce the torsional resistance of ProTaper finishing files,[462] as well as K3[30] and MTwo.[345] Also, cyclic fatigue occurs not only in the lateral aspect when an instrument rotates in a curved canal but also axially when an instrument is bound and released by canal irregularities.[48]

The torque generated during canal preparation depends on a variety of factors, and an important one is the contact area.[52] The size of the surface area contacted by an endodontic instrument is influenced by the instrumentation sequence or by the use of instruments with different tapers.[388] A crown-down approach is recommended to reduce torsional loads (and thus the risk of fracture) by preventing a large portion of the tapered rotating instrument from engaging root dentin (known as *taper lock*).[52,499]

The clinician can further modify torque by varying axial pressure, because these two factors are related (see Fig. 8.20).[388] In fact, a fairly light touch is recommended for all current NiTi instruments to avoid forcing the instrument into taper lock. The same effect might occur in certain anatomic situations, such as when canals merge, dilacerate, and divide.

The torsional behavior of NiTi rotary endodontic instruments cannot be

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described properly without advanced measurement systems and a new set of norms. However, the clinician must be able to interpret correctly the stress-strain curves for all rotary NiTi instruments used in the clinical setting to be able to choose an appropriate working torque and axial force.

Therefore a careful evaluation should be performed before the attempt is made to remove any retained fragment (see Fig. 8.25). In fact, Ward and others[481] suggest any attempt of fragment removal only when the fragment is located coronal of a significant root canal curve and thus visible with the aid of magnification.

There are sophisticated means and strategies to remove retained fragments, which are described in detail in Chapter 20 of this book.

Of note, assessment of physical parameters governing rotary root canal preparation was considered crucial because NiTi rotary in vitro had increased risk of fracture compared with K-files. Some clinicians also describe instrument fracture as a main issue for concern.[50]

In a study using plastic blocks, as many as 52 ProFile Series 29 instruments became permanently deformed.[450] Three fractures were reported in a subsequent study on ISO-norm ProFile size.04 instruments, and three other instruments were distorted.[66] An even higher fracture incidence was shown in a study on rotary instruments used in plastic blocks in a specially designed testing machine.[446] These findings were supported by two studies, in which high fracture incidences were reported for LightSpeed and Quantec rotary instruments used in a clinical setting.[32,375]

On the other hand, as stated earlier, a retrospective clinical study suggests similar outcomes with and without retained instrument fragments[434]; moreover, others' experience suggests that the number of rotary instrument fractures is lower than previously estimated.[112,489] Removal of such fragments is possible in many situations, but there is also the potential for further damage (e.g., perforation) rather than successful removal.[440,489] Consequently, a benefit-versus-risk analysis should be carried out prior to attempts to remove NiTi instrument fragments, addressing the reasons and the clinical consequences of instrument fracture.

Canal transportation

Perhaps the most frequent adverse outcomes during canal shaping are

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aberrations from the original canal path. Much has been written on the appearance of such aberrations using labels such as zip and elbow formation, ledging, perforation, stripping, and others.[484]

Canal transportation is at the root of all these clinical problems and may be defined as "the removal of canal wall structure on the outside curve in the apical half of the canal due to the tendency of files to restore themselves to their original linear shape during canal preparation."[3]

As files tend to straighten in the canal, this typically occurs toward the inner (or convex) radicular wall at midroot as well as to the outer curvature apically. Such a shift of the canal axis during shaping results in excessive loss of dentin and may ultimately result in strip perforation, while apical transportation may lead to zipping or overt apical perforation (see Fig. 8.39).

Conceptually, any canal preparation will shift the canal axis, which is often determined as the center of gravity in cross sections. It has been held that a transportation of about 100 to 150 μm may be clinically acceptable.[207,326]

If canal transportation has led to ledge formation, subsequent instruments will bypass the area of the ledge only when adequately precurved. In case of rotary instrumentation, hand-operated instruments of comparable size are then recommended (Fig. 8.64).

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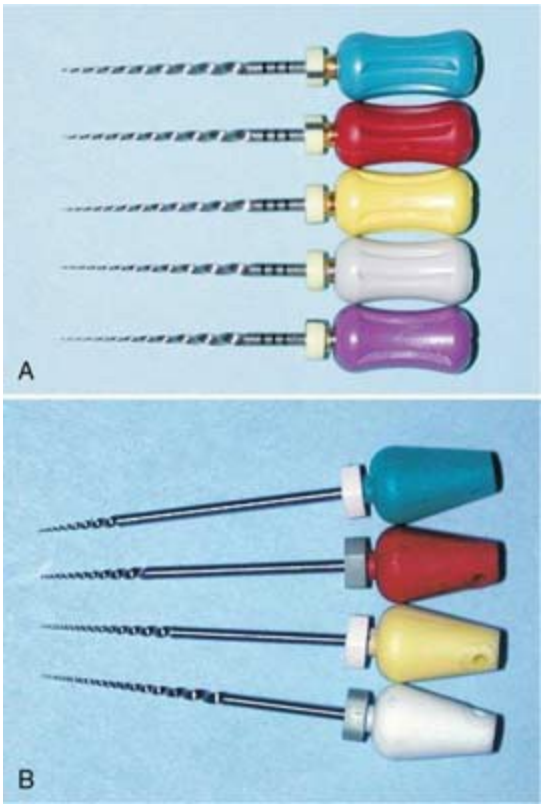

FIG. 8.64 Instruments with increased taper that can be used by hand. **A,** ProTaper instruments with special handles attached to rotary instrument shanks. **B,** GT hand instruments.

A) Close-up of five ProTaper instruments with spirally textured bodies and pointed tips.

B) Close-up of four GT hand instruments with different depths of spirally textured surface.

Perforation

As indicated, perforation may be the ultimate result of canal transportation when it occurs within the root canal system. Other perforations are those in the access cavity; a discussion of access cavity preparation may be found in Chapter 7. Obviously, preparing mineralized canals requires advanced operator skills and is facilitated by magnification (Fig. 8.65).

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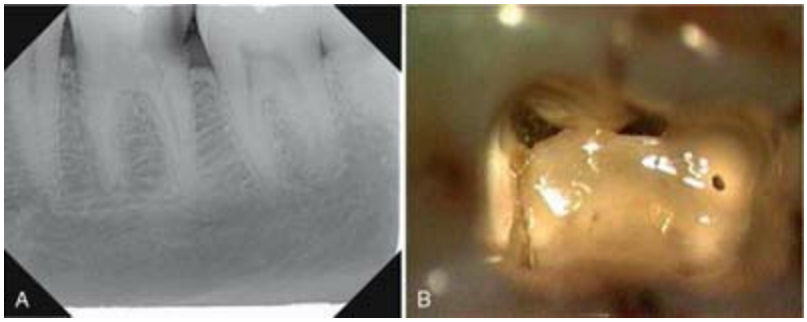

FIG. 8.65 Evidence of coronal hard-tissue deposition. **A,** Periapical radiograph of tooth #19 shows evidence of reduced coronal and radicular pulp space. **B,** Intraoral photograph, taken through an operating microscope (×25), of access cavity of the tooth shown in **A**; note the calcific metamorphosis.

A) Radiograph of a tooth shows radiopaque crown, neck and lower canals with black upper canals.

B) Close-up of an open crown exposes two black cavities in the dentin in presence of light.

Three types of perforations can be defined—strip perforations that occur toward the furcation in multirooted teeth (also known as “danger zones”[7]), perforations associated with canal curvatures, and perforations though the apical foramen.

Blockage

A canal may become impassable during the process of cleaning and shaping due to two distinct, but often connected, occurrences. A ledge is a dentinal shelf that is created by shaping instruments that straighten and dig into the convex side of the canal wall. In less severe cases, ledges can be corrected and smoothed out with precurved instruments. This condition may lead to false paths and impede optimal obturation, when WL cannot be reached with master cones.

A blockage refers mainly to a root canal area that is filled with densely compacted debris or collagenous pulp remnants (see Fig. 8.38). It may also be caused by other obstacles such as fractured files or remnants of a preexisting coronal or radicular filling materials. Clinically the presence of ledge or block is signaled by the inability for a straight flexible instrument to penetrate deeper into the root canal; however, this needs to be differentiated from a small or mineralized canal, which causes longer portions of the

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instrument's cutting flutes to bind.

Obviously, such a blockage prevents the apical canal portion from being disinfected. For details of strategies to deal with ledge and blockage, refer to Chapter 10.

Another reason for a perceived blockage may be a very abrupt canal curvature.

Sample protocol for contemporary cleaning and shaping procedures

- Using well-angulated preoperative radiographs, analyze case difficulty using well-established parameters.
- Place rubber dam and estimate WL.
- Prepare a conservative access cavity sufficient enough to reveal all root canal orifices.
- Scout canals with a #10 K-file in the presence of a lubricant.
- If the selected series of rotary instruments advances easily to the estimated WL, confirm patency and determine WL using an electronic apex locator.
- If the instruments meet resistance and the file does not progress gently to WL, use a dedicated NiTi instrument. It is prudent to modify the orifice to create a coronal receptacle for the subsequent rotaries. Negotiate, confirm patency, and determine WL.
- Create a reproducible glide path to WL with appropriate instruments.
- Irrigate with sodium hypochlorite throughout the shaping procedure.
- Advance the selected series of rotary instruments (based on canal anatomy) passively in the presence of sodium hypochlorite to shape the middle third. When shaping canals that have a larger buccal-lingual dimension, consider shaping as two canals.
- Clean cutting flutes routinely upon removal and remove debris with an alcohol-moistened gauze. If the selected rotary does not progress easily, remove irrigate, recapitulate with a #10 K-file, and choose a different, often smaller, instrument.
- Use copious irrigation, reverify canal patency and working length

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throughout and upon completion of shaping. Gauge the size of the foramen with an appropriate hand file.

- Protocol for irrigation:
  - Irrigate using copious amounts of sodium hypochlorite.
  - Activate the irrigant.
  - Select irrigation solution for smear layer management.
  - Perform final irrigation.
- Dry the canal thoroughly and obdurate with a technique that promotes a three-dimensional fill.
- Restore the endodontically treated tooth in a timely manner.

Summary

Cleaning and shaping are important, interdependent steps in root canal treatment. Cleaning, as demonstrated by an intracanal surface free of smear layer, can be done only after root canals have been sufficiently enlarged to accommodate adequate irrigation needles. Canal preparation is optimized when mechanical aims are fulfilled and enlargement is acceptable; such aims include avoiding both significant preparation errors and weakening of the radicular structure, which can result in fractures.

Taken together and performed to a high standard, the procedures described in this chapter lay the foundation for biologic success in both straightforward (Fig. 8.66) and more complicated (Fig. 8.67) clinical cases. Recall radiographs confirm favorable outcomes or biologic success (i.e., prevention or healing of periradicular periodontitis) over the years. Similarly, adherence to the principles discussed leads to predictable outcomes for root canal treatments.

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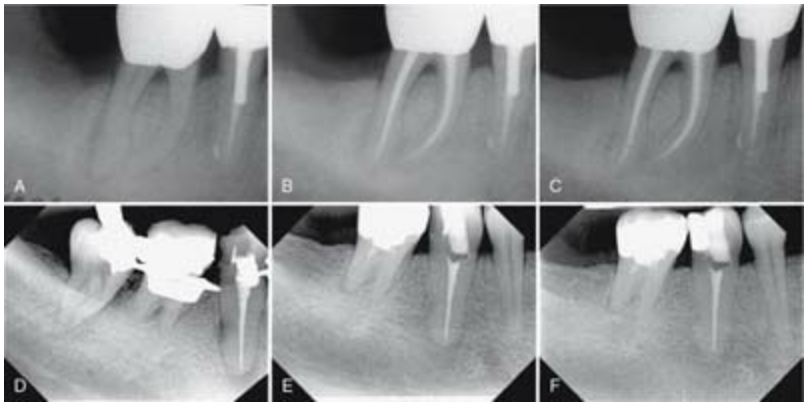

FIG. 8.66 Clinical cases treated according to the principles detailed in this chapter. **A,** Pretreatment radiograph of tooth #30 with a periradicular lesion. **B,** Postobturation radiograph. **C,** Two-year follow-up radiograph shows osseous healing. **D,** Immediate postobturation radiograph of tooth #29 shows both a periapical and a lateral osseous lesion. **E–F,** One-year and 3-year follow-up radiographs show progressing osseous healing. Note the imperfect obturation of tooth #30.

A) Radiograph of a tooth shows radiopaque irregular crown and an outline of radiopaque post.

B) Radiograph of a tooth shows radiopaque regular crown and an outline of radiopaque post.

C) Radiograph of a tooth shows radiopaque crown and an outline of radiopaque post.

D) Radiograph of a tooth shows radiopaque patch on the neck with radiopaque canal.

E) Radiograph of a tooth shows radiopaque crown and neck with radiolucent segmented canal.

F) Radiograph of a tooth shows radiopaque part of the crown with radiolucent neck and segmented canal.

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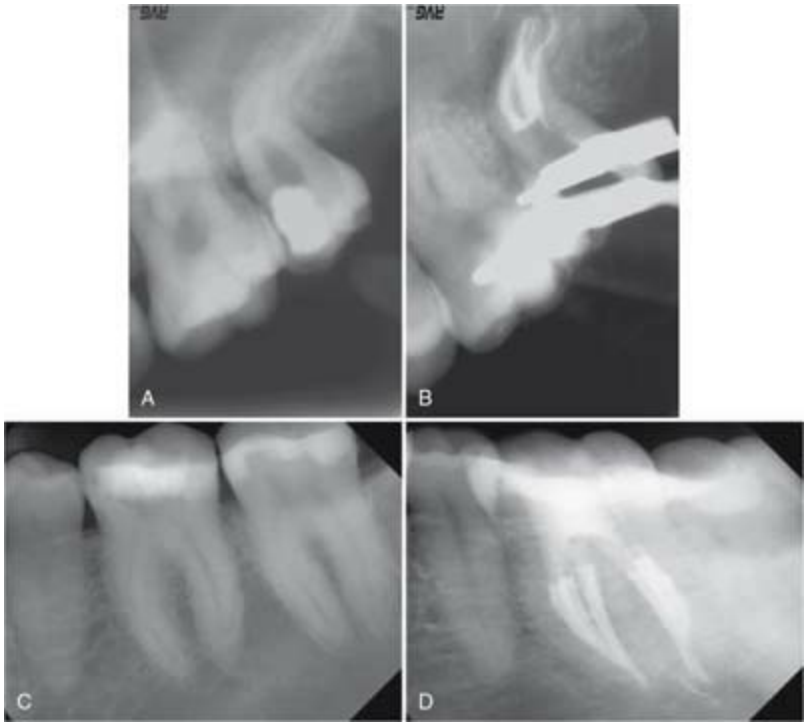

FIG. 8.67 Complicated clinical cases treated with hybrid techniques. **A,** Pretreatment radiograph of tooth #16 indicates laceration and significant curvature of all roots. **B,** Posttreatment radiograph shows multiple planes of curvature. **C,** Pretreatment radiograph of tooth #19, which was diagnosed with irreversible pulpitis. **D,** Angulated posttreatment radiograph shows three canals in the mesiobuccal root canal system, all of which were prepared to apical size #50.

A) Radiograph of a tooth shows radiopaque part of the crown.

B) Radiograph of a tooth shows radiopaque lower canals with a radiopaque outline of dental instruments.

C) Radiograph of a tooth shows radiopaque outline of white on the crown.

D) Radiograph of a tooth shows radiopaque patch on the neck with radiopaque canal. An outline of the clamp is visible.

E) Radiograph of a tooth shows radiopaque crown and lower canals with radiolucent upper canals.

Source: (*A–B*, Courtesy T. Clauder; ***C–D***, Courtesy Dr. H. Walsch.)

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References

1. Collins English dictionary ed 11 2003; HarperCollins Publishers England.
2. Dental root-canal instruments - part 1: Files, reamers, barbed broaches, rasps, paste carriers, explorers and cotton broaches 1992; International Organization for Standardization Geneva.
3. Glossary of endodontic terms 2015; American Association of Endodontists.
4. Treatment Standards 2018; American Association of Endodontists Chicago, IL.
5. Abbott PV, Hume WR, Heithersay GS. *Barriers to diffusion of Ledermix paste in radicular dentine*, Endod Dent Traumatol 2, 1989;5: 98.
6. Abbott PV, Hume WR, Heithersay GS. *Effects of combining Ledermix and calcium hydroxide pastes on the diffusion of corticosteroid and tetracycline through human tooth roots in vitro*, Endod Dent Traumatol 4, 1989;5: 188.
7. Abou-Rass M, Frank AL, Glick DH. *The anticurvature filing method to prepare the curved root canal*, J Am Dent Assoc 1980;101: 5.
8. Abou-Rass M, Jann JM, Jobe D, et al: *Preparation of space for posting: effect on thickness of canal walls and incidence of perforation in molars*, J Am Dent Assoc 1982;104: 634.
9. Ahmad M. *An analysis of breakage of ultrasonic files during root canal instrumentation*, Endod Dent Traumatol 2, 1989;5: 78.
10. Ahmad M, Pitt Ford TJ, Crum LA. *Ultrasonic debridement of root canals: acoustic streaming and its*

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www.konkur.in

possible role, J Endod 10, 1987;13: 490.

11. Ajuz NC, Armada L, Goncalves LS, et al: *Glide path preparation in S-shaped canals with rotary pathfinding nickel-titanium instruments*, J Endod 2013;39: 4.
12. Alapati SB, Brantley WA, Nusstein JM, et al: *Vickers hardness investigation of work-hardening in used NiTi rotary instruments*, J Endod 12, 2006;32: 1191.
13. Alapati SB, Brantley WA, Svec TA, et al: *Scanning electron microscope observations of new and used nickel-titanium rotary files*, J Endod 10, 2003;29: 667.
14. Albrecht LJ, Baumgartner JC, Marshall JG. *Evaluation of apical debris removal using various sizes and tapers of ProFile GT files*, J Endod 6, 2004;30: 425.
15. Allison DA, Weber CR, Walton RE. *The influence of the method of canal preparation on the quality of apical and coronal obturation*, J Endod 10, 1979;5: 298.
16. Alodeh MH, Doller R, Dummer PM. *Shaping of simulated root canals in resin blocks using the step-back technique with K-files manipulated in a simple in/out filling motion*, Int Endod J 3, 1989;22: 107.
17. Andersen M, Lund A, Andreasen JO, et al: *In vitro solubility of human pulp tissue in calcium hydroxide and sodium hypochlorite*, Endod Dent Traumatol 3, 1992;8: 104.
18. Anderson DN, Joyce AP, Roberts S, et al: *A comparative photoelastic stress analysis of internal root stresses between RC Prep and saline when applied to the Profile/GT rotary instrumentation system*, J Endod 3, 2006;32: 222.
19. Anderson MA, Price JW, Parashos P. *Fracture resistance of electropolished rotary nickel-titanium endodontic instruments*, J Endod 10, 2007;33: 1212.

Telegram: @dental_k

www.konkur.in

20. Aquilino SA, Caplan DJ. *Relationship between crown placement and the survival of endodontically treated teeth*, J Prosthet Dent 3, 2002;87: 256.
21. Arias A, Azabal M, Hidalgo JJ, et al: *Relationship between postendodontic pain, tooth diagnostic factors, and apical patency*, J Endod 2, 2009;35: 189.
22. Arias A, Hejlawy S, Murphy S, et al: *Variable impact by ambient temperature on fatigue resistance of heat-treated nickel titanium instruments*, Clin Oral Investig 2019;23: 3.
23. Arias A, Singh R, Peters OA. *Torque and force induced by ProTaper universal and ProTaper next during shaping of large and small root canals in extracted teeth*, J Endod 2014.
24. Asboe-Jorgensen V, Attstrom R, Lang NP, et al: *Effect of a chlorhexidine dressing on the healing after periodontal surgery*, J Periodontol 1, 1974;45: 13.
25. Attin T, Buchalla W, Zirkel C, et al: *Clinical evaluation of the cleansing properties of the noninstrumental technique for cleaning root canals*, Int Endod J 11, 2002;35: 929.
26. Aubut V, Pommel L, Verhille B, et al: *Biological properties of a neutralized 2.5% sodium hypochlorite solution*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2, 2010;109: e120.
27. Azarpazhooh A, Limeback H. *The application of ozone in dentistry: a systematic review of literature*, J Dent 2, 2008;36: 104.
28. Azim AA, Piasecki L, da Silva Neto UX, et al: *XP shaper, a novel adaptive core rotary instrument: micro-computed tomographic analysis of its shaping abilities*, J Endod 9, 2017;43:.
29. Backman CA, Oswald RJ, Pitts DL. *A radiographic comparison of two root canal instrumentation techniques*,

Telegram: @dental_k

www.konkur.in

J Endod 1, 1992;18: 16.
30. Bahia MG, Melo MC, Buono VT. *Influence of cyclic torsional loading on the fatigue resistance of K3 instruments*, Int Endod J 2008;41: 10.
31. Baker NE, Liewehr FR, Buxton TB, et al: *Antibacterial efficacy of calcium hydroxide, iodine potassium iodide, betadine, and betadine scrub with and without surfactant against E faecalis in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 3, 2004;98: 359.
32. Barbakow F, Lutz F. *The 'Lightspeed' preparation technique evaluated by Swiss clinicians after attending continuing education courses*, Int Endod J 1, 1997;30: 46.
33. Barbosa CAM, Goncalves RB, Siqueira JF, et al: *Evaluation of the antibacterial activities of calcium hydroxide, chlorhexidine, and camphorated paramonochlorophenol as intracanal medicament. A clinical and laboratory study*, J Endod 1997;23: 5.
34. Barbosa FO, Gomes JA, de Araujo MC. *Influence of sodium hypochlorite on mechanical properties of K3 nickel-titanium rotary instruments*, J Endod 2007;33: 8.
35. Bardsley S, Peters CI, Peters OA. *The effect of three rotational speed settings on torque and apical force with vortex rotary instruments in vitro*, J Endod 6, 2011;37: 860.
36. Barthel CR, Zimmer S, West G, et al: *Bacterial leakage in obturated root canals following the use of different intracanal medicaments*, Endod Dent Traumatol 2000;16:.
37. Basmadjian-Charles CL, Farge P, Bourgeois DM, et al: *Factors influencing the long-term results of endodontic treatment: a review of the literature*, Int Dent J 2002;52: 2.
38. Basrani B, Haapasalo M. *Update on endodontic irrigating solutions*, Endod Topics 2012;27: 1.

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39. Basrani B, Lemonie C. *Chlorhexidine gluconate*, Aust Endod J 2005;31: 2.
40. Basrani B, Santos JM, Tjäderhane L, et al: *Substantive antimicrobial activity in chlorhexidine-treated human root dentin*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 2.
41. Basrani B, Tjäderhane L, Santos JM, et al: *Efficacy of chlorhexidine- and calcium hydroxide-containing medicaments against enterococcus faecalis in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 5.
42. Basrani BR, Manek S, Sodhi RN, et al: *Interaction between sodium hypochlorite and chlorhexidine gluconate*, J Endod 2007;33: 8.
43. Baugh D, Wallace J. *The role of apical instrumentation in root canal treatment: a review of the literature*, J Endod 2005;31: 5.
44. Beltz RE, Torabinejad M, Pouresmail M. *Quantitative analysis of the solubilizing action of MTAD, sodium hypochlorite, and EDTA on bovine pulp and dentin*, J Endod 2003;29: 5.
45. Bergmans L, Moisaidis P, Teughels W, et al: *Bactericidal effect of Nd:YAG laser irradiation on some endodontic pathogens ex vivo*, Int Endod J 2006;39:.
46. Berutti E, Angelini E, Rigolone M, et al: *Influence of sodium hypochlorite on fracture properties and corrosion of ProTaper rotary instruments*, Int Endod J 2006;39: 9.
47. Berutti E, Cantatore G, Castellucci A, et al: *Use of nickel-titanium rotary PathFile to create the glide path: comparison with manual preflaring in simulated root canals*, J Endod 2009;35: 3.
48. Best S, Watson P, Pilliar R, et al: *Torsional fatigue and endurance limit of a size 30.06 ProFile rotary instrument*,

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www.konkur.in

Int Endod J 2004;37: 6.
49. Bhardwaj A, Bhardwaj A, Misuriya A, et al: *Nanotechnology in dentistry: present and future*, J Int Oral Health 2014;6: 1.
50. Bird DC, Chambers D, Peters OA. *Usage parameters of nickel-titanium rotary instruments: a survey of endodontists in the United States*, J Endod 9, 2009;35: 1193.
51. Blanken J, De Moor RJ, Meire M, et al: *Laser induced explosive vapor and cavitation resulting in effective irrigation of the root canal. Part 1: a visualization study*, Lasers Surg Med 2009;41: 7.
52. Blum JY, Cohen A, Machtou P, et al: *Analysis of forces developed during mechanical preparation of extracted teeth using profile NiTi rotary instruments*, Int Endod J 1999;32: 1.
53. Blum JY, Machtou P, Ruddle C, et al: *Analysis of mechanical preparations in extracted teeth using ProTaper rotary instruments: value of the safety quotient*, J Endod 2003;29:.
54. Boessler C, Paqué F, Peters OA. *The effect of electropolishing on torque and force during simulated root canal preparation with ProTaper shaping files*, J Endod 2009;35: 1.
55. Boessler C, Peters OA, Zehnder M. *Impact of lubricant parameters on rotary instrument torque and force*, J Endod 2007;33: 3.
56. Bonaccorso A, Canatatore G, Condorelli GG, et al: *Shaping ability of four nickel-titanium rotary instruments in simulated S-shaped canals*, J Endod 2009;35:.
57. Booth JR, Scheetz JP, Lemons JE, et al: *A comparison of torque required to fracture three different nickel-titanium*

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rotary instruments around curves of the same angle but of different radius when bound at the tip, J Endod 2003;29: 1.

58. Boutsioukis C, Kishen A. *Fluid dynamics of syringe-based irrigation to optimise anti-biofilm efficacy in root-canal disinfection*, Roots 2012;2012:.
59. Boutsioukis C, Lambrianidis T, Kastrinakis E. *Irrigant flow within a prepared root canal using various flow rates: a computational fluid dynamics study*, Int Endod J 2009;42: 2.
60. Boutsioukis C, Lambrianidis T, Verhaagen B, et al: *The effect of needle-insertion depth on the irrigant flow in the root canal: evaluation using an unsteady computational fluid dynamics model*, J Endod 2010;36:.
61. Boutsioukis C, Verhaagen B, Versluis M, et al: *Evaluation of irrigant flow in the root canal using different needle types by an unsteady computational fluid dynamics model*, J Endod 2010;36: 5.
62. Brantley WA, Luebke NH, Luebke FL, et al: *Performance of engine-driven rotary endodontic instruments with a superimposed bending deflection: V. Gates glidden and peeso drills*, J Endod 1994;20: 5.
63. Braun A, Kappes D, Kruse F, et al: *Efficiency of a novel rinsing device for the removal of pulp tissue in vitro*, Int Endod J 2005;38: 8.
64. Briseno Marroquin B, El-Sayed MA, Willershausen-Zönnchen B. *Morphology of the physiological foramen: I. Maxillary and mandibular molars*, J Endod 2004;30: 5.
65. Briseno Marroquin B, Pistorius A, Willershausen-Zönnchen B. *Canal transportation caused by a new instrumentation technique and three standard techniques*, J Endod 1996;22: 8.
66. Bryant ST, Thompson SA, Al-Omari MA, et al: *Shaping*

Telegram: @dental_k

www.konkur.in

ability of ProFile rotary nickel-titanium instruments with ISO sized tips in simulated root canals: Part 1, Int Endod J 1998;31: 4.

67. Bryant ST, Thompson SA, Al-Omari MA, et al: *Shaping ability of ProFile rotary nickel-titanium instruments with ISO sized tips in simulated root canals: Part 2*, Int Endod J 1998;31: 4.
68. Buchanan LS. *The standardized-taper root canal preparation - Part 2. GT file selection and safe handpiece-driven file use*, Int Endod J 2001;34: 1.
69. Buehler WH, Gilfrich JV, Wiley RC. *Effect of low temperature phase changes on the mechanical properties of alloys near composition NiTi*, J Appl Phys 1963;34: 5.
70. Bui T, Mitchell JC, Baumgartner JC. *Effect of electropolishing ProFile nickel-titanium rotary instruments on cyclic fatigue resistance, torsional resistance, and cutting efficiency*, J Endod 2008;34: 2.
71. Bulem ÜK, Kecici AD, Guldass HE. *Experimental evaluation of cyclic fatigue resistance of four different nickel-titanium instruments after immersion in sodium hypochlorite and/or sterilization*, J Appl Dent Sc 2013;21: 505.
72. Bürklein S, Hinschitza K, Dammaschke T, et al: *Shaping ability and cleaning effectiveness of two single-file systems in severely curved root canals of extracted teeth: Reciproc and WaveOne versus Mtwo and ProTaper*, Int Endod J 2012;45: 5.
73. Bürklein S, Schäfer E. *Apically extruded debris with reciprocating single-file and full-sequence rotary instrumentation systems*, J Endod 2012;38: 6.
74. Burleson A, Nusstein J, Reader A, et al: *The in vivo evaluation of hand/rotary/ultrasound instrumentation in*

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necrotic, human mandibular molars, J Endod 2007;33: 7.
75. Burroughs JR, Bergeron BE, Roberts MD, et al: *Shaping ability of three nickel-titanium endodontic file systems in simulated S-shaped root canals*, J Endod 2012;38: 1618.
76. Byström A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 5, 1985;1: 170.
77. Byström A, Sundqvist G. *The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy*, Int Endod J 1985;18: 1.
78. Cabral CT, Fernandes MH. *In vitro comparison of chlorhexidine and povidone-iodine on the long-term proliferation and functional activity of human alveolar bone cells*, Clin Oral Investig 2007;11: 2.
79. Card SJ, Sigurdsson A, Ørstavik D, et al: *The effectiveness of increased apical enlargement in reducing intracanal bacteria*, J Endod 2002;28: 11.
80. Caso A, Hung LK, Beirne OR. *Prevention of alveolar osteitis with chlorhexidine: a meta-analytic review*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 2.
81. Casper RB, Roberts HW, Roberts MD, et al: *Comparison of autoclaving effects on torsional deformation and fracture resistance of three innovative endodontic file systems*, J Endod 2011;37: 11.
82. Castelo-Baz P, Martín-Biedma B, Cantatore G, et al: *In vitro comparison of passive and continuous ultrasonic irrigation in simulated lateral canals of extracted teeth*, J Endod 2012;38:.
83. Chan R, Versiani MA, Friedman S, et al: *Efficacy of 3 supplementary irrigation protocols in the removal of hard*

Telegram: @dental_k

www.konkur.in

tissue debris from the mesial root canal system of mandibular molars, J Endod 2019; (in press).
84. Charles TJ, Charles JE. *The 'balanced force' concept for instrumentation of curved canals revisited*, Int Endod J 1998;31: 3.
85. Chen H, Teixeira FB, Ritter AL, et al: *The effect of intracanal anti-inflammatory medicaments on external root resorption of replanted dog teeth after extended extra-oral dry time*, Dent Traumatol 2008;24: 1.
86. Chen JE, Nurbaksh B, Layton G, et al: *Irrigation dynamics associated with positive pressure, apical negative pressure and passive ultrasonic irrigations: a computational fluid dynamics analysis*, Aust Endod J 2014;40: 2.
87. Cheung GS. *Instrument fractures: machanisms, removal of fragments, and clinical outcomes*, Endod Topics 2009;16: 1.
88. Cheung GS, Darvell BW. *Low-cycle fatigue of NiTi rotary instruments of various cross-sectional shapes*, Int Endod J 2007;40: 8.
89. Cheung GS, Liu CS. *A retrospective study of endodontic treatment outcome between nickel-titanium rotary and stainless steel hand filing techniques*, J Endod 7, 2009;35: 938-943.
90. Cheung GS, Shen Y, Darvell BW. *Does electropolishing improve the low-cycle fatigue behavior of a nickel-titanium rotary instrument in hypochlorite?*, J Endod 2007;33: 10.
91. Chow TW. *Mechanical effectiveness of root canal irrigation*, J Endod 1983;9: 11.
92. Chugal N, Clive JM, Spångberg LSW. *Endodontic infection: some biologic and treatment factors associated*

Telegram: @dental_k

www.konkur.in

with outcome, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 1.

93. Chugal NM, Clive JM, Spångberg LS. *A prognostic model for assessment of the outcome of endodontic treatment: Effect of biologic and diagnostic variables*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 3.
94. Cohenca N. Disinfection of root canal systems: the treatment of apical periodontitis 2014; Wiley Blackwell Hoboken, NJ, USA.
95. Coldero LG, McHugh S, MacKenzie D, et al: *Reduction in intracanal bacteria during root canal preparation with and without apical enlargement*, Int Endod J 2002;35: 5.
96. Coluzzi DJ. *Fundamentals of dental lasers: science and instruments*, Dent Clin North Am 2004;48: 4.
97. Coolidge ED. *The diagnosis and treatment of conditions resulting from diseased dental pulps*, J Natl Dent Assoc 1919;6:.
98. Cunningham CJ, Senia ES. *A three-dimensional study of canal curvatures in the mesial roots of mandibular molars*, J Endod 1992;18: 6.
99. Custer C. *Exact methods for locating the apical foramen*, J Natl Dent Assoc 1918;5:.
100. Cvek M, Nord CE, Hollender L. *Antimicrobial effect of root canal debridement in teeth with immature root. A clinical and microbiologic study*, Odontologisk Revy 1976;27: 1.
101. D'Amario M, Baldi M, Petricca R, et al: *Evaluation of a new nickel-titanium system to create the glide path in root canal preparation of curved canals*, J Endod 2013;39: 1581.
102. Dai L, Khechen K, Khan S, et al: *The effect of QMix, an experimental antibacterial root canal irrigant, on removal*

Telegram: @dental_k

www.konkur.in

of canal wall smear layer and debris, J Endod 2011;37: 1.
103. Dakin HD. *On the use of certain antiseptic substances in the treatment of infected wounds*, Br Med J 1915;28: 2.
104. Dandakis C, Lambrianidis T, Boura P. *Immunologic evaluation of dental patient with history of hypersensitivity reaction to sodium hypochlorite*, Endod Dent Traumatol 2000;16: 4.
105. Davies A. *The mode of action of chlorhexidine*, J Periodontal Res Suppl 1973;12:.
106. Davis JM, Maki J, Bahcall JK. *An in vitro comparison of the antimicrobial effects of various endodontic medicaments on enterococcus faecalis*, J Endod 2007;33: 5.
107. Davis RD, Marshall JG, Baumgartner JC. *Effect of early coronal flaring on working length change in curved canals using rotary nickel-titanium versus stainless steel instruments*, J Endod 2002;28: 6.
108. De Moor RJ, Blanken J, Meire M, et al: *Laser induced explosive vapor and cavitation resulting in effective irrigation of the root canal. Part 2: evaluation of the efficacy*, Lasers Surg Med 2009;41: 7.
109. de Vasconcelos RA, Murphy S, Carvalho CA, et al: *Evidence for reduced fatigue resistance of contemporary rotary instruments exposed to body temperature*, J Endod 2016;42: 5.
110. De-Deus G, Brandão MC, Barino B, et al: *Assessment of apically extruded debris produced by the single-file ProTaper F2 technique under reciprocating movement*, Oral Surg Oral Med Oral Pathol Oral Radiol & Endod 2010;110: 3.
111. De-Deus G, Namen F, Galan J, et al: *Soft chelating irrigation protocol optimizes bonding quality of*

Telegram: @dental_k

www.konkur.in

resilon/epiphany root fillings, J Endod 2008;34: 6.

112. DeFiore PM, Genov KA, Komaroff E, et al: *Nickel-titanium rotary instrument fracture: a clincal practice assessment*, Int Endod J 2006;39: 9.
113. Degerness RA, Bowles WR. *Dimension, anatomy and morphology of the mesiobuccal root canal system in maxillary molars*, J Endod 6, 2010;36: 985.
114. Delany GM, Patterson SS, Miller CH, et al: *The effect of chlorhexidine gluconate irrigation on the root canal flora of freshly extracted necrotic teeth*, Oral Surg Oral Med Oral Pathol 1982;53: 5.
115. Denton GW. *Chlorhexidine*, Block SS Disinfection, sterilization and preservation 1991; Lea and Febiger Philadelphia 274-289.
116. Desai P, Himel V. *Comparative safety of various intracanal irrigation systems*, J Endod 2009;35: 4.
117. Dietz DB, Di Fiore PM, Bahcall JK, et al: *Effect of rotational speed on the breakage of nickel-titanium rotary files*, J Endod 2000;26: 2.
118. DiVito E, Peters OA, Olivi G. *Effectiveness of the erbium:YAG laser and new design radial and stripped tips in removing the smear layer after root canal instrumentation*, Lasers Med Sci 2012;27:.
119. Duerig TW. *Some unresolved aspects of nitinol*, Mat Sc Eng A 2006;438–440: 69.
120. ElAyouti A, Weiger R, Lost C. *The ability of root ZX apex locator to reduce the frequency of overestimated radiographic working length*, J Endod 2002;28: 2.
121. Elnaghy AM. *Cyclic fatigue resistance of ProTaper next nickel-titanium rotary files*, Int Endod J 2014;47: 11.
122. Elnaghy AM, Mandorah A, Elsaka SE. *Effectiveness of XP-endo finisher, EndoActivator, and file agitation on*

Telegram: @dental_k

www.konkur.in

debris and smear layer removal in curved root canals: a comparative study, Odontology 2017;105: 2.

123. Emilson CG, Ericson T, Heyden G, et al: *Uptake of chlorhexidine to hydroxyapatite*, J Periodontal Res Suppl 1973;12:.
124. European Society of Endodontology. *Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology*, Int Endod J 2006;39: 12.
125. Estrela C, Estrela CR, Barbin EL, et al: *Mechanism of action of sodium hypochlorite*, Braz Dent J 2002;13: 2.
126. Estrela C, Estrela CR, Decurcio DA, et al: *Antimicrobial efficacy of ozonated water, gaseous ozone, sodium hypochlorite and chlorhexidine in infected human root canals*, Int Endod J 2007;40: 2.
127. Eun HC, Lee AY, Lee YS. *Sodium hypochlorite dermatitis*, Contact Derm 1984;11: 1.
128. Evans MD, Baumgartner JC, Khemaleelakul SU, et al: *Efficacy of calcium hydroxide: chlorhexidine paste as an intracanal medication in bovine dentin*, J Endod 2003;29: 5.
129. Fairbourn DR, McWalter GM, Montgomery S. *The effect of four preparation techniques on the amount of apically extruded debris*, J Endod 1987;13: 3.
130. Falk KW, Sedgley CM. *The influence of preparation size on the mechanical efficacy of root canal irrigation in vitro*, J Endod 2005;31: 10.
131. Fardal O, Turnbull RS. *A review of the literature on use of chlorhexidine in dentistry*, J Am Dent Assoc 1986;112: 6.
132. Faria G, Celes MR, De Rossi A, et al: *Evaluation of chlorhexidine toxicity injected in the paw of mice and*

Telegram: @dental_k

www.konkur.in

added to cultured l929 fibroblasts, J Endod 2007;33: 6.
133. Fava LR. *The double-flared technique: an alternative for biomechanical preparation*, J Endod 1983;9: 2.
134. Fidler A. *Kinematics of 2 reciprocating endodontic motors: the difference between actual and set values*, J Endod 2014;40: 7.
135. Fouad AF, Krell KV, McKendry DJ, et al: *Clinical evaluation of five electronic root canal length measuring instruments*, J Endod 1990;16: 9.
136. Frais S, Ng YL, Gulabivala K. *Some factors affecting the concentration of available chlorine in commercial sources of sodium hypochlorite*, Int Endod J 2001;34: 3.
137. Frank AL. *Therapy for the divergent pulpless tooth by continued apical formation*, J Am Dent Assoc 1966;72: 1.
138. Friedman S. *Management of post-treatment endodontic disease: a current concept of case selection*, Aust Endod J 2000;26: 3.
139. Friedman S. *Prognosis of initial endodontic therapy*, Endod Topics 2002;2: 1.
140. Friedman S, Abitbol T, Lawrence HP. *Treatment outcome in endodontics: the Toronto study. Phase 1: Initial treatment*, J Endod 2003;29: 12.
141. Gambarini G. *Cyclic fatigue of nickel-titanium rotary instruments after clinical use with low- and high-torque endodontic motors*, J Endod 2001;27: 12.
142. Gambarini G. *Rationale for the use of low-torque endodontic motors in root canal instrumentation*, Endod Dent Traumatol 2000;16: 3.
143. Gambarini G, Gerosa R, De Luca M, et al: *Mechanical properties of a new and improved nickel-titanium alloy for endodontic use: an evaluation of file flexibility*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: 6.

Telegram: @dental_k

www.konkur.in

144. Gambarini G, Grande NM, Plotino G, et al: *Fatigue resistance of engine-driven rotary nickel-titanium instruments produced by new manufacturing methods*, J Endod 2008;34: 8.
145. Garofalo RR, Ede EN, Dorn SO, et al: *Effect of electronic apex locators on cardiac pacemaker function*, J Endod 2002;28: 12.
146. Garvey LH, Roed-Petersen J, Husum B. *Anaphylactic reactions in anaesthetised patients - four cases of chlorhexidine allergy*, Acta Anaesthesiol Scand 2001;45: 10.
147. Gatewood RS, Himel VT, Dorn SO. *Treatment of the endodontic emergency: a decade later*, J Endod 1990;16: 6.
148. Gavini G, Santos MD, Caldeira CL, et al: *Nickel-titanium instruments in endodontics: a concise review of the state of the art*, Braz Oral Res Suppl 1, 2018;32:.
149. Gazzaneo I, Vieira GCS, Perez AR, et al: *Root canal disinfection concentration, and retention time*, J Endod 2019;45: 6.
150. George R, Meyers IA, Walsh LJ. *Laser activation of endodontic irrigants with improved conical laser fiber tips for removing smear layer in the apical third of the root canal*, J Endod 2008;34: 12.
151. Ghori S, Gulabivala K, Premdas C, et al: *Evaluation of the antimicrobial efficacy of electrochemically activated water on selected isolates from the root canal*, Int Endod J 2002;85: 35.
152. Giardino L, Ambu E, Savoldi E, et al: *Comparative evaluation of antimicrobial efficacy of sodium hypochlorite, MTAD, and tetraclean against enterococcus faecalis biofilm*, J Endod 2007;33: 7.

Telegram: @dental_k

www.konkur.in

153. Glosson CR, Haller RH, Dove B, et al: *A comparison of root canal preparations using Ni-Ti hand, Ni-Ti engine-driven, and K-Flex endodontic instruments*, J Endod 1995;21:.
154. Gluskin A, Peters CI, Peters OA. *Minimally invasive endodontics: challenging prevailing paradigms*, Br Dent J 2014;216:.
155. Gluskin AH, Brown DC, Buchanan LS. *A reconstructed computerized tomographic comparison of Ni-Ti rotary GT files versus traditional instruments in canals shaped by novice operators*, Int Endod J 2001;34: 6.
156. Goerig AC, Michelich RJ, Schultz HH. *Instrumentation of root canals in molar using the step-down technique*, J Endod 1982;8: 12.
157. Goldberg F, De Silvio AC, Manfre S, et al: *In vitro measurement accuracy of an electronic apex locator in teeth with simulated apical root resorption*, J Endod 2002;28: 6.
158. Goldman M, Kronman JH, Goldman LB, et al: *New method of irrigation during endodontic treatment*, J Endod 1976;2: 9.
159. Gomes BP, Ferraz CC, Vianna ME, et al: *In vitro antimicrobial activity of several concentrations of sodium hypochlorite and chlorhexidine gluconate in the elimination of enterococcus faecalis*, Int Endod J 2001;34: 6.
160. Gomes BP, Sato E, Ferraz CC, et al: *Evaluation of time required for recontamination of coronally sealed canals medicated with calcium hydroxide and chlorhexidine*, Int Endod J 2003;36: 9.
161. Goodman A, Reader A, Beck M, et al: *An in vitro comparison of the efficacy of the step-back technique*

Telegram: @dental_k

www.konkur.in

versus a step-back/ultrasonic technique in human mandibular molars, J Endod 1985;11: 6.

162. Grande NM, Plotino G, Butti A, et al: *Modern endodontic NiTi systems: morphological and technical characteristics, Part I: "new generation" Ni-Ti systems*, Endod Therapy 2005;5: 11.
163. Grande NM, Plotino G, Pecci R, et al: *Cyclic fatigue resistance and three-dimensional analysis of instruments from two nickel-titanium systems*, Int Endod J 2006;39: 10.
164. Grawehr M, Sener B, Waltimo T, et al: *Interactions of ethylenediamine tetraacetic acid with sodium hypochlorite in aqueous solutions*, Int Endod J 2003;36: 6.
165. Greenstein G, Berman C, Jaffin R. *Chlorhexidine. An adjunct to periodontal therapy*, J Periodontol 1986;57: 6.
166. Gu LS, Kim JR, Ling J, et al: *Review of contemporary irrigant agitation techniques and devices*, J Endod 2009;35:.
167. Guivarc'h M, Ordioni U, Ahmed HM, et al: *Sodium hypochlorite accident: a systematic review*, J Endod 2017;43: 1.
168. Gulabivala K: Personal communication, 2009.
169. Gulabivala K, Ng Y-L, Gilbertson M, et al: *The fluid mechanics of root canal irrigation*, Physiol Meas 2010;31: 12.
170. Gulabivala K, Patel B, Evans G, et al: *Effects of mechanical and chemical procedures on root canal surfaces*, Endod Topics 2005;10: 1.
171. Gulabivala K, Stock CJ, Lewsey JD, et al: *Effectiveness of electrochemically activated water as an irrigant in an infected tooth model*, Int Endod J 2004;37: 9.
172. Gutmann JL, Gao Y. *Alteration in the inherent metallic and surface properties of nickel-titanium root canal*

Telegram: @dental_k

www.konkur.in

instruments to enhance performance, durability and safety: a focused review, Int Endod J 2011;45: 2.

173. Ha JH, Park SS. *Influence of glide path on the screw-in effect and torque of nickel-titanium rotary files in simulated resin root canals*, Restor Dent Endod 2012;37: 4.
174. Haapasalo HK, Sirén EK, Waltimo TMT, et al: *Inactivation of local root canal medicaments by dentine: an in vitro study*, Int Endod J 2000;22: 2.
175. Haapasalo M: *Composition and method for irrigation of a prepared dental root canal*, 2008.
176. Haapasalo M, Shen Y, Qian W, et al: *Irrigation in endodontics*, Dent Clin North Am 2010;54: 2.
177. Haapasalo M, Shen Y, Wang Z, et al: *Irrigation in endodontics*, Br Dent J 2014;216: 6.
178. Haapasalo M, Udnaes T, Endal U. *Persistent, recurrent, and acquired infection of the root canal system post-treatment*, Endod Topics 2003;6: 1.
179. Haapasalo M, Wang Z, Shen Y, et al: *Tissue dissolution by a novel multisonic ultracleaning system and sodium hypochlorite*, J Endod 2014;40: 8.
180. Habets JM, Geursen-Reitsma AM, Stolz E, et al: *Sensitization to sodium hypochlorite causing hand dermatitis*, Contact Derm 1986;15: 3.
181. Haenni S, Schmidlin PR, Müller B, et al: *Chemical and antimicrobial properties of calcium hydroxide mixed with irrigating solutions*, Int Endod J 2003;36: 2.
182. Haikel Y, Gasser P, Allemann C. *Dynamic fracture of hybrid endodontic hand instruments compared with traditional files*, J Endod 1991;17: 5.
183. Haikel Y, Serfaty R, Bateman G, et al: *Dynamic and cyclic fatigue of engine-driven rotary nickel-titanium*

Telegram: @dental_k

www.konkur.in

endodontic instruments, J Endod 1999;25: 6.

184. Haikel Y, Serfaty R, Wilson P, et al: *Mechanical properties of nickel-titanium endodontic instruments and the effect of sodium hypochlorite treatment*, J Endod 1998;24: 11.
185. Hamblin MR, Hasan T. *Photodynamic therapy: a new antimicrobial approach to infectious disease?*, Photochem Photobiol Sci 2004;3: 5.
186. Hand RE, Smith ML, Harrison JW. *Analysis of the effect of dilution on the necrotic tissue dissolution property of sodium hypochlorite*, J Endod 1978;4: 2.
187. Hasselgren G. *Where shall the root filling end?*, NY St Dent J 1994;60: 6.
188. Hasselgren G, Olsson B, Cvek M. *Effects of calcium hydroxide and sodium hypochlorite on the dissolution of necrotic porcine muscle tissue*, J Endod 1988;14: 3.
189. Hata G, Hayami S, Weine FS, et al: *Effectiveness of oxidative potential water as a root canal irrigant*, Int Endod J 2001;34: 4.
190. Hayashi Y, Yoneyama T, Yahata Y, et al: *Phase transformation behaviour and bending properties of hybrid nickel-titanium rotary endodontic instruments*, Int Endod J 2007;40:.
191. Haznedaroglu F, Ersev H. *Tetracycline HCl solution as a root canal irrigant*, J Endod 2001;27: 12.
192. Heling I, Chandler NP. *Antimicrobial effect of irrigant combinations within dentinal tubules*, Int Endod J 1998;31: 1.
193. Hems RS, Gulabivala K, Ng YL, et al: *An in vitro evaluation of the ability of ozone to kill a strain of enterococcus faecalis*, Int Endod J 2005;38: 1.
194. Hermann B. Calciumhydroxyd als Mittel zum Behandeln

Telegram: @dental_k

www.konkur.in

und Füllen von Zahnwurzelkanälen 1920; Germany University of Würzburg.

195. Herold KS, Johnson BR, Wenckus CS. *A scanning electron microscopy evaluation of microfractures, deformation and separation in EndoSequence and profile nickel-titanium rotary files using an extracted molar tooth model*, J Endod 2007;33: 6.
196. Hess W. *Formation of root canals in human teeth*, J Natl Dent Assoc 1921;3:.
197. Hidalgo E, Dominguez C. *Mechanisms underlying chlorhexidine-induced cytotoxicity*, Toxicol In Vitro 2001;15: 4-5.
198. Hilfer PB, Bergeron BE, Mayerchak MJ, et al: *Multiple autoclave cycle effects on cyclic fatigue of nickel-titanium rotary files produced by new manufacturing methods*, J Endod 2011;37: 1.
199. Hilt BR, Cunningham CJ, Shen C, et al: *Torsional properties of stainless-steel and nickel-titanium files after multiple autoclave sterilizations*, J Endod 2000;26: 2.
200. Horiba N, Hiratsuka K, Onoe T, et al: *Bactericidal effect of electrolyzed neutral water on bacteria isolated from infected root canals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 1.
201. Hsieh YD, Gau CH, Kung Wu SF, et al: *Dynamic recording of irrigating fluid distribution in root canals using thermal image analysis*, Int Endod J 2007;40: 1.
202. Huang TY, Gulabivala K, Ng YL. *A bio-molecular film ex-vivo model to evaluate the influence of canal dimensions and irrigation variables on the efficacy of irrigation*, Int Endod J 2008;41: 1.
203. Hübscher W, Barbakow F, Peters OA. *Root canal preparation with FlexMaster: asessment of torque and*

Telegram: @dental_k

www.konkur.in

force in relation to canal anatomy, Int Endod J 2003;36: 12.

204. Hugo WB, Longworth AR. *Some aspects of the mode of action of chlorhexidine*, J Pharmacol 1964;16:.
205. Hülsmann M, Hahn W. *Complications during root canal irrigation—literature review and case reports*, Int Endod J 2000;33: 3.
206. Hülsmann M, Heckendorff M, Lennon A. *Chelating agents in root canal treatment: mode of action and indications for their use*, Int Endod J 2003;36: 12.
207. Hülsmann M, Peters OA, Dummer PMH. *Mechanical preparation of root canals: shaping goals, techniques and means*, Endod Topics 2005;10: 1.
208. Hülsmann M, Pieper K. *Use of an electronic apex locator in the treatment of teeth with incomplete root formation*, Endod Dent Traumatol 1989;5: 5.
209. Hülsmann M, Rödig T, Nordmeyer S. *Complications during root canal irrigation*, Endod Topics 2007;16: 1.
210. Hülsmann M, Schade M, Schäfers F. *A comparative study of root canal preparation with HERO 642 and Quantec SC rotary Ni-Ti instruments*, Int Endod J 2001;34: 7.
211. Ichikawa K, Nakamura HK, Ogawa N, et al: *R&D of long-term life support system by using electrochemically activated biofilm reactor of aquatic animals for space examinations*, Biol Sci Space 1999;13: 4.
212. Ingle JI. *A standardized endodontic technique using newly development instruments and filling materials*, Oral Surg Oral Med Oral Pathol 1, 1961;14: 83.
213. Isom TL, Marshall JG, Baumgartner JC. *Evaluation of root thickness in curved canals after flaring*, J Endod 1995;21: 7.
214. Izu KH, Thomas SJ, Zhang P, et al: *Effectiveness of*

Telegram: @dental_k

www.konkur.in

sodium hypochlorite in preventing inoculation of periapical tissue with contaminated patency files, J Endod 2004;30: 2.

215. Jeansonne MJ, White RR. *A comparison of 2.0% chlorhexidine gluconate and 5.25% sodium hypochlorite as antimicrobial endodontic irrigants*, J Endod 1994;20: 6.
216. Jensen LE, Murphy S, Williamson AE, et al: *Root canal preparation in mandibular premolars with TRUShape and Vortex Blue: A micro-computed tomography study*, Aust Endod J 2019;45: 1.
217. Jensen SA, Walker TL, Hutter JW, et al: *Comparison of the cleaning efficacy of passive sonic activation and passive ultrasonic activation after hand instrumentation in molar root canals*, J Endod 1999;25: 11.
218. Jiang LM, Lak B, Eijsvogels LM, et al: *Comparison of the cleaning efficacy of different final irrigation techniques*, J Endod 2012;38:.
219. Johal S, Baumgartner JC, Marshall FJ. *Comparison of the antimicrobial efficacy of 1.3% NaOCl/BioPure MTAD to 5.25% NaOCl/15% EDTA for root canal irrigation*, J Endod 2007;33:.
220. Johnson E, Lloyd A, Kuttler S, et al: *Comparison between a novel nickel-titanium alloy and 508 nitinol on the cyclic fatigue life of ProFile 25/.04 rotary instruments*, J Endod 2008;34: 11.
221. Jungbluth H, Peters C, Peters OA, et al: *Physicochemical and pulp tissue dissolution properties of some household bleach brands compared with a dental sodium hypochlorite solution*, J Endod 2012;38:.
222. Karagöz-Küçükay I, Ersev H, Engin-Akkoca E, et al: *Effect of rotational speed on root canal preparation with Hero 642 rotary Ni-Ti instruments*, J Endod 2003;29: 7.

Telegram: @dental_k

www.konkur.in

223. Keate KC, Wong M. *Comparison of endodontic file tip quality*, J Endod 1990;16: 10.
224. Kell T, Arzarpazhooh A, Peters OA, et al: *Torsional profiles of new and used 20/.06 GT series X and GT rotary endodontic instruments*, J Endod 2009;35: 9.
225. Kho P, Baumgardner JC. *A comparison of the antimicrobial efficacy of NaOCl/Biopure MTAD versus NaOCl/EDTA against enterococcus faecalis*, J Endod 2006;32:.
226. Kishen A. *Advanced therapeutic options for endodontic biofilms*, Endod Topics 2012;22:.
227. Kishor N. *Oral tissue complications during endodontic irrigation-a literature review*, NY St Dent J 2013;79: 37-42.
228. Kleier DJ, Averbach RE, Mehdipour O. *The sodium hypochlorite accident: experience of diplomates of the American Board of Endodontics*, J Endod 2008;34: 11.
229. Klotz MD, Gerstein H, Bahn AN. *Bacteremia after topical use of prednisolone in infected pulps*, J Am Dent Assoc 1965;71: 4.
230. Kobayashi C, Suda H. *New electronic canal measuring device based on the ratio method*, J Endod 1994;20: 3.
231. Koch KA, Brave DG. *Real world endo sequence file*, Dent Clin North Am 2004;48: 1.
232. Kolosowski KP, Sodhi RN, Kishen A, et al: *Qualitative time-of-flight secondary ion mass spectrometry analysis of root dentin irrigated with sodium hypochlorite, EDTA, or chlorhexidine*, J Endod 2015;41: 10.
233. Komorowski R, Grad H, Wu XY, et al: *Antimicrobial substantivity of chlorhexidine-treated bovine root dentin*, J Endod 2000;26: 6.
234. Kramkowski TR, Bahcall J. *An in vitro comparison of*

Telegram: @dental_k

www.konkur.in

torsional stress and cyclic fatigue resistance of ProFile GT and ProFile GT series X rotary nickel-titanium files, J Endod 2009;35: 3.

235. Krause TA, Liewehr FR, Hahn CL. *The antimicrobial effect of MTAD, sodium hypochlorite, doxycycline, and citric acid on enterococcus faecalis*, J Endod 2007;33: 1.
236. Krautheim AB, Jermann TH, Bircher AJ. *Chlorhexidine anaphylaxis: case report and review of the literature*, Contact Derm 2004;50: 3.
237. Krell KV, Johnson RJ. *Irrigation patterns of ultrasonic endodontic files. Part II. Diamond coated files.*, J Endod 2, 1988;14: 535.
238. Krishan R, Paque F, Ossareh A, et al: *Impacts of conservative endodontic cavity on root canal instrumentation efficacy and resistance to fracture assessed in incisors, premolars, and molars*, J Endod 2014;40: 8.
239. Kuhn G, Jordan L. *Fatigue and mechanical properties of nickel-titanium endodontic instruments*, J Endod 2002;28: 10.
240. Kuyk JK, Walton RE. *Comparison of the radiographic appearance of root canal size to its actual diameter*, J Endod 1990;16: 11.
241. Kyomen SM, Caputo AA, White SN. *Critical analysis of the balanced force technique in endodontics*, J Endod 1994;20: 7.
242. Lambrianidis T, Margelos J, Beltes P. *Removal efficiency of calcium hydroxide dressing from the root canal*, J Endod 1999;25: 2.
243. Larsen CM, Watanabe I, Glickman GN, et al: *Cyclic fatigue analysis of a new generation of nickel titanium rotary instruments*, J Endod 2009;35: 3.

Telegram: @dental_k

www.konkur.in

244. Lautenschlager EP, Jacobs JJ, Marshall GW, et al: *Brittle and ductile torsional failures of endodontic instruments*, J Endod 1977;3: 5.
245. Law A. *Considerations for regeneration procedures*, J Endod Suppl, 2013;3: 39.
246. Leeb J. *Canal orifice enlargement as related to biomechanical preparation*, J Endod 1983;9: 11.
247. Lenet BJ, Komorowski R, Wu XY, et al: *Antimicrobial substantivity of bovine root dentin exposed to different chlorhexidine delivery vehicles*, J Endod 2000;26: 11.
248. Leonardo MR, Tanomaru Filho M, Silva LA, et al: *In vivo antimicrobial activity of 2% chlorhexidine used as a root canal irrigating solution*, J Endod 1999;25: 3.
249. Lewis BB, Chestner SB. *Formaldehyde in dentistry: a review of mutagenic and carcinogenic potential*, J Am Dent Assoc 1981;103: 3.
250. Li UM, Lee BS, Shih CT, et al: *Cyclic fatigue of endodontic nickel titanium rotary instruments: static and dynamic tests*, J Endod 2002;28: 6.
251. Liang YH, Jiang LM, Jiang L, et al: *Radiographic healing after a root canal treatment performed in single-rooted teeth with and without ultrasonic activation of the irrigant: a randomized controlled trial*, J Endod 2013;39:.
252. Lim SS, Stock CJ. *The risk of perforation in the curved canal: anticurvature filing compared with the stepback technique*, Int Endod J 1987;20: 1.
253. Lima KC, Fava LR, Siqueira JF Jr. *Susceptibilities of enterococcus faecalis biofilms to some antimicrobial medications*, J Endod 2001;27: 10.
254. Lin LM, Rosenberg PA, Lin J. *Do procedural errors cause endodontic treatment failure?*, J Am Dent Assoc 2005;136: 2.

Telegram: @dental_k

www.konkur.in

255. Lindskog S, Pierce AM, Blomlof L. *Chlorhexidine as a root canal medicament for treating inflammatory lesions in the periodontal space*, Endod Dent Traumatol 1998;14: 4.
256. Lipski M, Buczkowska-Radlinska J, Gora M. *Loss of sight caused by calcium hydroxide paste accidentally splashed into the eye during endodontic treatment: case report*, J Can Dent Assoc 2012;78:.
257. Loe H. *Does chlorhexidine have a place in the prophylaxis of dental diseases?*, J Periodontal Res Suppl 1973;12:.
258. Loe H, Schiott CR. *The effect of mouthrinses and topical application of chlorhexidine on the development of dental plaque and gingivitis in man*, J Periodontal Res 1970;5: 2.
259. Loshon CA, Melly E, Setlow B, et al: *Analysis of the killing of spores of bacillus subtilis by a new disinfectant, Sterilox*, J Appl Microbiol 2001;91: 6.
260. Loushine RJ, Weller RN, Hartwell GR. *Stereomicroscopic evaluation of canal shape following hand, sonic, and ultrasonic instrumentation*, J Endod 1989;15: 9.
261. Lumley PJ, Walmsley AD. *Effect of precurving on the performance of endosonic K files*, J Endod 1992;18: 5.
262. Lumley PJ, Walmsley AD, Walton RE, et al: *Cleaning of oval canals using ultrasonic or sonic instrumentation*, J Endod 1993;19: 9.
263. Lussi A, Messerli L, Hotz P, et al: *A new non-instrumental technique for cleaning and filling root canals*, Int Endod J 1995;28: 1.
264. Lussi A, Nussbacher U, Grosrey J. *A novel noninstrumented technique for cleansing the root canal system*, J Endod 1993;19: 11.

Telegram: @dental_k

www.konkur.in

265. Macedo R, Verhaagen B, Rivas DF, et al: *Cavitation measurement during sonic and ultrasonic activated irrigation*, J Endod 2014;40: 4.
266. Malkhassian G. *Antibacterial effectiveness of a final rinse with MTAD and intracanal medication with 2% chlorhexidine gel in teeth with apical periodontitis*, Department of endodontics 2007; University of Toronto Canada.
267. Malkhassian G, Manzur AJ, Legner M, et al: *Antibacterial efficacy of MTAD final rinse and two percent chlorhexidine gel medication in teeth with apical periodontitis: a randomized double-blinded clinical trial*, J Endod 2009;35: 11.
268. Manzur A, González AM, Pozos A, et al: *Bacterial quantification in teeth with apical periodontitis related to instrumentation and different intracanal medications: a randomized clinical trial*, J Endod 2007;33: 2.
269. Marais JT, Brozel VS. *Electro-chemically activated water in dental unit water lines*, Br Dent J 1999;187: 3.
270. Marais JT, Williams WP. *Antimicrobial effectiveness of electro-chemically activated water as an endodontic irrigation solution*, Int Endod J 2001;34: 3.
271. Margelos J, Eliades G, Verdelis C, et al: *Interaction of calcium hydroxide with zinc oxide-eugenol type sealers: a potential clinical problem*, J Endod 1997;23: 1.
272. Margolis HC, Moreno EC, Murphy BJ. *Importance of high pKA acids in cariogenic potential of plaque*, J Dent Res 1985;64: 5.
273. Markan S, Lehl G, Kapoor S. *Recent advances of nanotechnology in endodontics, conservative and preventive dentistry-A review*, J Dent Oral Biol 2017;2:.
274. Marshall FJ, Pappin JB. A crown-down pressureless

Telegram: @dental_k

www.konkur.in

preparation root canal enlargement technique. Technique Manual 1980; Oregon Health Sciences University Portland, OR.

275. Mayeda DL, Simon JH, Aimar DF, et al: *In vivo measurement accuracy in vital and necrotic canals with the endex apex locator*, J Endod 1993;19: 11.
276. Mayer BE, Peters OA, Barbakow F. *Effects of rotary instruments and ultrasonic irrigation on debris and smear layer scores: a scanning electron microscopic study*, Int Endod J 2002;35: 7.
277. McCann JT, Keller DL, LaBounty GL. *Remaining dentin/cementum thickness after hand or ultrasonic instrumentation*, J Endod 1990;16: 3.
278. McRay B, Cox TC, Cohenca N, et al: *A micro-computed tomography-based comparison of the canal transportation and centering ability of ProTaper Universal rotary and WaveOne reciprocating files*, Quintessence Int 2014;45:.
279. Messer HH, Feigal RJ. *A comparison of the antibacterial and cytotoxic effects of parachlorophenol*, J Dent Res 1985;64: 5.
280. Metzger Z, Teperovich E, Zary R, et al: *The Self-Adjusting File (SAF). Part 1: respecting the root canal anatomy—a new concept of endodontic files and its implementation*, J Endod 2010;36: 4.
281. Mickel AK, Chogle S, Liddle J, et al: *The role of apical size determination and enlargement in the reduction of intracanal bacteria*, J Endod 2007;33: 1.
282. Miyai K, Ebihara A, Hayashi Y, et al: *Influence of phase transformation on the torsional and bending properties of nickel-titanium rotary endodontic instruments*, Int Endod J 2006;39: 2.
283. Mize SB, Clement DJ, Pruett JP, et al: *Effect of*

Telegram: @dental_k

www.konkur.in

sterilization on cyclic fatigue of rotary nickel-titanium endodontic instruments, J Endod 1998;24: 12.

284. Mizutani T, Ohno N, Nakamura H. *Anatomical study of the root apex in the maxillary anterior teeth*, J Endod 1992;18: 7.
285. Mohammadi Z. *Sodium hypochlorite in endodontics: an update review*, Int Dent J 2008;58:.
286. Mohammadi Z, Abbott PV. *The properties and applications of chlorhexidine in endodontics*, Int Endod J 2009;42:.
287. Molina B, Glickman G, Vandrangi P, et al: *Evaluation of root canal debridement of human molars using the GentleWave system*, J Endod 2015;41: 10.
288. Möller AJ. *Microbiological examination of root canals and periapical tissues of human teeth, Methodological studies*, Odontol Tidskr 1966;74: 5.
289. Möller AJ, Fabricius L, Dahlén G, et al: *Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys*, Scand J Dent Res 1981;89: 6.
290. Moorer WR, Wesselink PR. *Factors promoting the tissue dissolving capability of sodium hypochlorite*, Int Endod J 1982;15: 4.
291. Morgental RD, Singh A, Sappal H, et al: *Dentin inhibits the antibacterial effect of new and conventional endodontic irrigants*, J Endod 2013;39: 4.
292. Morgental RD, Vier-Pelisser FV, Kopper PMP, et al: *Cutting efficiency of conventional and martensitic nickel-titanium instruments for coronal flaring*, J Endod 2013;39: 12.
293. Mullaney TP. *Instrumentation of finely curved canals*, Dent Clin North Am 1979;23: 4.
294. Müller P, Guggenheim B, Schmidlin PR. *Efficacy of*

Telegram: @dental_k

www.konkur.in

gasiform ozone and photodynamic therapy on a multispecies oral biofilm in vitro, Eur J Oral Sci 2007;115: 1.

295. Nagayoshi M, Kitamura C, Fukuizumi T, et al: *Antimicrobial effect of ozonated water on bacteria invading dentinal tubules*, J Endod 2004;30: 11.
296. Nair PN. *On the causes of persistent apical periodontitis: a review*, Int Endod J 2006;39: 4.
297. Nair PN, Sjögren U, Krey G, et al: *Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study*, J Endod 1990;16: 12.
298. Nakagawa RK, Alves JL, Buono VT, et al: *Flexibility and torsional behaviour of rotary nickel-titanium PathFile, RaCe ISO 10, Scout RaCe and stainless steel K-File hand instruments*, Int Endod J 2014;47: 290.
299. Neelakantan P, Romero M, Vera J, et al: *Biofilms in endodontics-current status and future directions*, Int J Mol Sci 2017;18: 8.
300. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health*, Int Endod J 2011;44: 7.
301. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2: tooth survival*, Int Endod J 2011;44: 7.
302. Nielsen BA, Baumgartner JC. *Comparison of the EndoVac system to needle irrigation of root canals*, J Endod 2007;33: 5.
303. Alves Vde O, Bueno CE, Cunha RS, et al: *Comparison among manual instruments and PathFile and Mtwo rotary*

Telegram: @dental_k

www.konkur.in

instruments to create a glide path in the root canal preparation of curved canals, J Endod 2012;38: 1.

304. O'Hoy PY, Messer HH, Palamara JE. *The effect of cleaning procedures on fracture properties and corrosion of NiTi files*, Int Endod J 2003;36: 11.
305. Okano M, Nomura M, Hata S, et al: *Anaphylactic symptoms due to chlorhexidine gluconate*, Arch Dermatol 1989;125: 1.
306. Oncag O, Hosgor M, Hilmioglu S, et al: *Comparison of antibacterial and toxic effects of various root canal irrigants*, Int Endod J 2003;36: 6.
307. Ordinola-Zapata R, Bramante CM, Brandao Garcia R, et al: *The antimicrobial effect of new and conventional endodontic irrigants on intra-orally infected dentin*, Acta Odontol Scand 2012; (epub ahead of print).
308. Ørstavik D, Haapasalo M. *Disinfection by endodontic irrigants and dressings of experimentally infected dentinal tubules*, Endod Dent Traumatol 1990;6: 4.
309. Ørstavik D, Pitt Ford TR. Essential endodontology: prevention and treatment of apical periodontitis ed 1 1998; Blackwell Science Oxford, UK.
310. Otsuka K, Ren X. *Physical metallurgy of Ti–Ni-based shape memory alloys*, Progr Mater Sci 2005;50: 511.
311. Ounsi HF, Salameh Z, Al-Shalan T, et al: *Effect of clinical use of the cyclic fatigue resiatance of ProTaper Nickel-titanium rotary instruments*, J Endod 2007;33: 6.
312. Paqué F, Ganahl D, Peters OA. *Effects of root canal preparation on apical geometry assessed by micro-computed tomography*, J Endod 2009;35: 7.
313. Paqué F, Musch U, Hülsmann M. *Comparison of root canal preparation using RaCe and ProTaper rotary Ni-Ti instruments*, Int Endod J 2005;38: 1.

Telegram: @dental_k

www.konkur.in

314. Paqué F, Rechenberg DK, Zehnder M. *Reduction of hard-tissue debris accumulation during rotary root canal instrumentation by etidronic acid in a sodium hypochlorite irrigant*, J Endod 2012;38: 5.
315. Paquette L. *The effectiveness of chlorhexidine gluconate as an intracanal medication in endodontics: an in vivo microbiological study*, Department of Endodontics 2004; University of Toronto Canada.
316. Paquette L, Legner M, Fillery ED, et al: *Antibacterial efficacy of chlorhexidine gluconate intracanal medication in vivo*, J Endod 2007;33: 7.
317. Paredes-Vieyra J, Enriquez FJ. *Success rate of single- versus two-visit root canal treatment of teeth with apical periodontitis: a randomized controlled trial*, J Endod 2012;38: 9.
318. Park H. *A comparison of greater taper files, ProFiles, and stainless steel files to shape curved root canals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 6.
319. Parris J, Wilcox L, Walton R. *Effectiveness of apical clearing: histological and radiographical evaluation*, J Endod 1994;20: 5.
320. Pasqualini D, Mollo L, Scotti N, et al: *Postoperative pain after manual and mechanical glide path: a randomized clinical trial*, J Endod 2012;38: 1.
321. Patino PV, Biedma BM, Liebana CR, et al: *The influence of a manual glide path on the separation rate of NiTi rotary instruments*, J Endod 2005;31: 2.
322. Pereira ES, Singh R, Arias A, et al: *In vitro assessment of torque and force generated by novel ProTaper next instruments during simulated canal preparation*, J Endod 2013;39: 12.
323. Pereira EJ, Gomes RO, Leroy AM, et al: *Mechanical*

Telegram: @dental_k

www.konkur.in

behavior of M-Wire and conventional NiTi wire used to manufacture rotary endodontic instruments, Dent Mater 2013;29: 12.

324. Peters CI, Koka RS, Highsmith S, et al: *Calcium hydroxide dressings using different preparation and application modes: density and dissolution by simulated tissue pressure*, Int Endod J 2005;38:.
325. Peters LB, van Winkelhoff AJ, Buijs JF, et al: *Effects of instrumentation, irrigation and dressing with calcium hydroxide on infection in pulpless teeth with periapical bone lesions*, Int Endod J 2002;35: 1.
326. Peters OA. *Current challenges and concepts in the preparation of root canal systems: a review*, J Endod 2004;30: 8.
327. Peters OA, Arias A. *Shaping, disinfection and obturation for molars*, Peters OA The guidebook for molar endodontics 2016; Springer Verlag Heidelberg 133-167.
328. Peters OA, Arias A, Paque F. *A micro-computed tomographic assessment of root canal preparation with a novel instrument, TRUShape, in mesial roots of mandibular molars*, J Endod 2015;41: 9.
329. Peters OA, Barbakow F. *Dynamic torque and apical forces of ProFile.04 rotary instruments during preparation of curved canals*, Int Endod J 2002;35: 4.
330. Peters OA, Boessler C, Zehnder M. *Effect of liquid and paste-type lubricants on torque values during simulated rotary root canal instrumentation*, Int Endod J 2005;38: 4.
331. Peters OA, de Azevedo Bahia MG, Pereira ES. *Contemporary root canal preparation: innovations in biomechanics*, Dent Clin North Am 2017;61: 1.
332. Peters OA, Gluskin AK, Weiss RA, et al: *An in vitro assessment of the physical properties of novel Hyflex*

Telegram: @dental_k

www.konkur.in

nickel-titanium rotary instruments, Int Endod J 2012;45: 11.

333. Peters OA, Kappeler S, Bucher W, et al: *Engine-driven preparation of curved root canals: measuring cyclic fatigue and other physical parameters*, Aust Endod J 2002;28: 1.
334. Peters OA, Kappeler S, Bucher W, et al: *Maschinelle aufbereitung gekrümmter wurzelkanäle: messaufbau zur darstellung physikalischer parameter*, Schw Monatsschr Zahnmed 2001;111:.
335. Peters OA, Morgental RD, Schulze KA, et al: *Determining cutting efficiency of nickel-titanium coronal flaring instruments used in lateral action*, J Int Endod 2013.
336. Peters OA, Paqué F. *Root canal preparation of maxillary molars with the self-adjusting file: a micro-computed tomography study*, J Endod 2011;37: 1.
337. Peters OA, Peters CI, Schönenberger K, et al: *ProTaper rotary root canal preparation: effects of canal anatomy on final shape analysed by micro CT*, Int Endod J 2003;36: 2.
338. Peters OA, Peters CI, Schönenberger K, et al: *ProTaper rotary root canal preparation: assessment of torque and force in relation to canal anatomy*, Int Endod J 2003;36: 2.
339. Peters OA, Roelicke JO, Baumann MA. *Effect of immersion in sodium hypochlorite on torque and fatigue resistance of nickel-titanium instruments*, J Endod 2007;33: 5.
340. Peters OA, Schönenberger K, Laib A. *Effects of four NiTi preparation techniques on root canal geometry assessed by micro computed tomography*, Int Endod J 2001;34: 3.
341. Pettiette MT, Delano EO, Trope M. *Evaluation of success rate of endodontic treatment performed by students with*

Telegram: @dental_k

www.konkur.in

stainless-steel K-files and nickel-titanium hand files, J Endod 2001;27: 2.

342. Pham NH, Weiner JM, Reisner GS, et al: *Anaphylaxis to chlorhexidine. Case report. Implication of immunoglobulin E antibodies and identification of an allergenic determinant*, Clin Exp Allergy 2000;30: 7.
343. Pineda F, Kuttler Y. *Mesiodistal and buccolingual roentgenographic investigation of 7,275 root canals*, Oral Surg Oral Med Oral Pathol 1972;33: 1.
344. Pirani C, Iacono F, Generali L, et al: *HyFlex EDM: superficial features, metallurgical analysis and fatigue resistance of innovative electro discharge machined NiTi rotary instruments*, Int Endod J 2016;49: 5.
345. Plotino G, Grande NM, Sorci E, et al: *A comparison of cyclic fatigue between used and new Mtwo Ni–Ti rotary instruments*, Int Endod J 2006;39: 9.
346. Plotino G, Grande NM, Sorci E, et al: *Influence of a brushing working stroke on the fatigue life of NiTi rotary instruments*, Int Endod J 2007;40: 1.
347. Portenier I, Waltimo T, Ørstavik D, et al: *Killing of enterococcus faecalis by MTAD and chlorhexidine digluconate with or without cetrimide in the presence or absence of dentine powder or BSA*, J Endod 2006;32: 2.
348. Pruett JP, Clement DJ, Carnes DL Jr. *Cyclic fatigue testing of nickel-titanium endodontic instruments*, J Endod 1997;23: 2.
349. Radcliffe CE, Potouridou L, Qureshi R, et al: *Antimicrobial activity of varying concentrations of sodium hypochlorite on the endodontic microorganisms actinomyces israelii, A. naeslundii, Candida albicans and Enterococcus faecalis*, Int Endod J 2004;37: 7.
350. Ram Z. *Effectiveness of root canal irrigation*, Oral Surg

Telegram: @dental_k

www.konkur.in

Oral Med Oral Pathol 1977;44: 2.
351. Rasimick BJ, Nekich M, Hladek MM, et al: *Interaction between chlorhexidine digluconate and EDTA*, J Endod 2008;34: 12.
352. Ray HA, Trope M. *Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration*, Int Endod J 1995;28: 1.
353. Ray JJ, Kirkpatrick TC, Rutledge RE. *Cyclic fatigue of EndoSequence and K3 rotary files in a dynamic model*, J Endod 2007;33: 12.
354. Reeh ES, Messer HH. *Long-term paresthesia following inadvertent forcing of sodium hypochlorite through perforation in maxillary incisor*, Endod Dent Traumatol 4, 1989;5: 200.
355. Regalado Farreras DC, Garcia Puente C, Estrela C. *Chemical burn in an endodontist's eye during canal treatment*, J Endod 2014;40: 8.
356. Richman MJ. *The use of ultrasonics in root canal therapy and root resection*, J Dent Med 1957;12: 1.
357. Ricucci D, Langeland K. *Apical limit of root canal instrumentation and obturation, part 2. A histological study*, Int Endod J 1998;31: 6.
358. Ricucci D, Russo J, Rutberg M, et al: *A prospective cohort study of endodontic treatments of 1,369 root canals: results after 5 years*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 6, 2011;112: 825.
359. Ringel AM, Patterson SS, Newton CW, et al: *In vivo evaluation of chlorhexidine gluconate solution and sodium hypochlorite solution as root canal irrigants*, J Endod 1982;8: 5.
360. Roane JB. *Principles of preparation using the balanced force technique*, Hardin J Clark's clinical dentistry 1991;

Telegram: @dental_k

www.konkur.in

JB Lippincott Co Philadelphia 1-39.

361. Roane JB, Powell SE. *The optimal instrument design for canal preparation*, J Am Dent Assoc 1986;113: 4.
362. Roane JB, Sabala CL, Duncanson MG Jr. *The "balanced force" concept for instrumentation of curved canals*, J Endod 1985;11: 5.
363. Rollison S, Barnett F, Stevens RH. *Efficacy of bacterial removal from instrumented root canals in vitro related to instrumentation technique and size*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 3, 2002;94: 366.
364. Ruparel NB, Teixeira FB, Ferraz CC, et al: *Direct effect of intracanal medicaments on survival of stem cells of the apical papilla*, J Endod 2012;38: 10.
365. Rzhanov EA, Belyeva TS. *Design features of rotary root canal instruments*, ENDO (Lond) 2012;6: 29.
366. Sabins RA, Johnson JD, Hellstein JW. *A comparison of the cleaning ability of short-term sonic and ultrasonic passive irrigation after hand instrumentation in molar root canals*, J Endod 2003;29: 10.
367. Safavi E, Spångberg LS, Langeland K. *Root canal dentinal tubule disinfection*, J Endod 1990;16: 5.
368. Safavi KE, Nichols FC. *Effect of calcium hydroxide on bacterial lipopolysaccharide*, J Endod 1993;19: 2.
369. Salehrabi R, Rotstein I. *Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study*, J Endod 2004;30: 12.
370. Salzgeber RM, Brilliant JD. *An in vivo evaluation of the penetration of an irrigating solution in root canals*, J Endod 10, 1977;3: 394.
371. Sanghvi Z, Mistry K. *Design features of rotary instruments in endodontics*, J Ahmedabad Dental Coll Hosp 2011;2:.

Telegram: @dental_k

www.konkur.in

372. Sarkar NK, Redmond W, Schwaninger B, et al: *The chloride corrosion behaviour of four orthodontic wires*, J Oral Rehab 1983;10: 2.
373. Sathorn C, Parashos P, Messer HH. *Effectiveness of single- versus multiple-visit endodontic treatment of teeth with apical periodontitis: a systematic review and meta-analysis*, Int Endod J 2005;38: 6.
374. Sato I, Ando-Kurihara N, Kota K, et al: *Sterilization of infected root-canal dentine by topical application of a mixture of ciprofloxacin, metronidazole and minocycline in situ*, Int Endod J 1996;29: 2.
375. Sattapan B, Nervo GJ, Palamara JE, et al: *Defects in rotary nickel-titanium files after clinical use*, J Endod 2000;26: 3.
376. Sattapan B, Palamara JE, Messer HH. *Torque during canal instrumentation using rotary nickel-titanium files*, J Endod 2000;26: 3.
377. Saunders WP, Saunders EM. *Comparison of three instruments in the preparation of the curved root canal using the modified double-flared technique*, J Endod 1994;20: 9.
378. Saunders WP, Saunders EM. *Effect of noncutting tipped instruments on the quality of root canal preparation using a modified double-flared technique*, J Endod 1992;18: 1.
379. Schaeffer MA, White RR, Walton RE. *Determining the optimal obturation length: a meta-analysis of literature*, J Endod 2005;31: 4.
380. Schäfer E. *Effects of four instrumentation techniques on curved canals: a comparison study*, J Endod 1996;22: 12.
381. Schäfer E. *Root canal instruments for manual use: a review*, Endod Dent Traumatol 1997;13: 2.
382. Schäfer E, Diey C, Hoppe W, et al: *Roentgenographic*

Telegram: @dental_k

www.konkur.in

investigation of frequency and degree of canal curvatures in human permanent teeth, J Endod 2002;28: 3.

383. Schäfer E, Tepel J. *Cutting efficiency of Hedstrom, S and U files made of various alloys in filing motion*, Int Endod J 1996;29: 302.
384. Schäfer E, Vlassis M. *Comparative investigation of two rotary nickel-titanium instruments: ProTaper versus RaCe, Part 1: Shaping ability in simulated curved canals*, Int Endod J 2004;37: 4.
385. Schäfer E, Vlassis M. *Comparative investigation of two rotary nickel-titanium instruments: ProTaper versus RaCe, Part 2. Cleaning effectiveness and shaping ability in severely curved root canals of extracted teeth*, Int Endod J 2004;37: 4.
386. Schilder H. *Cleaning and shaping the root canal*, Dent Clin North Am 1974;18: 2.
387. Schrader C, Ackermann M, Barbakow F. *Step-by-step description of a rotary root canal preparation technique*, Int Endod J 1999;32: 4.
388. Schrader C, Peters OA. *Analysis of torque and force during step-back with differently tapered rotary endodontic instruments in vitro*, J Endod 2005;31: 2.
389. Schrader C, Sener B, Barbakow F. *Evaluating the sizes of lightspeed instruments*, Int Endod J 1998;31: 4.
390. Schroeder A. *[Ledermix 1962—Ledermix today. Evaluation after 13 years of experience]*, Zahnarztl Prax 1975;26: 9.
391. Scully C, Ng YL, Gulabivala K. *Systemic complications due to endodontic manipulations*, Endod Topics 2003;4: 1.
392. Senia ES, Marshall FJ, Rosen S. *The solvent action of sodium hypochlorite on pulp tissue of extracted teeth*, Oral Surg Oral Med Oral Pathol 1971;31: 1.

Telegram: @dental_k

www.konkur.in

393. Serene TP, Adams JD, Saxena A. Nickel-Titanium instruments: applications in Endodontics 1995; Ishiaku EuroAmerica St. Louis.
394. Serper A, Calt S, Dogan AL, et al: *Comparison of the cytotoxic effects and smear layer removing capacity of oxidative potential water, NaOCl and EDTA*, J Oral Sci 2001;43: 4.
395. Seto BG, Nicholls JI, Harrington GW. *Torsional properties of twisted and machined endodontic files*, J Endod 1990;16: 8.
396. Shabahang S, Pouresmail M, Torabinejad M. *In vitro antimicrobial efficacy of MTAD and sodium hypochlorite*, J Endod 2003;29: 7.
397. Shabahang S, Torabinejad M. *Effect of MTAD on enterococcus faecalis-contaminated root canals of extracted human teeth*, J Endod 2003;29: 9.
398. Shen Y, Cheung GS, Bian Z, et al: *Comparison of defects in ProFile and ProTaper systems after clinical use*, J Endod 2006;32: 1.
399. Shen Y, Zhou HM, Zheng YF, et al: *Current challenges and concepts of the thermomechanical treatment of nickel-titanium instruments*, J Endod 2013;39: 163.
400. Shrestha A, Kishen A. *The effect of tissue inhibitors on the antibacterial activity of chitosan nanoparticles and photodynamic therapy*, J Endod 2012;38:.
401. Shrestha A, Hamblin MR, Kishen A. *Photoactivated rose bengal functionalized chitosan nanoparticles produce antibacterial/biofilm activity and stabilize dentin-collagen*, Nanomed 2014;10: 3.
402. Shrestha A, Kishen A. *Antibacterial nanoparticles in endodontics: a review*, J Endod 2016;42: 10.
403. Shuping GB, Ørstavik D, Sigurdsson A, et al: *Reduction*

Telegram: @dental_k

www.konkur.in

of intracanal bacteria using nickel-titanium rotary instrumentation and various medications, J Endod 2000;26: 12.

404. Sigurdsson A, Garland RW, Le KT, et al: *Healing of periapical lesions after endodontic treatment with the GentleWave procedure: A prospective multicenter clinical study*, J Endod 2018;44: 3.
405. Sigurdsson A, Garland RW, Le KT, et al: *12-month healing rates after endodontic therapy using the novel GentleWave system: A prospective multicenter clinical study*, J Endod 2016;42: 7.
406. Sigurdsson A, Le KT, Woo SM, et al: *Six-month healing success rates after endodontic treatment using the novel GentleWave System: The pure prospective multi-center clinical study*, J Clin Exp Dent 2016;8: 3.
407. Silvaggio J, Hicks ML. *Effect of heat sterilization on the torsional properties of rotary nickel-titanium endodontic files*, J Endod 1997;23: 12.
408. Sipert CR, Hussne RP, Nishiyama CK, et al: *In vitro antimicrobial activity of Fill Canal, Sealapex, Mineral Trioxide Aggregate, Portland cement and EndoRez*, Int Endod J 2005;38: 8.
409. Siqueira JF Jr. *Aetiology of root canal treatment failure: why well-treated teeth can fail*, Int Endod J 2001;34: 1.
410. Siqueira JF Jr, Batista MM, Fraga RC, et al: *Antibacterial effects of endodontic irrigants on black-pigmented gram-negative anaerobes and facultative bacteria*, J Endod 1998;24: 6.
411. Siqueira JF Jr, de Uzeda M. *Intracanal medicaments: evaluation of the antibacterial effects of chlorhexidine, metronidazole, and calcium hydroxide associated with three vehicles*, J Endod 1997;23: 3.

Telegram: @dental_k

www.konkur.in

412. Siqueira JF Jr, Paiva SS, Rocas IN. *Reduction in the cultivable bacterial populations in infected root canals by a chlorhexidine-based antimicrobial protocol*, J Endod 2007;33: 5.
413. Siqueira JF Jr, Rocas IN, Favieri A, et al: *Chemomechanical reduction of the bacterial population in the root canal after instrumentation and irrigation with 1%, 2.5%, and 5.25% sodium hypochlorite*, J Endod 2000;26: 6.
414. Siqueira JF Jr, Rocas IN, Paiva SS, et al: *Bacteriologic investigation of the effects of sodium hypochlorite and chlorhexidine during the endodontic treatment of teeth with apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 1.
415. Siqueira JFJ, Alves FR, Almeida BM, et al: *Ability of chemomechanical preparation with either rotary instruments or self-adjusting file to disinfect oval-shaped root canals*, J Endod 2010;36: 11.
416. Siren EK, Haapasalo MPP, Waltimo TMT, et al: *In vitro antibacterial effect of calcium hydroxide combined with chlorhexidine or iodine potassium iodide on enterococcus faecalis*, Eur J Oral Sci 2004;112: 4.
417. Siren EK, Lavonious E, Kontakiotis E. *Effects of Ca(OH)2 gutta-percha points on bacteria in root canals.*, J Dent Res 2000;543: 79.
418. Sirtes G, Waltimo T, Schaetzle M, et al: *The effects of temperature on sodium hypochlorite short-term stability, pulp dissolution capacity, and antimicrobial efficacy*, Int Endod J 2005;31: 9.
419. Sjögren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int

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www.konkur.in

Endod J 1997;30: 5.
420. Sjögren U, Figdor D, Spångberg L, et al: *The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing*, Int Endod J 1991;24: 3.
421. Soares JA, Leonardo MR, da Silva LA, et al: *Effect of rotary instrumentation and of the association of calcium hydroxide and chlorhexidine on the antisepsis of the root canal system in dogs*, Braz Oral Res 2006;20: 2.
422. Solovyeva AM, Dummer PM. *Cleaning effectiveness of root canal irrigation with electrochemically activated anolyte and catholyte solutions: a pilot study*, Int Endod J 2000;33: 6.
423. Sonntag D. *Schneidengeometrie und Efficienz vollrotierender Nickel-Titan-Feilen (in German)*, Endodontie 2003;12:.
424. Sonntag D, Peters OA. *Effect of prion decontamination protocols on nickel-titanium rotary surfaces*, J Endod 2007;33: 4.
425. Southard DW, Oswald RJ, Natkin E. *Instrumentation of curved molar root canals with the Roane technique*, J Endod 1987;13: 10.
426. Southard SR, Drisko CL, Killoy WJ, et al: *The effect of 2% chlorhexidine digluconate irrigation on clinical parameters and the level of bacteroides gingivalis in periodontal pockets*, J Periodontol 1989;60: 6.
427. Souza MA, Menon CZ, Nery LF, et al: *Effect of root canal preparation techniques on chlorhexidine substantivity on human dentin: a chemical analysis*, Clin Oral Investig 2018;22: 2.
428. Spanaki-Voreadi AP, Kerezoudis NP, Zinelis S. *Failure mechanism of ProTaper Ni-Ti rotary instruments during clinical use: fractographic analysis*, Int Endod J 2006;39:

Telegram: @dental_k

www.konkur.in

3.
429. Spångberg L. *Instruments, materials, and devices*, Cohen S Burns RC Pathways of the pulp 1998; Mosby St. Louis, MO 476-531.
430. Spångberg L, Engström B, Langeland K. *Biologic effects of dental materials. 3. Toxicity and antimicrobial effect of endodontic antiseptics in vitro*, Oral Surg Oral Med Oral Pathol 1973;36: 6.
431. Spångberg L, Rutberg M, Rydinge E. *Biologic effects of endodontic antimicrobial agents*, J Endod 1979;5: 6.
432. Spångberg LS, Barbosa SV, Lavigne GD. *AH 26 releases formaldehyde*, J Endod 1993;19: 12.
433. Spencer NCO, Sunday JJ, Georgina OKEO, et al: *Comparative stabilizing effects of some anticoagulants on fasting blood glucose of diabetics and non-diabetics, determined by spectrophotometry (Glucose Oxidase)*, Asian J Med Sc 2011;3:.
434. Spili P, Parashos P, Messer HH. *The impact of instrument fracture on outcome of endodontic treatment*, J Endod 2005;31: 12.
435. Stabholz A, Rotstein I, Torabinejad M. *Effect of preflaring on tactile detection of the apical constriction*, J Endod 2, 1995;21: 92.
436. Stenman E, Spångberg LS. *Machining efficiency of Flex-R, K-Flex, Trio-Cut, and S Files*, J Endod 1990;16: 12.
437. Stenman E, Spångberg LS. *Root canal instruments are poorly standardized*, J Endod 1993;19: 7.
438. Stojic S, Shen Y, Qian W, et al: *Antibacterial and smear layer removal ability of a novel irrigant, QMiX*, J Endod 2012;45: 4.
439. Sunada I. *New method for measuring the length of the root canal*, J Dent Res 1962;41: 375.

Telegram: @dental_k

www.konkur.in

440. Suter B, Lussi A, Sequiera P. *Probablity of removing fratured instruments from root canals*, Int Endod J 2005;38: 2.
441. Svec TA, Powers JM. *Effects of simulated clinical conditions on nickel-titanium rotary files*, J Endod 1999;25: 11.
442. Tan BT, Messer HH. *The quality of apical canal preparation using hand and rotary instruments with specific criteria for enlargement based on initial apical file size*, J Endod 2002;28: 9.
443. Tanomaru Filho M, Leonardo MR, da Silva LA. *Effect of irrigating solution and calcium hydroxide root canal dressing on the repair of apical and periapical tissues of teeth with periapical lesion*, J Endod 2002;28: 4.
444. Tay FR, Hosoya Y, Loushine RJ, et al: *Ultrastructure of intraradicular dentin after irrigation with BioPure MTAD. II. The consequence of obturation with an epoxy resin-based sealer*, J Endod 2006;32: 5.
445. Tay FR, Pashley DH, Loushine RJ, et al: *Ultrastructure of smear layer-covered intraradicular dentin after irrigation with BioPure MTAD*, J Endod 2006;32: 3.
446. Tepel J. Experimentelle Untersuchungen über die maschinelle Wurzelkanalaufbereitung 2000; Quintessenz Verlags-GmbH Berlin, Germany.
447. Testarelli L, Plotino G, Al-Sudani D, et al: *Bending properties of a new nickel-titanium alloy with a lower percent by weight of nickel*, J Endod 2011;37: 9.
448. Thibodeau B, Teixeira F, Yamauchi M, et al: *Pulp revascularization of immature dog teeth with apical periodontitis*, J Endod 2007;33: 6.
449. Thompson SA. *An overview of nickel-titanium alloys used in dentistry*, Int Endod J 2000;33: 4.

Telegram: @dental_k

www.konkur.in

450. Thompson SA, Dummer PM. *Shaping ability of lightspeed rotary nickel-titanium instruments in simulated root canals. Part 1*, J Endod 1997;23: 11.
451. Thompson SA, Dummer PM. *Shaping ability of ProFile.04 Taper Series 29 rotary nickel-titanium instruments in simulated root canals. Part 1*, Int Endod J 1997;30: 1.
452. Thompson SA, Dummer PM. *Shaping ability of ProFile.04 Taper Series 29 rotary nickel-titanium instruments in simulated root canals. Part 2*, Int Endod J 1997;30: 1.
453. Torabinejad M. *Passive step-back technique. A sequential use of ultrasonic and hand instruments*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1994;77: 4.
454. Torabinejad M, Johnson WB: *Irrigation solution and methods for use*, 2003.
455. Torabinejad M, Shabahang S, Aprecio RM, et al: *The antimicrobial effect of MTAD: an in vitro investigation*, J Endod 2003;29: 6.
456. Torabinejad M, Walton R. Principles and practice of endodontics ed 4 2008; Saunders St. Louis.
457. Tripi TR, Bonaccorso A, Condorelli GG. *Cyclic fatigue of different nickel-titanium endodontic rotary instruments*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: 4.
458. Troian CH, So MV, Figuereido JA, et al: *Deformation and fracture of RaCe and K3 endodontic instruments according to the number of uses*, Int Endod J 2006;39: 8.
459. Trope M. *The vital tooth- its importance in the study and practice of endodontics*, Endod Topics 2003;5: 1.
460. Turpin YL, Chagneau F Bartier, et al: *Impact of torsional and bending inertia on root canal instruments*, J Endod

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www.konkur.in

2001;27: 5.
461. Turpin YL, Chagneau F, Vulcain JM. *Impact of two theoretical cross-sections on torsional and bending stresses of nickel-titanium root canal instrument models*, J Endod 2000;26: 7.
462. Ullmann CJ, Peters OA. *Effect of cyclic fatigue on static fracture loads in ProTaper nickel-titanium rotary instruments*, J Endod 2005;31: 3.
463. Ungerechts C, Bårdsen A, Fristad I. *Instrument fracture in root canals - where, why, when and what? A study from a student clinic*, Int Endod J 2014;47: 2.
464. Usman N, Baumgartner JC, Marshall JG. *Influence of instrument size on root canal debridement*, J Endod 2004;30: 2.
465. van der Sluis LW, Versluis M, Wesselink PR. *Passive ultrasonic irrigation of the root canal: a review of the literature*, Int Endod J 6, 2007;40: 415.
466. van der Sluis LW, Wu MK, Wesselink PR. *A comparison between a smooth wire and a K-file in removing artificially placed dentine debris from root canals in resin blocks during ultrasonic irrigation*, Int Endod J 2005;38: 9.
467. van der Sluis LW, Wu MK, Wesselink PR. *The efficacy of ultrasonic irrigation to remove artificially placed dentine debris from human root canals prepared using instruments of varying taper*, Int Endod J 2005;38: 10.
468. Verma N, Sangwan P, Tewari S, et al: *Effect of different concentrations of sodium hypochlorite on outcome of rimary root canal treatment: a randomized controlled trial*, J Endod 2019;45: 4.
469. Versiani MA, Leoni GB, Steier L, et al: *Micro-computed tomography study of oval-shaped canals prepared with the*

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www.konkur.in

self-adjusting file, Reciproc, WaveOne, and ProTaper universal systems, J Endod 2013;39:.

470. Viana AC, Gonzales BM, Buono VT, et al: *Influence of sterilization on mechanical properties and fatigue resistance of nickel-titanium rotary endodontic instruments*, Int Endod J 2006;39:.
471. Vianna ME, Gomes BP, Berber VB, et al: *In vitro evaluation of the antimicrobial activity of chlorhexidine and sodium hypochlorite*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 1.
472. Vianna ME, Horz HP, Gomes BP, et al: *In vivo evaluation of microbial reduction after chemo-mechanical preparation of human root canals containing necrotic pulp tissue*, Int Endod J 2006;39: 6.
473. Vier FV, Figuereido JA. *Prevalence of different periapical lesions associated with human teeth and their correlation with the presence and extension of apical external root resorption*, Int Endod J 2002;35:.
474. Walia HM, Brantley WA, Gerstein H. *An initial investigation of the bending and torsional properties of nitinol root canal files*, J Endod 1988;14: 7.
475. Walmsley AD. *Ultrasound and root canal treatment: the need for scientific evaluation*, Int Endod J 1987;20: 3.
476. Walsch H. *The hybrid concept of NiTi rotary instrumentation*, Dent Clin North Am 2004;48: 1.
477. Waltimo T, Trope M, Haapasalo M, et al: *Clinical efficacy of treatment procedures in endodontic infection control and one year follow-up of periapical healing*, J Endod 2005;31: 12.
478. Waltimo TM, Ørstavik D, Siren EK, et al: *In vitro susceptibility of Candida albicans to four disinfectants and their combinations*, Int Endod J 1999;32: 6.

Telegram: @dental_k

www.konkur.in

479. Wang R, Shen Y, Ma J, et al: *Evaluation of the effect of needle position on irrigant flow in the C-shaped root canal using a computational fluid dynamics model*, J Endod 2015;41: 6.
480. Wang Z, Shen Y, Haapasalo M. *Effectiveness of endodontic disinfecting solutions against young and old enterococcus faecalis biofilms in dentin canals*, J Endod 10, 2012;38: 1376.
481. Ward JR, Parashos P, Messer HH. *Evaluation of an ultrasonic technique to remove fractured rotary nickel-titanium endodontic instruments from root canals: clinical cases*, J Endod 2003;29: 11.
482. Weiger R, El Ayouti A, Löst C. *Efficiency of hand and rotary instruments in shaping oval root canals*, J Endod 2002;28: 8.
483. Weine FS, Healey HJ, Gerstein H, et al: *Pre-curved files and incremental instrumentation for root canal enlargement*, J Can Dent Assoc 1970;36: 4.
484. Weine FS, Kelly RF, Lio PJ. *The effect of preparation procedures on original canal shape and on apical foramen shape*, J Endod 8, 1975;1: 255-262.
485. Weller RN, Brady JM, Bernier WE. *Efficacy of ultrasonic cleaning*, J Endod 1980;6: 9.
486. West JD, Roane JB. *Cleaning and shaping the root canal system*, Cohen S Burns RC Pathways of the pulp 1998; Mosby St. Louis, MO 203-257.
487. Wilson BL, Broberg C, Baumgardner JC, et al: *Safety of electronic apex locators and pulp testers in patients with implanted cardiac pacemakers or cardioverter/defibrillators*, J Endod 2006;32: 847.
488. Windley W 3rd, Teixeira F, Levin L, et al: *Disinfection of immature teeth with a triple antibiotic paste*, J Endod

Telegram: @dental_k

www.konkur.in

2005;31: 6.

489. Wolcott S, Wolcott J, Ishley D, et al: *Separation incidence of ProTaper rotary instruments: a large cohort clinical evaluation*, J Endod 2006;32: 12.

490. Wu MK, Dummer PM, Wesselink PR. *Consequences of and strategies to deal with residual post-treatment root canal infection*, Int Endod J 2006;39: 5.

491. Wu MK, van der Sluis LW, Wesselink PR. *The capability of two hand instrumentation techniques to remove the inner layer of dentine in oval canals*, Int Endod J 3, 2003;36: 218.

492. Wu MK, Wesselink PR, Walton RE. *Apical terminus location of root canal treatment procedures*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 1.

493. Yamamura B, Cox TC, Heddaya B, et al: *Comparing canal transportation and centering ability of endosequence and vortex rotary files by using micro-computed tomography*, J Endod 2012;38: 1121.

494. Yamashita JC, Tanomaru Filho M, Leonardo MR, et al: *Scanning electron microscopic study of the cleaning ability of chlorhexidine as a root-canal irrigant*, Int Endod J 2003;36: 6.

495. Yao JH, Schwartz SA, Beeson TJ. *Cyclic fatigue of three types of rotary nickel-titanium files in a dynamic model*, J Endod 2006;32: 1.

496. Yared G. *Canal preparation using only one Ni-Ti rotary instrument: preliminary observations*, Int Endod J 2008;41: 4.

497. Yared GM, Bou Dagher FE, Machtou P. *Cyclic fatigue of profile rotary instruments after clinical use*, Int Endod J 2000;33: 3.

498. Yared GM, Bou Dagher FE, Machtou P. *Cyclic fatigue of*

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www.konkur.in

profile rotary instruments after simulated clinical use, Int Endod J 1999;32: 2.

499. Yared GM, Bou Dagher FE, Machtou P. *Failure of ProFile instruments used with high and low torque motors*, Int Endod J 2001;34: 6.
500. Yared GM, Dagher FE. *Influence of apical enlargement on bacterial infection during treatment of apical periodontitis*, J Endod 1994;20: 11.
501. Yguel-Henry S, Vannesson H, von Stebut J. *High precision, simulated cutting efficiency measurement of endodontic root canal instruments: influence of file configuration and lubrication*, J Endod 1990;16: 9.
502. Yun HH, Kim SK. *A comparison of the shaping abilities of 4 nickel-titanium rotary instruments in simulated root canals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 2.
503. Zamany A, Safavi K, Spångberg LS. *The effect of chlorhexidine as an endodontic disinfectant*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 5.
504. Zehnder M. *Root canal irrigants*, J Endod 2006;32: 5.
505. Zehnder M, Paqué F. *Disinfection of the root canal system during root canal re-treatment*, Endod Topics 2008;19: 58-73.
506. Zehnder M, Schmidlin PR, Sener B, et al: *Chelation in root canal therapy reconsidered*, J Endod 2005;31: 11.
507. Zehnder M, Söderling E, Salonen J, et al: *Preliminary evaluation of bioactive glass S53P4 as an endodontic medication in vitro*, J Endod 2004;30: 4.
508. Zeltner M, Peters OA, Paqué F. *Temperature changes during ultrasonic irrigation with different inserts and modes of activation*, J Endod 2009;35: 4.
509. Zerella JA, Fouad AF, Spångberg LS. *Effectiveness of a*

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www.konkur.in

calcium hydroxide and chlorhexidine digluconate mixture as disinfectant during retreatment of failed endodontic cases, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 6.

510. Zhu WC, Gyamfi J, Niu LN, et al: *Anatomy of sodium hypochlorite accidents involving facial ecchymosis - a review*, J Dent 2013;41: 11.
511. Zinelis S, Magnissalis EA, Margelos J, et al: *Clinical relevance of standardization of endodontic files dimensions according to the ISO 3630-1 specification*, J Endod 2002;28: 5.
512. Zmener O, Banegas G. *Comparison of three instrumentation techniques in the preparation of simulated curved root canals*, In Endod J 1996;29: 5.

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9: Obturation of the cleaned and shaped root canal system

Anita Aminoshariae, William T. Johnson, James C. Kulild, Franklin Tay

CHAPTER OUTLINE

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Importance of effectively sealing the root canal system

The authors of a meta-analysis of factors influencing the efficacy of nonsurgical root canal treatment (NS RCT) reported that the following four factors influenced success: the absence of a periapical lesion (PL), root canal obturations containing no radiographic voids, obturation to within 2.0 mm of the radiographic apex, and an adequate coronal restoration.[196] On the basis of the current best available evidence, both the quality of the endodontic treatment and the coronal restoration are significant in any healing outcome.[104]

In a recent controlled animal study, evidence suggests that cleaning and shaping (C&S) of the radicular space is more important than the method of obturation.[240] PLs were created by removing the pulp and leaving the teeth open to the oral cavity. In the control group, the canals were cleaned and shaped before obturation with gutta-percha (GP) and a resin-based sealer. The teeth of the experimental group were cleaned and shaped as in the control group but left unobturated. At 190 days, the animals were killed and histological evaluations were performed. There was no difference in the healing between the instrumented and obturated teeth and the instrumented and unobturated teeth.

To date, there is no effective method for determining whether C&S procedures have been effective.[10,11] There is some evidence that the criteria of clean dentinal filings and/or enlargement beyond the first file to bind at the working length have proven to result in a healing outcome.[244] Although the length of preparation has been emphasized, the irregular canal diameter may be a more significant factor in healing or nonhealing outcomes.[133] Evidence indicates root canal systems (RCS) are often underprepared in the apical one third of the RCS.[56] Historically, culturing has been employed and obturation delayed until a “negative” culture was obtained. In contemporary endodontic treatment, culturing has been abandoned during routine care.[250] When dealing with vital pulp tissue, preventing bacterial contamination is a primary concern. In RCS with a necrotic pulp, the organisms involved in the disease

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process are primarily facultative or obligate anaerobes that may not grow in culture. Molecular microbiologic techniques (polymerase chain reaction) have demonstrated that a variety of organisms are present that do not grow in culture.[13,237,273] The role these organisms play in the disease process is not well understood.[189] The reader is referred to Chapters 15 and 16 for a fuller discussion.

The process of C&S determines both the degree of disinfection and the ability to obturate the radicular space. Obturation is therefore a reflection of the C&S and is evaluated on the basis of length, taper, density, level of GP obturation, and the coronal seal, that is, an adequate provisional or definitive restoration (Fig. 9.1). It is not possible to assess the quality of the seal established during obturation with a radiograph, and it is important to recognize that no material or technique prevents leakage.[4,58,81,139,182,209,210] Indeed, obtaining an impervious seal may not be feasible because of the porous tubular structure of dentin[4] and canal irregularities.

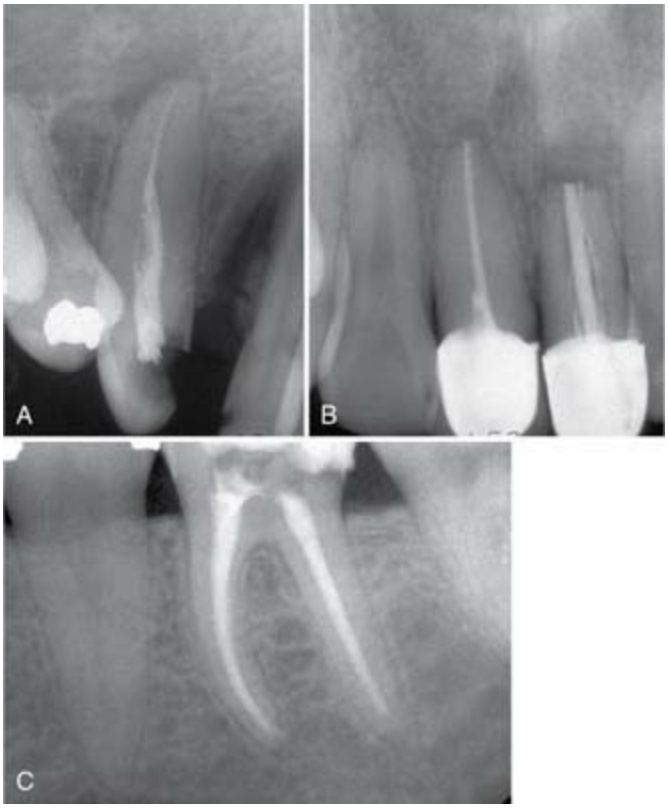

FIG. 9.1 Examples of inadequate obturation. **A,** Maxillary right canine with adequate length but lacking density and no coronal seal. Central

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incisor is filled to adequate length, but obturation exhibits voids. **B,** Maxillary central incisors. Maxillary right central incisor exhibits a lack of density and taper. Maxillary left central incisor has voids and unfilled canal space. **C,** Mandibular left first molar with adequate obturation; provisional restoration shows poor adaptation on the distal because of the failure to remove caries.

A) Radiograph of a tooth shows a missing part of crown, radiopaque neck and canals.

B) Radiograph of teeth shows two radiopaque crowns with radiolucent canals.

C) Radiograph of a tooth shows a radiolucent neck and radiopaque canals.

The primary etiology of pulpal and periradicular pathosis is, as discussed in Chapter 16, microbiological.[141,181,186,239] Pulpal remnants, necrotic tissues, bacteria, and bacterial by-products remaining in the inaccessible areas of a C&S RCS can initiate and/or perpetuate a PL because the host defense mechanisms are unable to remove them. Studies indicate that an RCS cannot be completely cleaned and disinfected.[10,11,122] Obturation of the radicular space is necessary to minimize coronal leakage from the oral environment. Obturation reduces coronal leakage and bacterial contamination, seals the apex from the periapical tissue fluids, and entombs the remaining irritants in the canal.

Coronal leakage has been proposed to contribute to treatment failure based on in vitro leakage studies.[293,294] The clinical implication is that retreatment has to be performed in those teeth that are not restored definitively 3 months after NS RCT. The clinical significance of this issue has been challenged.[232,233] A recent systematic review and meta-analysis of the results derived from nine similar studies indicate that poor-quality NS RCT and poor-quality coronal restorations have similar odds in adversely affecting the healing of apical periodontitis.[104] Based on the best available current evidence, the odds for healing apical periodontitis increase with both adequate NS RCT and an adequate restorative treatment.[104] It must be emphasized that these two factors were examined in isolation in those studies. These postoperative prognostic factors, along with other preoperative and intraoperative prognostic factors, have been reported to contribute significantly to periapical healing in primary and secondary NS RCT.[194] Nevertheless, maintaining an effective coronal seal through optimal

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obturation of the instrumented RCS and placing an appropriate restoration, as discussed in Chapter 23, should be considered essential components of a positive healing outcome for NS RCT.[124,132]

Clinicians are concerned with whether it is more appropriate to place a definitive restorative material instead of a temporary material to prevent leakage.[169,295] This is a controversial issue, as previous studies failed to provide definitive evidence to demonstrate that the presence of a definitive restoration will contribute to a long-term healing outcome for NS RCT when those RCS are optimally obturated.[62,243] These results have been supported by a more recent prospective clinical study that examined the factors affecting the outcomes of NS RCT.[194] In this study, the authors reported that the type of coronal restoration had no significant influence on periapical healing as long as those restorations were of good quality. Paradoxically, the same group of authors reported in a separate study that cast restorations, when compared with temporary restorations, significantly improved the survival of teeth that had undergone primary or secondary NS RCT.[194] This may be due to protection of remaining, weakened tooth structure by full-coverage cast restorations that enabled those teeth to function longer. Regarding the necessity for retreatment of root-filled teeth without definitive restorations, there is no clear evidence to justify that retreatment has to be performed in teeth in which a temporary restoration had been inserted for more than 3 months solely because of the suspicion of microleakage.[5,144] One may consider replacing the temporary restoration with a definitive coronal restoration immediately and to observe the tooth for at least 3 months before placing a full-coverage restoration.[144]

Three-dimensional obturation of the RCS is essential to a long-term healing outcome. The RCS should be sealed apically, coronally, and laterally. Various methods have been advocated for obturation. Unfortunately, all materials and techniques result in some degree of leakage.[314] Although a poorly obturated RCS and leakage are correlated, there are no standardized techniques to evaluate and measure microleakage.[134] Also, radiographic evaluation of obturation does not correlate well with leakage.[119] An adequate two-dimensional radiographic appearance of the obturation may not correlate with an adequate seal (Fig. 9.2).[119] Variation in radiographic interpretation by the clinician, the overlying osseous structures, and the lack of uniformity in

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the obturation materials are significant variables.[83,84,146,290]

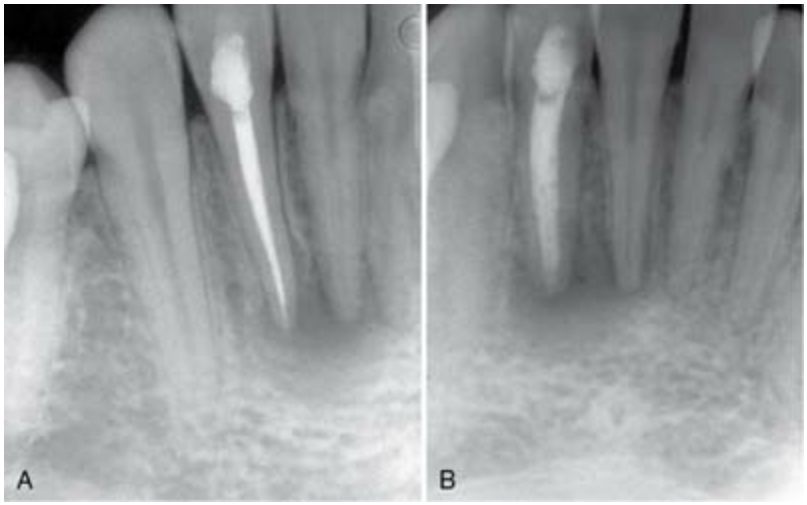

FIG. 9.2 **A,** Posttreatment radiograph of a mandibular left lateral incisor with ostensibly adequate obturation. **B,** Angled view reveals voids.

A) Radiograph of a tooth shows a radiopaque crown, radiopaque neck and canals.

B) Radiograph of a tooth shows a radiopaque crown, radiolucent neck and radiopaque canals.

The diagnostic outcome of NS RCT is based on clinical and radiographic findings. In a series of prospective studies, the Toronto group evaluated the success and failure of NS RCT at 4 to 6 years after completion of treatment. For primary NS RCT,[72,93,97,177] teeth with preexisting apical periodontitis were found to have a lower healing rate (82%) compared with teeth without a periapical radiolucency (93%). Better outcomes were associated with teeth without a periapical radiolucency, with single roots, and without midtreatment complications, such as a root perforation. Teeth were treated by using a flared preparation and vertical compaction of warm GP or step-back preparation and lateral compaction of GP. Differences were noted with the adequacy of the obturation and the treatment technique. Adequate length had a higher healing rate (87%) when compared with inadequate length (77%). The flared preparation and vertical compaction had a higher success rate (90%) when compared with a step-back preparation and lateral compaction

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(80%). For retreatment NS RCT,[73,92] teeth with preexisting apical periodontitis were also found to have a lower healing rate (80%) compared with teeth without a periapical radiolucency (93%). Better outcomes were achieved in teeth with an inadequate previous obturation, without perforation and a radiolucency. Similar results were reported in a more recent prospective clinical study that examined the effects of primary or secondary NS RCT on periapical healing.[194] The percentage of roots with complete periapical healing after primary (83%) or retreatment (80%) NS RCT was similar. Absence of preoperative apical periodontitis was identified as one of the prognostic factors affecting primary or secondary NS RCT. In the presence of apical periodontitis, treatment prognosis was significantly improved by the presence of a smaller PL. These studies agree with earlier work[275,282] indicating preexisting apical pathosis as a major factor reducing a favorable prognosis and highlighted obturation technique as a factor influencing healing or nonhealing outcomes.

Although periapical radiographs have been used to examine the healing of posttreatment apical periodontitis since 1922,[195] their suitability for evaluating the outcomes of NS RCT has recently been challenged, in that previously reported outcomes based on this assessment modality may have overestimated the healing rates of apical periodontitis.[12,313] With the advent of cone-beam computed tomography (CBCT), a better diagnosis of PL within the cancellous bone can be made. This is because CBCT creates reconstructed images from slices of data in any plane and location of the volume of interest, thus eliminating the lack of three-dimensional assessment and anatomical noise, which hampers the accuracy of periapical radiography. This results in a higher signal-to-noise ratio and image contrast, and improves the detection of periapical radiolucencies.[217,253] Although intraoral radiographs are the imaging modality of choice, when two-dimensional intraoral radiography is inconclusive, the use of CBCT imaging was reported to have twice the odds of detecting periapical pathosis than traditional periapical radiography.[12] Though different expectations in endodontic outcomes provide insight into the definition of the success of these procedures, it remains questionable whether this gain of additional information with CBCT in endodontic outcomes would lead to a better healing outcome in endodontic treatment. The results of these studies

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emphasized that developing good technical skills for obturating an RCS should not be underscored and that suitable canal obturation methods should be developed to achieve more compacted, void-free filling materials and at the correct length.

Historical perspectives

Over the past 70 to 80 years, the dental community has seen attempts to improve the nature of root canal obturation with cements/sealers and with variations in the delivery of GP to the prepared RCS. During this era, the impetus for these developments was based heavily on the continued belief in the concept of focal infection, elective localization, the hollow-tube theory, and the concept that the primary cause for failure of NS RCT was the apical percolation of fluids, and microorganisms into a poorly obturated RCS.[81] Despite endeavors reporting an adequate RCS obturation radiographically, a "perfect" seal is not achievable, as most current materials have no antimicrobial activity and do not seal the RCS.[47] From this chronological perspective of technical and scientific thought, this chapter clarifies and codifies contemporary concepts in the obturation of the C&S RCS.

Timing of obturation

Factors influencing the appropriate time to obturate an RCS include the patient's signs and symptoms, status of the pulp and periradicular tissues, the degree of difficulty, and patient management.

Vital pulp tissue

At present, the consensus is that one-step treatment procedures are acceptable when the patient exhibits a completely or partially vital pulp.[175] Removal of the normal or inflamed pulp tissue and performance of the procedure under aseptic conditions should result in a healing outcome because of the relative absence of bacterial contamination. Obturation at the initial visit also precludes contamination as a result of coronal leakage during the period between patient visits.

Elective NS RCT for restorative reasons can be completed in one visit

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provided the pulp is vital, to some degree, and time permits. Obturation of RCS in patients whose condition is urgent depends on the pretreatment diagnosis. When pain occurs as the result of irreversible pulpitis, obturation can occur at the initial visit because removal of the vital pulp tissue will generally resolve the patient's pain.

Necrotic pulp tissue

Patients who present with pulp necrosis, with or without asymptomatic periradicular pathosis, for example, asymptomatic apical periodontitis, chronic apical abscess, or condensing osteitis, may be treated in one visit based on the best available information. When patients present with acute symptoms caused by pulp necrosis and acute periradicular abscess, obturation is generally delayed until the patient is asymptomatic. However, more than 30 years ago, investigators demonstrated that cases with soft-tissue swelling could be completed in one visit with appropriate endodontic treatment, incision for drainage, and a regimen of antibiotics.[277] Management of these patients, however, may be more difficult should problems persist or become worse after the completion of treatment.

During the 1970s, there was concern about the timing of obturation. Performing endodontic treatment in one visit was controversial. Conventional wisdom suggested that patients would have a higher incidence of posttreatment pain. However, clinical studies[185,213,298] and systematic reviews[175] indicated that there was no significant difference in the healing rates of apical periodontitis between single-visit and multiple-visit NS RCT. Patients experienced less frequency of short-term postobturation pain after single-visit than those receiving multiple-visit NS RCT.[281]

With the introduction of the concept of biofilms, other investigators examined the intracanal microbial status of 16 mesial roots of human mandibular first molars with primary apical periodontitis immediately after one-visit NS RCT.[190] In that study, the instrumented RCS were irrigated with 5.25% NaOCl and 17% ethylenediaminetetraacetic acid (EDTA), and obturated with GP and zinc oxide eugenol sealer. The apical portion of the root of each tooth was surgically removed and prepared for correlative light and transmission electron microscopy examination. Fourteen of the 16 root-treated teeth revealed residual intracanal infection after instrumentation,

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antimicrobial irrigation, and obturation. The microbes existed mostly as biofilms in inaccessible recesses of instrumented main canals, the intercanal isthmus, and accessory/lateral canals.

In a histobacteriological study, the in vivo microbiological status of the mesial roots of mandibular molars with primary apical periodontitis were examined after single-visit or two-visit NS RCT.[298] Those roots were instrumented and irrigated with 5% NaOCl, 17% EDTA, and 2% chlorhexidine. In the single-visit group, the RCS were immediately obturated, while in the two-visit group, calcium hydroxide ($Ca[OH]_2$) dressing was placed in the RCS for 1 week prior to obturation. In the single-visit group, no RCS, out of the six RCS examined, was completely free of bacteria. Residual biofilms were identified in the main RCS, isthmus, apical ramifications, and dentinal tubules. In the two-visit group, two out of seven roots were rendered bacteria-free. Residual biofilms were found predominantly in the isthmus and ramifications, intermixed with necrotic tissues and debris. Taken together, these studies illustrate that microbes are extremely difficult to eliminate within the complex RCS and continued endeavors have been made to improve microbiological status. Current evidence points out that the use of 5% NaOCl and 17% EDTA is efficacious in reducing microbes.[194] When one appointment is not achievable, the use of $Ca(OH)_2$ has been advocated as it remains the best medicament available to reduce residual microbial flora.[157] Calcium hydroxide has been advocated as an antimicrobial and temporary dressing in necrotic cases that cannot be treated in one visit[274] because investigators noted that bacteria in instrumented, unfilled RCS can multiply and reach their pretreatment numbers in 2 to 4 days.[48] Nevertheless, the ability of $Ca(OH)_2$ to completely eradicate microbial species from the RCS has recently been questioned. In vitro studies demonstrated that the antibacterial activity of $Ca(OH)_2$ can be inactivated by dentin.[116] Other clinical studies reported that the number of bacterial positive RCS did not decrease after the use of $Ca(OH)_2$ as an interappointment dressing.[222] A systematic review and meta-analysis of eight clinical studies concluded that $Ca(OH)_2$ is useful but have limited effectiveness in completely eliminating bacteria from human RCS when evaluated by culture techniques.[249] Although $Ca(OH)_2$ has a wide range of antimicrobial activity against common

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endodontic pathogens and is an effective antiendotoxin agent, it is less effective against *Enterococcus faecalis* and *Candida albicans*.[91,184] Alternative interappointment dressings such as the use of chlorhexidine gel, $Ca(OH)_2$ in chlorhexidine gel, or triple antibiotic paste (metronidazole, minocycline, and ciprofloxacin) has been proposed, with dichotomous results.[15,75,203,272,302] Although there are clinical trials to support the use of $Ca(OH)_2$ or alternative interappointment dressings, they do not reduce microbes beyond levels already achieved by canal preparation with NaOCl.[172,176,271] It is fair to say that interappointment antimicrobial dressings are fairly well accepted by clinicians in cases that cannot be treated in a single visit.

In general, obturation can be performed after C&S procedures when the RCS can be dried and the patient is not experiencing significant pain and or swelling. An exception is the presence or persistence of exudation from the canal. Obturation of an RCS that cannot be dried is contraindicated. Procedural concerns also dictate the time of obturation. Difficult cases may require more time for preparation and can be managed more uneventfully in multiple appointments. Patients may require multiple short appointments because of medical conditions, their psychological state of mind, and/or fatigue.

Length of obturation

One of the controversies in endodontics that remains unresolved is the apical limit of NS RCT and obturation.[194,231,255] Early studies identified the dentinocemental junction as the apical limit for obturation.[153] However, this histologic landmark cannot be determined clinically and it has been found to be irregular within the canal. For example, the dentinocemental junction may be several millimeters higher on the mesial canal wall when compared with the distal wall. Additionally, the dentinocemental junction does not coincide with the narrowest portion of the canal or apical constriction. The reader is referred to Chapter 7 for more information about this anatomy.

Traditionally, the apical point of termination has been approximately 1 mm from the radiographic apices as determined by radiographs. Kuttler[153] noted that the apical anatomy consists of the major diameter of the foramen and the

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minor diameter of the constriction (Fig. 9.3), with the apical constriction identified as the narrowest portion of the canal. The average distance from the foramen to the constriction was found to be 0.5 mm, with the foramen varying in distance from the apex up to 2.5 mm. Kuttler also noted that the foramen-to-constriction distance increases with age because of cementum deposition. Supporting this finding, other investigators reported that the location of the foramen was not at the apex. Deviations occurred in 92% of the roots and averaged 0.6 mm.[46] Another study noted the average apex-to-constriction distance was 0.9 mm and that 95% of the constrictions were between 0.5 and 1 mm in diameter.[85] This study also noted that the classic apical anatomy described by Kuttler was present in only 46% of teeth. Other variations identified were a tapering constriction, the multiconstriction, and the parallel constriction. Other investigators examined 230 roots of permanent teeth stereomicroscopically and with radiographs.[31] Results of this study indicated a deviation of the foramen from the apex in 76% of the roots with microscopy and 57% with radiography. The mean distance was 1 mm.

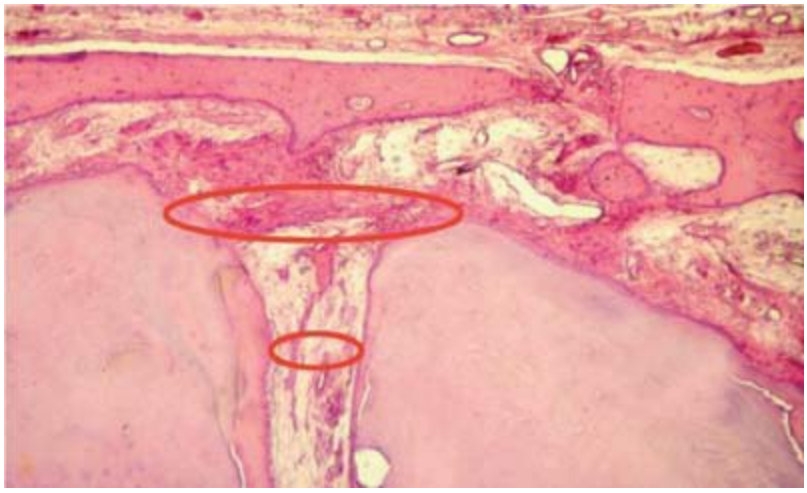

FIG. 9.3 Histologic section of a root apex, demonstrating anatomy of the classic foramen and constriction.

Micrograph of the upper part of root tip shows a hollow bony foramen and narrow constriction in the lower root both encircled in red ellipsoid.

The authors of a scanning electron microscopic study reported that no foramina coincided with the longitudinal axis of the root with the distance

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ranging from 0.2 to 3.8 mm (Fig. 9.4).[114] Another group of authors, using CBCT, reported that the most frequent position of the apical foramen was central but it varied.[90] Root resorption is an additional factor in length determination. Resorption is more common with pulp necrosis and apical bone resorption and this can result in loss of the constriction (Fig. 9.5).[94,173] On the basis of these findings, it appears that an RCS obturated to the radiographic apex reflect an overextension of the obturating material. If overextension occurs that cannot be retrieved, and there appears to be resultant nerve damage, the practitioner is obligated to refer the patient to a qualified practitioner skilled in cases of this type.[105]

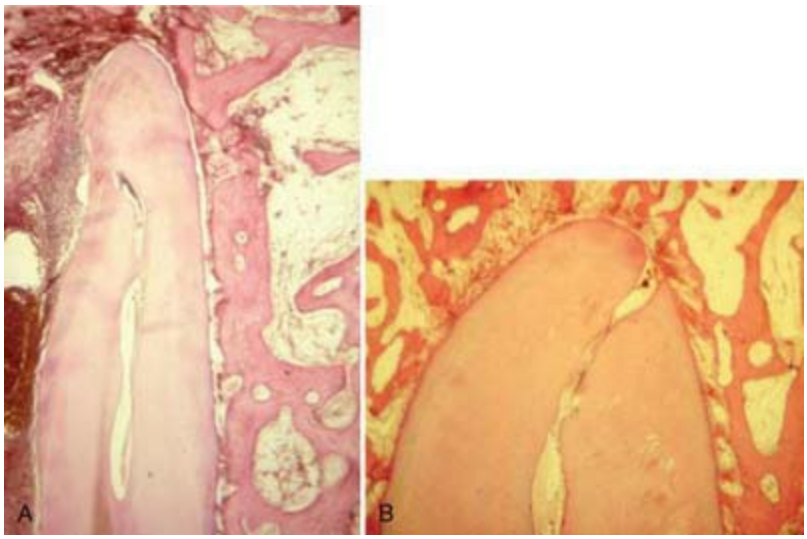

FIG. 9.4 Histologic sections demonstrating the foramen exiting short of the root apex.

A) Micrograph of the cross section of root tip shows a canal with a hollow bony foramen near the root outline.

B) Micrograph of the cross section of root tip shows a canal with a hollow bony foramen exiting the root outline.

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FIG. 9.5 Scanning electron microscopy of a tooth exhibiting a necrotic pulp and apical pathosis and resorption.

Micrograph of a tooth shows a textured surface with two large hollow canals.

In a study by the Toronto group on the prognosis of retreatment identified perforation,[73] pretreatment periradicular disease and adequate length of the root canal filling were identified as factors significantly influencing healing or nonhealing outcomes. The authors speculated that canals obturated more than 2 mm short of the radiographic apex harbored necrotic tissue, bacteria, and irritants that, when retreated, could be cleaned and sealed. The success rate for negotiating the apical unfilled canal was 74%.

Controversy also exists regarding the role that accessory canals play in healing and nonhealing outcomes (Fig. 9.6). A scanning electron microscopy (SEM) study of the apical anatomy of each tooth group except third molars noted no pattern for foraminal openings.[113] The number of accessory canals ranged from 1 to 16. Although lateral canals can be associated with pathosis, one study that examined root-treated teeth from human cadavers reported no relationship between unfilled lateral canals and periradicular pathosis.[21] Accessory/lateral canals are often obturated by chance and only serendipitously identified on a posttreatment radiograph (Fig. 9.7).

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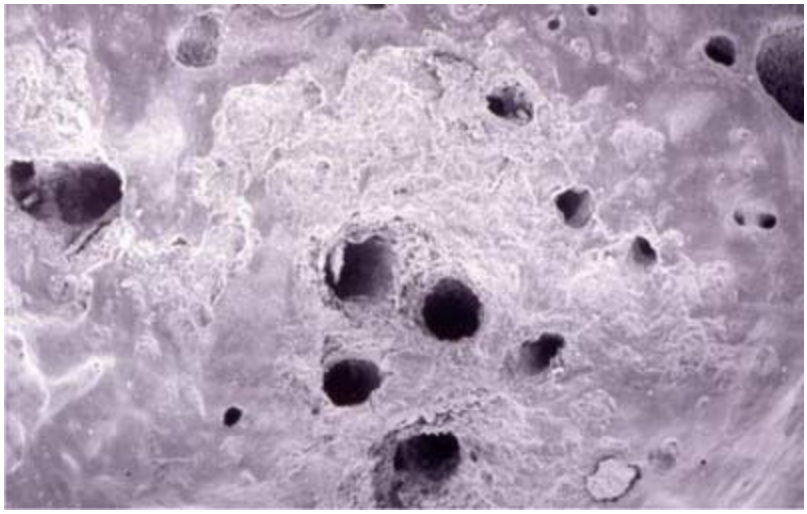

FIG. 9.6 Scanning electron microscopy of the apex of an extracted tooth that was removed because of pulp necrosis. Note the multiple accessory foramina and resorption.

Micrograph of a tooth shows an extremely textured surface with many holes of different sizes.

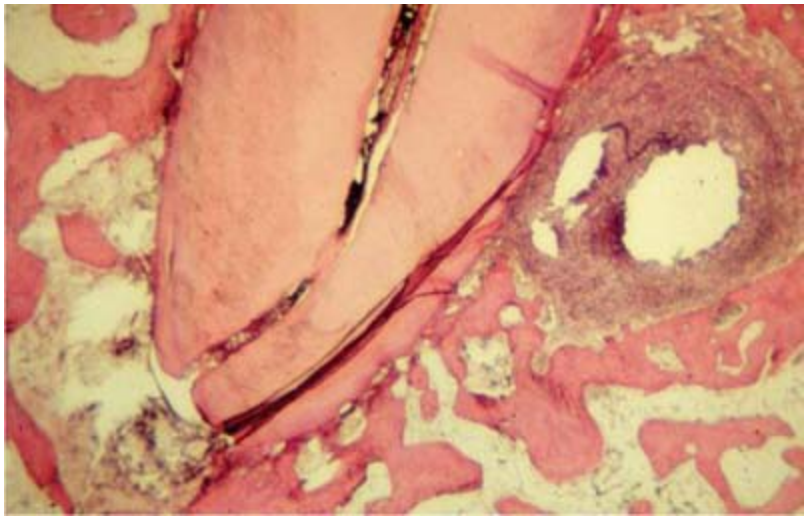

FIG. 9.7 Histologic section of a mesial root of a mandibular molar with a lateral canal present and associated lesion. Will the lesion resolve after the removal of the main canal contents, or will the lesion persist because of necrotic pulpal remnants in the lateral canal? The question remains unanswered.

Micrograph of a tooth with gingival surface shows a canal exiting through the root tip. A yellow patch with an outline of red is visible near the tooth.

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A subsequent histobacteriological study also found no evidence to support the concept that lateral canals must be obturated to achieve a healing long-term treatment outcome (Fig. 9.8).[236] In cases with vital pulps, forcing obturation materials into lateral canals resulted in unnecessary damage to the periradicular tissues with consequential inflammation. In cases with necrotic, infected pulps in which lateral canals appeared radiographically obturated, they were actually not sealed or disinfected. Moreover, the remaining tissue in the ramification was inflamed and enmeshed with obturation materials. This, however, does not mean that lateral canals should not be optimally debrided and disinfected by contemporary irrigant delivery and agitation techniques to reduce microbes and/or biofilms that reside within those spaces prior to obturation of the RCS with obturation materials and sealers.

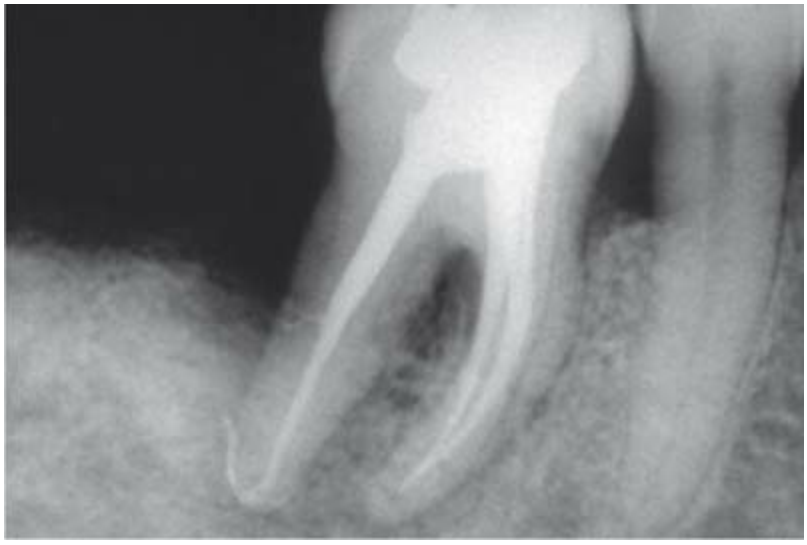

FIG. 9.8 Posttreatment radiograph of a mandibular right first molar with a lateral canal associated with the distal root.

Radiograph of a tooth shows a radiopaque crown, neck, left canal and laterally segmented right canal.

The importance of length control in obturation relates to possible overextension of root filling materials. In a quantitative systematic review of the literature,[255] the authors critically analyzed 12 outcome studies that fulfilled the following criteria: (i) a minimum follow-up period of 2 years; (ii) data available on termination of obturation/instrumentation; (iii) adequate

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definition of treatment failure; (iv) data available on healing/nonhealing outcomes of NS RCT in relation to the obturation/instrumentation length; and (v) presence or absence of a periapical radiolucency. The 12 studies were classified into three categories based on the obturation length from the radiographic apex: (A) 0 to 1 mm; (B) >1 mm but <3 mm; and (C) obturated past the radiographic apex, including sealer. From the 12 studies, only 4 studies that included data that could be placed into the three length categories were further subjected to meta-analysis. Results of the meta-analysis indicated that the healing rate in group A (obturated 0 to 1 mm from apex) was marginally better than group C (obturated past apex) by 29%. Although group A demonstrated better healing than group B (obturated >1 mm short), the difference was not statistically significant. The authors concluded that a higher healing rate was achieved when treatment involved obturation short of the apex.

The aforementioned results derived from the sophisticated meta-analysis of multiple publications highlighted the data presented in the classic work performed by Sjögren and coworkers on healing of root-treated teeth with necrotic pulps and PL 8 to 10 years after treatment.[275] When those teeth were obturated within 2 mm of the apex, 94% revealed normal periapical conditions with the use of periapical radiography at the follow-up examination. Conversely, roots with overextended root fillings and those with obturations more than 2 mm short of the apex demonstrated significantly lower healing rates of 76% and 68%, respectively.

Similar results were also reported in a prospective clinical study on the healing[194] and survival[194] of teeth that had undergone primary or secondary NS RCT for 2 to 4 years. Absence of root-filling overextension was found to be a highly significant postoperative prognostic factor affecting the healing of both primary and secondary NS RCT.[194] With respect to tooth survival, overextension of GP obturations did not have any effect on tooth survival within the first 22 months but significantly increased the risk of tooth loss beyond 22 months.[194] This prognostic factor was common to both primary and secondary NS RCT. The authors attributed the delayed effect of overextension of GP obturations on tooth survival to the possibility of subcritical cracks created by excessive forces during compaction of GP, which eventually propagated during function and ultimately resulted in

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catastrophic failure.

On the basis of biologic and clinical principles, the conclusions derived from studies performed for more than two decades apart are all agreeable in that instrumentation and obturation should not extend beyond the apical foramen.[191] This was also demonstrated in a histological study that evaluated 41 human root-filled teeth from 36 patients.[234] In six cases exhibiting overextensions, histologic examination revealed severe inflammation.

Whereas the guideline of 1 mm from the radiographic apex remains rational when using radiographs, the point of apical termination of the preparation and obturation remains empirical. The use of an apex locator in conjunction with radiographs and sound clinical judgment makes this decision more logical. The need to compact the GP and sealer against the apical dentin matrix is necessary to prevent overextension of obturation materials into the periapical tissues. Deciding where the apical constriction of the RCS lies is based on the clinician's basic knowledge of apical anatomy, tactile sensation, radiographic interpretation, apex locators, apical bleeding, and, if not anesthetized, the patient's response.

Preparation for obturation

During the C&S process, organic pulpal materials and inorganic dentinal debris accumulate on the canal wall, producing an amorphous irregular smear layer (Fig. 9.9).[170,178,215] As shown in one of these studies, the smear layer is superficial, with a thickness of 1 to 5 μm.[178] This superficial debris can be packed into the dentinal tubules to various distances.[3]

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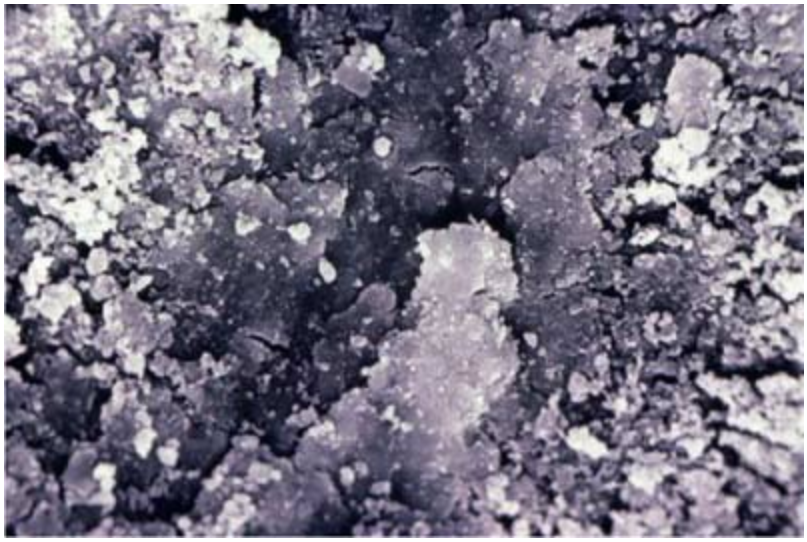

FIG. 9.9 Scanning electron microscopy of a prepared canal wall. The tubules are covered with a smear layer of organic and inorganic material.

Micrograph of a canal wall shows a granuled layer of smear over the wall surface.

In cases of pulpal necrosis, this layer may also be contaminated with bacteria and their by-products. For example, one study reported that bacteria can extend 10 to 150 μm into the dentinal tubules of necrotic teeth.[261] Another study noted that capillary action and fluid dynamics play a role in packing debris into the tubules.[5] Another investigation noted a mean penetration of 479 μm after a 28-day incubation period.[219]

The smear layer is not a complete barrier to bacteria but may act as a physical barrier, decreasing bacterial penetration into tubules. This was illustrated by a study reporting that removal of the smear layer permitted colonization of the dentinal tubules at a significantly higher rate when compared with leaving the smear layer in place.[82]

The smear layer may also interfere with adhesion and penetration of sealers into dentinal tubules.[305] Evidence indicates that sealer penetration into dentinal tubules does not occur when the smear layer is present.[99,211] For example, one study reported that removal of the smear layer permitted Roth 811 (Roth International, Ltd., Chicago, IL, USA), Calciobiotic root canal sealer (CRCS; Coltène/Whaledent, Cuyahoga Falls, OH, USA), and Sealapex (SybronEndo, Orange, CA, USA) to penetrate to between 35 and 80 μm,

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whereas the presence of the smear layer obstructed tubular penetration of all sealers.[297] Other studies reported that smear layer removal increased bond strength and reduced microleakage in teeth obturated with AH-26 (DENTSPLY Maillefer, Ballaigues, Switzerland).[86,100] Another investigation reported that a combination of smear layer removal, AH-26 as the sealer, and vertical compaction of GP had a cumulative effect in reducing leakage.[289]

There does not appear to be a consensus on removing the smear layer before obturation and the advantages/disadvantages of smear layer removal remains inconclusive.[59,262,265] However, growing evidence supports removal of the smear layer before obturation.[131,194,265] The organic debris present in the smear layer might constitute a substrate for bacterial growth.[215] It has been suggested that the smear layer prohibits sealer contact with the canal wall and permits leakage.[27] Bacterial penetration in the presence of a smear layer in RCS obturated with thermoplasticized GP and sealer has been shown to be significantly higher than with smear layer removal before obturation.[215] An additional consideration is the presence of viable bacteria that remain in the dentinal tubules and use the smear layer for sustained growth and activity.[39] Removal of the smear layer introduces the possibility of reinfecting the dentinal tubules if leakage occurs.[262] However, one study demonstrated that bacteria present before obturation are not viable after obturation.[77]

The smear layer may also interfere with the action of irrigants used as disinfectants.[204] When the smear layer is not removed, the dentinal tubules provide a long-term sanctuary for previously entrapped microorganisms to participate in reinfections. Additionally, the smear layer may slowly disintegrate and dissolve around leaking obturation materials or it may be removed by bacterial by-products such as acids and enzymes.[262]

The smear layer may interfere with the adhesion and penetration of root canal sealers. It also may prevent GP penetration during thermoplastic obturation techniques.[115] Significant tubular penetrations of GP and sealers have been reported with thermoplasticized obturations[115] and with dentin-bonded composite resins.[162] Removal of the smear layer also enhances the adhesion of sealers to dentin and tubular penetration.[162,201,262,304] Root canal filling materials adapt better to the canal walls after smear layer

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removal.[79,201,304,305]

An additional method for removing the smear layer involves sonic and ultrasonic instruments. In early studies of ultrasonic instrumentation, investigators noted the technique was effective in removing the smear layer.[69] Another investigator also demonstrated smear layer removal with ultrasonication and NaOCl.[53] One study compared the cleaning efficacy of short-term sonic and ultrasonic passive irrigation with 5.25% NaOCl after hand instrumentation in the apical 3 to 6 mm of maxillary molar root canals.[241] Passive sonic or ultrasonic irrigation for 30 seconds resulted in significantly cleaner canals than hand filing alone, and ultrasonic irrigation produced significantly cleaner canals than irrigation. However, other studies found ultrasonication and NaOCl to be ineffective in removing the smear layer.[23,300] In a more recent study, a group of investigators reported that the use of the Vibringe, EndoActivator, or needle irrigation did not significantly improve sealer penetration when compared with conventional irrigation.[36]

After the completion of C&S procedures, removal of the smear layer is generally accomplished by irrigating the canal with 17% disodium EDTA and 5.25% NaOCl (Fig. 9.10).[24] Chelators remove the inorganic components leaving the organic tissue elements intact. Sodium hypochlorite is necessary for removal of the remaining organic components. Citric acid has also been shown to be an effective method for removing the smear layer,[22,125] as has doxycycline[20] and tetracycline.[121]

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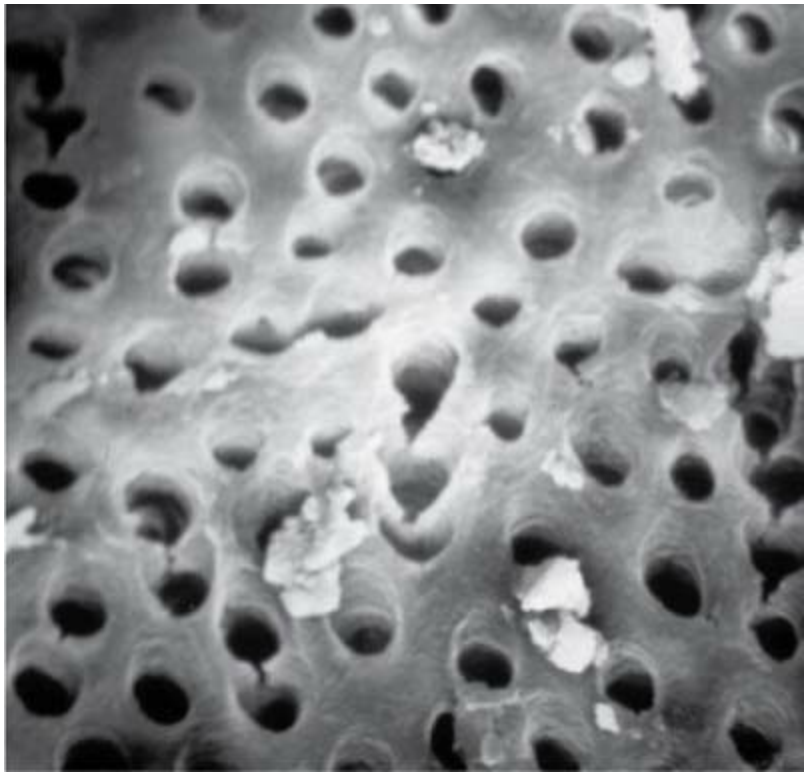

FIG. 9.10 Scanning electron microscopy of the canal wall after removal of the smear layer with 17% EDTA and 5.25% sodium hypochlorite.

Micrograph of a canal wall shows a porous surface with chunks of white fragments scattered over the surface and inside the pores.

Chelating agents were introduced to endodontic treatment by Nygaard-Østby in 1957 for treatment of calcified narrow RCS.[199] EDTA is the chelating solution customarily used in endodontic treatment. It is available in both liquid and viscous forms with common concentrations between 15% and 17%,[131] and its effectiveness is related to time, pH, and concentration.[199] Demineralization results in increased dentin permeability[112] because of the removal of the smear layer and plugs and enlargement of the tubules. It appears that the tubular enlargement is due to selective removal of the peritubular dentin.[129] The action of chelators and acids appears to be more effective in the coronal and middle thirds of the root and is reduced apically.[131,167] This reduced activity may reflect canal size.[210] This is a clinical concern because of the more irregular structure of dentin in the apical third. Another investigation demonstrated marked variations in the apical

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portion of the root,[183] including accessory RCS, areas of resorption and repaired resorptions, pulp stones, irregular or absent primary tubules, irregular secondary dentin, and cementum-like tissue lining the apical root canal wall. The variable structure of the apical region of human teeth presents challenges to the use of endodontic obturation techniques requiring adhesives, because this may influence the dentin bonding ability in the apical region.[183]

Although EDTA is mildly cytotoxic,[299] the irrigant appears to be biocompatible when used clinically;[199] however, irreversible decalcification of periapical bone and neuroimmunological disturbances have been noted.[259] EDTA decreases the microhardness of dentin.[67] Extrusion of both NaOCl and EDTA in clinical treatment should be avoided.[120,216,286]

The recommended time for removal of the smear layer is 1 to 5 minutes.[51,131,254] The small particles of the smear layer are primarily inorganic with a high surface-to-mass ratio that facilitates removal by acids and chelators. Investigators have found that a 1-minute exposure to 10 mL of EDTA was adequate to remove the smear layer and that a 10-minute exposure caused excessive removal of both peritubular and intratubular dentin.[51]

The use of EDTA in combination with NaOCl is recommended[24,316] and may enhance the cleaning[167] and antimicrobial effects of these solutions when compared with using them alone.[49] Although the use of EDTA alone does not cause erosion of the canal wall dentin, it should be noted the adjunctive use of NaOCl with EDTA may result in erosion of the intraradicular dentin depending on the timing and the concentrations of irrigants employed.[171,227,319]

New smear layer–removing irrigants are commercially available that combine calcium chelation of the inorganic component of the smear layer with antimicrobial activities. Examples of these irrigants include QMix 2in1 Irrigating Solution (DENSPLY Tulsa Dental Specialties).[16,70]

The ideal root canal obturation materials

Various endodontic materials have been advocated for obturation of the RCS.

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Most techniques employ a core material and sealer. Regardless of the core material, a sealer is essential to every technique and helps achieve a fluid-tight seal.

The American Association of Endodontists' *Guide to Clinical Endodontics* outlines contemporary endodontic treatment.[2] NS RCT of permanent teeth "involves the use of biologically acceptable chemical and mechanical treatment of the RCS to promote healing and repair of the periradicular tissues." The process is accomplished under aseptic conditions with rubber dam isolation. Regarding obturation, the guide states, "Root canal sealers are used in conjunction with a biologically acceptable semi-solid or solid obturating material to establish an adequate seal of the RCS." In this area, the guidelines indicate, "Paraformaldehyde-containing paste or obturating materials have been shown to be unsafe. Root canal obturation with paraformaldehyde-containing materials is below the standard of care for endodontic treatment" (Fig. 9.11). Chapter 27 gives further information about this issue.

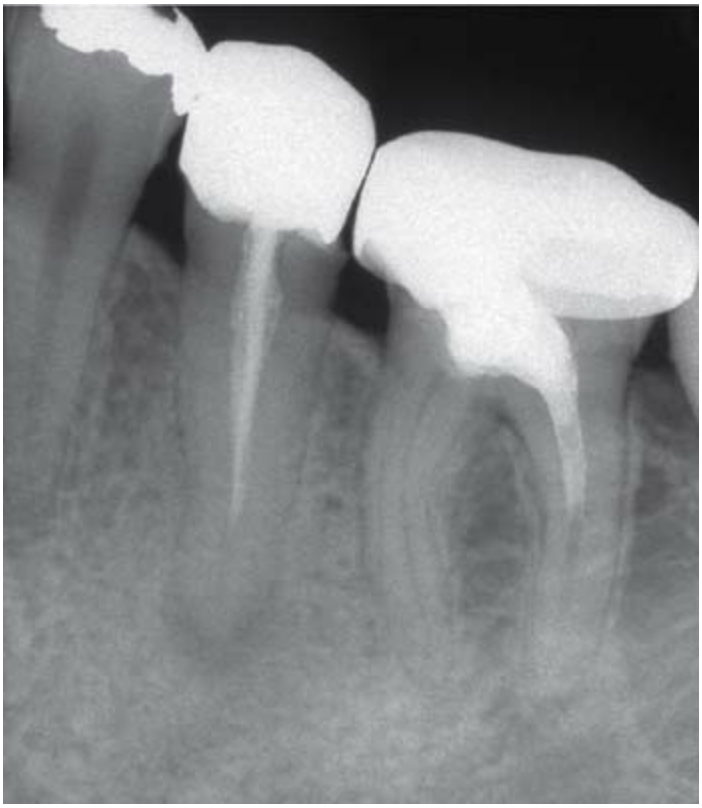

FIG. 9.11 A periapical radiograph of a mandibular left second premolar

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and first molar, demonstrating Sargenti paste NS RCT. In addition to the toxic material, the technique often accompanies inadequate C&S procedures.

Radiograph of a tooth shows a radiopaque crown, with dark neck and canals. An outline of canal filling paste is visible.

Assessment of nonsurgical treatment is based primarily on the posttreatment radiographic examination. The radiographic criteria for evaluating obturation include the following categories: length, taper, density, GP and sealer removal to the facial cementoenamel junction in anterior teeth and to the canal orifice in posterior teeth, and an adequate provisional or definitive restoration (Fig. 9.12).

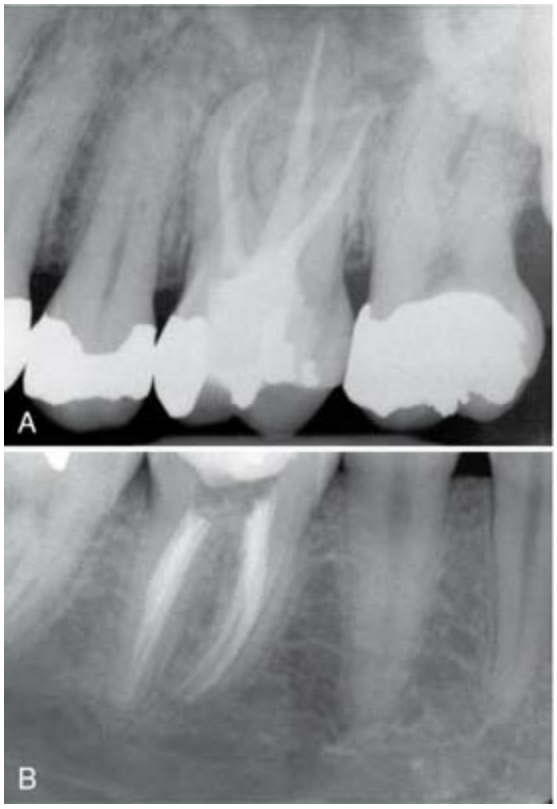

FIG. 9.12 **A,** Posttreatment radiograph of a maxillary right first molar demonstrating adequate length, density, and taper. **B,** Posttreatment radiograph of a mandibular right first molar with an adequate obturation.

A) Radiograph of a tooth shows a radiopaque part of the crown, with radiolucent neck and canals.

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B) Radiograph of a tooth shows a radiopaque crown, with dark neck and radiopaque canals.

Quality assurance is accomplished through a careful evaluation of treatment procedures. Only by this approach can deficiencies be identified and corrected. Although the anatomy and morphology of RCS vary tremendously, the obturated RCS should reflect the original shape of the RCS. Procedural errors in preparation, such as loss of length control, ledging, apical transportation, apical perforation, stripping perforation, and separated instruments, may not be correctable. Errors in obturation, such as length, voids, inadequate removal of prior obturation materials, and temporization, may be correctable.

Radiographic interpretation may vary among clinicians because of differences in radiopacity in root canal sealer/cements, constituents in specific brands of GP, interpretation of voids in vivo versus in vitro,[318] the overlying bony anatomy, radiographic angulation, and the limited two-dimensional view of the obturated RCS.

An often overlooked aspect in the assessment of RCS obturation is the density of the apical portion of the obturation. The apical third of the RCS may be filled with a sea of root canal cement and a single master cone or poorly compacted mass of previously softened GP. Radiographically, the apical third of the canal appears less radiodense. An ill-defined outline to the canal wall is evident, along with obvious gaps or voids in the filling material or its adaptation to the confines of the RCS. Because of the use of highly radiopaque root canal sealers/cements, the apical portion may be filled only with sealer giving the clinician the false impression of a dense, three-dimensional obturation with GP.

Root canal sealers vary in radiopacity.[230,285] Some contain silver particles or significant amounts of barium sulfate to enhance their radiopacity. Although these components may enhance the visualization of anatomic structures such as lateral canals, it is important to realize they do not increase the sealing ability of the sealer and the quality of the obturation. They may also give the impression that an RCS is well obturated when voids are masked by the density of the sealer. It is erroneous to claim that obturations with highly radiopaque sealers are better than those made with less radiopaque materials. This type of comparison and claim to superiority are

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both unfounded and unwarranted. The radiographic appearance or aesthetic appearance of the obturated canal system should be secondary to meticulous C&S. Although assessment of the RCS obturation is based on radiographic findings, root canal sealers do not have to be highly radiopaque to be effective.

Types of sealers

Root canal sealers are necessary to seal the space between the dentinal wall and the obturating core interface. Sealers also fill voids and irregularities in the RCS, lateral and accessory canals, and spaces between GP points used in lateral compaction. Sealers also serve as lubricants during the obturation process. Grossman outlined the properties of an ideal sealer (Box 9.1).[110] Presently, no sealer satisfies all the criteria.

Box 9.1

Properties of an Ideal Sealer

- Exhibits tackiness when mixed to provide good adhesion between it and the canal wall when set
- Establishes a hermetic seal
- Radiopaque, so that it can be seen on a radiograph
- Very fine powder, so that it can mix easily with liquid
- No shrinkage on setting
- No staining of tooth structure
- Bacteriostatic, or at least does not encourage bacterial growth
- Exhibits a slow set
- Insoluble in tissue fluids
- Tissue tolerant; that is, nonirritating to periradicular tissue
- Soluble in a common solvent if it is necessary to remove the root canal filling

Sealers should be biocompatible and well tolerated by the periradicular

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tissues.[140] All sealers exhibit toxicity when freshly mixed; however, their toxicity is greatly reduced on setting.[142,155] Sealers are resorbable when exposed to tissues and tissue fluids, but not all extruded sealers are predictably removed from the periradicular tissues.[17,235] Tissue healing and repair generally appear unaffected by most sealers, provided there are no adverse breakdown products of the sealer over time.[35,40-42,44] Breakdown products from sealers may have an adverse effect on the proliferative capability of periradicular cell populations, specifically in the presence of the apical lesion.[235] As a result, sealers should not be placed routinely in the periradicular tissues as part of an obturation technique.

The most popular sealers are zinc oxide–eugenol formulations, $Ca(OH)_2$ sealers, glass ionomer sealers, resin-based epoxy resin or methacrylate resin sealers, and the recently introduced calcium silicate–based sealers. Despite claims by the manufacturers on the advantages of each class, there is no evidence-based data, based on randomized clinical trials, demonstrating the superiority of one sealer over another. Regardless of the sealer selected, all exhibit toxicity until they have set. For this reason, extrusion of sealers into the periradicular tissues should be avoided (Fig. 9.13).[246]

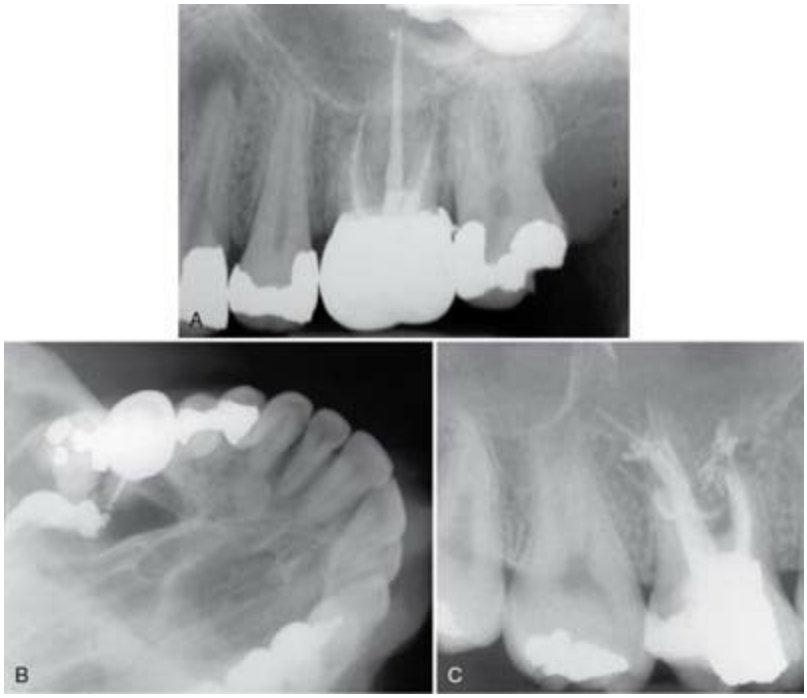

FIG. 9.13 **A,** Extrusion of sealer evident on the posttreatment

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radiograph of a maxillary first molar. The separated lentulo spiral in the mesiobuccal root indicates a possible method of sealer placement. **B,** Maxillary occlusal film demonstrates that the sealer is located in the maxillary sinus. Correction by nonsurgical techniques is not possible. **C,** Maxillary right first molar with extrusion of the sealer and GP.

A) Radiograph of a tooth shows a radiopaque crown and canals.

B) Radiograph of set of teeth shows a tooth with radiopaque crown. A radiopaque outline of the sealer is visible.

C) Radiograph of a tooth shows a radiopaque crown and irregular canals. A radiopaque outline due to the extrusion of the sealer is visible.

Zinc oxide and eugenol

Zinc oxide–eugenol sealers have a history of successful use over an extended period of time. Zinc oxide–eugenol sealers will resorb if extruded into the periradicular tissues.[17] They exhibit a slow setting time,[7] shrinkage on setting,[143] solubility,[221] and they can stain tooth structure.[151] An advantage to this sealer group is antimicrobial activity.[6,19,123]

Marketed as Pulp Canal Sealer (Kerr Endo) and Pulp Canal Sealer EWT (extended working time), this sealer is popular with clinicians using thermoplastic techniques.

Grossman modified the formulation and introduced a nonstaining formula in 1958 but it has been discontinued (Table 9.1),[111] but Pulp Canal Sealer EWT (>6 hours on the mixing pad) and EWT (Kerr) have a similar formulation. Tubli-Seal (Kerr) is a catalyst/base zinc oxide–eugenol sealer that is convenient to mix but has a faster setting time when compared with the liquid/powder sealers. Tubli-Seal EWT provides an extended working time.

Table 9.1

Formula for Zinc Oxide–Eugenol Root Canal Sealer

Powder	Liquid
Zinc oxide	42 parts
Staybelite resin	27 parts

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Bismuth subcarbonate	15 parts
Barium sulfate	15 parts
Sodium borate, anhydrous	1 part

Although zinc oxide–eugenol sealers possess marked cytotoxic and tissue-irrigating potencies in ex vivo cell culture studies,[142] their clinical usefulness has been well demonstrated in retrospective and prospective human clinical studies.[66,291] This discrepancy between the results of in vitro and in vivo cytotoxicity testing may be explained by the fact that most cell culture systems are represented by only one cell type, that is, no cell-cell interactions, which is often monoclonal in origin. Another important issue to consider is the fact that culture conditions are not homeostatic and there is no elimination of toxic substances as there would be in vivo. By contrast, the human body possesses a lymphatic system and periapical defenses such as polymorphonuclear leukocytes, plasma cells, and macrophages to help eliminate toxic substances.[55] These mechanisms do not exist in a culture plate and must be taken into account for interpretations of the results of cell culture-based cytotoxicity studies reported in the endodontic literature.

Calcium hydroxide sealers

Calcium hydroxide sealers were developed for therapeutic activity. It was thought that these sealers would exhibit antimicrobial activity and have osteogenic–cementogenic potential. Unfortunately, these actions have not been demonstrated.[78,184] Solubility is required for release of $Ca(OH)_2$ and sustained activity. This is inconsistent with the purpose of a sealer. CRCS is a zinc oxide–eugenol sealer with $Ca(OH)_2$ as one ingredient. Sealapex (SybronEndo) is a catalyst/base system. The base contains zinc oxide, $Ca(OH)_2$, butyl benzene, sulfonamide, and zinc stearate. The catalyst contains barium sulfate and titanium dioxide as radiopacifiers in addition to resin, isobutyl salicylate, and aerosol R972. Apexit and Apexit Plus (Ivoclar Vivadent, Schaan, Liechtenstein) consist of an activator (disalicylate, bismuth hydroxide/bismuth carbonate, and fillers) and a base [$Ca(OH)_2$, hydrated colophonium (i.e., pine resin) and fillers].

Although an osteogenic response has been observed with $Ca(OH)_2$-based root canal sealers,[128,276,284] the ability of these sealers to sustain a high pH

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over time and produce an alkaline pH at the root surface has been questioned.[89]

Better biocompatibility and osteogenic potential have been reported by $Ca(OH)_2$-producing tricalcium silicate-based root canal sealers than those with conventional $Ca(OH)_2$ sealers (Sealapex).[61]

Noneugenol sealers

Developed from a periodontal dressing, Nogenol (GC America, Alsip, IL) is a root canal sealer without the irritating effects of eugenol. The base contains zinc oxide, barium sulfate, and bismuth oxychloride.

Glass ionomer sealers

Glass ionomers have been advocated for use in obturation because of their dentin-bonding properties. Ketac-Endo (3M ESPE, Minneapolis, MN, USA) enables adhesion between the material and the canal walls.[98] It has been reported to have compatible outcomes compared with previous studies.[98] However, it is difficult to properly treat the dentinal walls in the apical and middle thirds with conditioning agents to receive the glass ionomer sealer. A disadvantage of glass ionomers is that they are more difficult to remove if retreatment is required.[187] This sealer has minimal antimicrobial activity.[63]

Activ GP (Brasseler USA) consists of a glass ionomer–impregnated GP cone with a glass ionomer external coating and a glass ionomer sealer (Fig. 9.14). Available in 0.04 and 0.06 tapered cones, the sizes are laser verified to ensure a more precise fit. This single cone technique is designed to provide a bond between the dentinal canal wall and the master GP cone that would be impregnated with glass ionomer filler particles resulting in an alleged monoblock.[95]

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FIG. 9.14 Activ GP (Brasseler USA, Savannah, GA) glass ionomer–coated GP points and sealer.

Close-up of two Activ gutta percha system shows two bottles in a liquid and powder combination with a set of gutta percha points coated with glass ionomer.

Resin sealers

Resin sealers have a long history of use, provide adhesion, and do not contain eugenol. There are two major categories: epoxy resin and methacrylate resin.

Epoxy resin sealers

AH-26 (DENTSPLY DeTrey, Konstanz, Germany) is a slow-setting epoxy resin that was found to release formaldehyde when setting.[149,278] AH Plus (DENTSPLY DeTrey) is a modified formulation of AH-26 in which formaldehyde is not released (Fig. 9.15).[163] The sealing abilities of AH-26 and AH Plus appear comparable. AH Plus is an epoxy resin-amine based system that comes in two tubes. The epoxide paste tube contains a diepoxide (bisphenol A diglycidyl ether) and fillers as the major ingredients, while the amine paste tube contains a primary monoamine, a secondary diamine, a disecondary diamine, silicone oil, and fillers as the major ingredients. It

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exhibits a working time of approximately 4 hours.

FIG. 9.15 AH Plus sealer is a resin formulation.

Close-up of two AH plus sealer tubes used for sealing root canals.

Source: (Courtesy DENTSPLY, Konstanz, Germany. All rights owned by and used with permission from DENTSPLY International, Inc.)

Methacrylate resin sealers

Four generations of methacrylate resin-based root canal sealers have been marketed for commercial use.[147,212] The first generation of hydrophilic methacrylate resin-based material (Hydron; Hydron Technologies, Inc., Pompano Beach, FL, USA) was designed for en masse obturations and appeared in the mid-1970s when scientific foundations behind dentin bonding were in their infancy stage of development. The major component of Hydron was poly [2-hydroxyethyl methacrylate] (poly [HEMA]), which was injected into an RCS and polymerized in situ within the RCS without the adjunctive use of a core obturation material. Hydron became obsolete in the 1980s as subsequent clinical outcomes were unacceptable.[156]

Following the marketing of self-priming, self-etching, and self-adhesive resin cement technologies in restorative dentistry, functionally analogous, low viscosity methacrylate resin-based root canal sealers have since been available for use in endodontics. This type of bondable root canal sealers has

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been aggressively promoted with the highly desirable property of creating monoblocks within the RCS space.[288] The term *monoblock* refers to the idealized scenario in which the canal space becomes perfectly filled with a gap-free, solid mass that consists of different materials and interfaces, with the purported advantages of simultaneously improving the seal and fracture resistance of an obturated RCS.

The second generation of bondable sealer is nonetching and hydrophilic in nature and does not require the adjunctive use of a dentin adhesive. It is designed to flow into accessory canals and dentinal tubules to facilitate resin tag formation for retention and seal after smear layer removal with NaOCl and EDTA. EndoREZ (Ultradent Products Inc., South Jordan, UT, USA) is a dual-cured radiopaque hydrophilic methacrylate sealer that contains nonacidic diurethane dimethacrylate. The addition of triethyleneglycol dimethacrylate to the sealer composition renders it hydrophilic, so that it may be used in the wet environment of the RCS and be very effective in penetrating dentinal tubules and forming long resin tags.[29,287] The sealer was found to seal best when applied to slightly moist intraradicular dentin.[324] EndoREZ is recommended for use with either a conventional GP cone or specific EndoREZ points that are resin-coated GP. A retrospective clinical and radiographic study evaluating the 10-year treatment outcome of one-visit NS RCT using GP and the EndoREZ sealer reported accumulative probability of success of 92.1% after 10 years.[323] The authors concluded that EndoREZ may be recommended as an alternative to other commonly used root canal sealers. Unfortunately, no additional root canal sealer was used in that study for comparison.

New generations of self-etching (third generation) and self-adhesive (fourth generation) resin cements have been introduced to restorative dentists to simplify bonding procedures. They became commercially available shortly after the introduction of those resin cement systems. The third-generation self-etching sealers contain a self-etching primer and a dual-cured resin composite root canal sealer. The use of self-etching primers reintroduced the concept of incorporating smear layers created by hand/rotary instruments along the sealer-dentin interface. An acidic primer is applied to the dentin surface that penetrates through the smear layer and demineralizes the superficial dentin. The acidic primer is air-dried to remove the volatile carrier

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and then a dual-cured moderately filled flowable resin composite sealer is applied and polymerized. Provided that these materials are sufficiently aggressive to etch through thick smear layers, the technique sensitivity of bonding to RCS may be reduced when smear layers are inadvertently retained in the apical third of instrumented canal walls.

Third-generation methacrylate resin-based sealers that incorporate the use of self-etching primers became popularized following the introduction of Resilon (Resilon Research LLC, Madison, CT, USA), a dimethacrylate-containing polycaprolactone-based thermoplastic root filling material.[266] Resilon has been removed from the market and is no longer available.

The fourth-generation methacrylate resin-based sealers (e.g., MetaSEAL, Parkell Inc., RealSeal SE, SybronEndo) is functionally analogous to a similar class of recently introduced self-adhesive resin luting cements in that they have further eliminated the separate etching/bonding step.[229] Acidic resin monomers that are originally present in dentin adhesive primers are now incorporated into the resin-based sealer/cement to render them self-adhesive to dentin substrates. The combination of an etchant, a primer, and a sealer into an all-in-one self-etching, self-adhesive sealer is advantageous in that it reduces the application time as well as errors that may occur during each bonding step. MetaSEAL is the first commercially available fourth-generation self-adhesive dual-curable sealer.[158] The sealer purportedly bonds to thermoplastic root-filling materials as well as radicular dentin via the creation of hybrid layers in both substrates. MetaSEAL is also marketed as Hybrid Bond SEAL (Sun Medical Co. Ltd., Shiga, Japan) and had been reported to produce equivalent or slightly inferior sealing properties as conventional nonbonding epoxy resin-based sealers.[28,202]

Silicone sealers

RoekoSeal (Coltène/Whaledent AG, Altstätten, Switzerland) is a polydimethylsiloxane that has been reported to expand slightly on setting.[205]

GuttaFlow and GuttaFlow2 (Coltène/Whaledent) are cold flowable matrices that are triturated. They consist of GP in a particulate form less than 30 μm added to RoekoSeal (Fig. 9.16). The material is provided in capsules for trituration. The technique involves injection of the material into the RCS, followed by placement of a single master cone. The material provides a

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working time of 15 minutes and it cures in 25 to 30 minutes. Evidence suggests that the material fills canal irregularities with consistency[320] and is biocompatible,[65,174] but the setting time is inconsistent and may be delayed by final irrigation with sodium hypochlorite.[37]

FIG. 9.16 GuttaFlow trituration capsule and injection syringe (Coltène/Whaledent, Cuyahoga Falls, OH).

Close-up of the gutta flow trituration system shows a cylindrical capsule with a cap and a gun-shaped injection syringe with a metallic barrel and V-shaped trigger point.

GuttaFlow bioseal, the most recent introduction to this family of silicone sealers, incorporates bioactive ingredients such as calcium and silicate into the original formulation, which is claimed by the manufacturer to stimulate tissue healing and regeneration.[52] A recent study reported that GuttaFlow bioseal promotes spontaneous differentiation of human periodontal ligament stem cells into cementoblast-like cells in the absence of adjunctive growth factors.[238] If these results can be validated in in vitro experiments, GuttaFlow bioseal should have the potential to promote the regeneration of the cementum in teeth with apical periodontitis or chronic abscess.

Tri/dicalcium silicate sealers

Root canal sealers based on tricalcium silicates and dicalcium silicates are the

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latest group of bioactive root canal sealers introduced for obturation of an RCS.[135] These sealers are an outgrowth of the popularity of mineral trioxide aggregate (MTA) materials, which are based on tricalcium silicate and dicalcium silicate, a water-setting hydraulic powder used for various surgical and vital pulp therapy treatments.[18,214,292] This type of root canal sealer is attractive because of the bioactivity that has been reported for MTA-type materials,[198] which are also known for being hydrophilic.[54]

MTA is a hydraulically active powder that contains primarily tricalcium silicate, dicalcium silicate, and a radiopaque powder, often bismuth oxide. Because of staining of crown dentin by the bismuth oxide component, which may be rendered brown in the presence of NaOCl, gray in the presence of chlorhexidine, or even black in the presence of glutaraldehyde, the radiopacifier has now been replaced with other materials such as zirconia dioxide or tantalum oxide in some commercial formulations. The radiopaque agent, bismuth oxide, zirconia, or tantalum oxide, is important, without which MTA would not be distinguishable on a radiograph. Minor phases of tricalcium aluminate and calcium sulfate are often present. Some MTA-type products contain calcium carbonate, or tetracalcium aluminoferrite. The term MTA is used here to denote all the tri- and dicalcium silicate products.

Tri/dicalcium silicate cements/sealers are set by reaction with water and form a highly alkaline (pH ; 12) mixture consisting of a rigid matrix of calcium silicate hydrates and $Ca(OH)_2$.[68,71] These hydrates form on the surface of the original calcium silicate particles, and hydration gradually penetrates inward. When a tricalcium silicate cement sets, the dimensional change is less than 0.1% expansion, which helps with creating a barrier, which is especially important for its use during endodontic treatment. Setting time for tricalcium silicate cements is lengthy, about 165 minutes for the initial set and less than 6 hours for the final set, which has been a major drawback for using them in some indications, but not for its use as a sealer. During the early stage of development, the inconsistency of MTA products with water has rendered it not suitable for use as a sealer. Additionally, the coarseness of the first MTA products, intended for surgical uses, made them unsuitable for use as a sealer, as their film thickness was too high (>50 μm).

To date, many tri/dicalcium silicate-based sealers have been available commercially (Figs. 9.17–9.19).[135,166,179,267,296,301]

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FIG. 9.17 BioRoot RCS (Septodont, Saint-Maur-des- Fossés, France).

A packaging of BioRoot water-based root canal sealer with a bottle and a mini scoop.

FIG. 9.18 iRoot SP (Innovative BioCeramix Inc., Vancouver, Canada; *aka* EndoSequence BC sealer; Brasseler USA, Savannah, GA).

An iRoot SP syringe shows a plastic barrel, plunger and a tip.

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FIG. 9.19 MTA Plus (NuSmile, Houston, TX).

A mineral trioxide aggregate with bottles of Portland cement and bismuth oxide. A mixture of the components is visible on a glass panel with a spatula and a mini scoop.

They include the following: MTA Fillapex (Ângelus Indústria de Produtos Odontlógicos Ltda; Londrina, Paraná, Brazil), iRoot SP (Innovative BioCeramix Inc., Vancouver, Canada; *aka* Endosequence BC sealer; Brasseler USA, Savannah, GA, USA), Endo CPM Sealer (EGEO SRL, Buenos Aires, Argentina), MTA Plus (NuSmile, Houston, TX, USA), Endoseal MTA (Maruchi, Wonju, Korea), Total Fill BC Sealer (FKG Dentaire, La-Chaux-de-Fonds, Switzerland), BioRoot RCS (Septodont, Saint-Maur-des- Fossés, France), Seal Plus BC (MK Life, Porto Alegre, RS, Brazil). Table 9.2 lists what is known about the components of these sealers.

Table 9.2

Composition of Tricalcium Silicate Root Canal Sealers

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Product	Manufacturer	System	Composition	Radiopacifier
NeoMTA Plus	Nu Smile, Houston, TX, USA	Powder/water-based gel	Tri/dicalcium silicate	Ta_2O_5
NeoSealer		Organic liquid paste in Syringe	Tri/dicalcium silicate	Ta_2O_5
BioRoot RCS	Septodont, Saint-Maur-des- Fossés, France	Powder/water-based liquid	Tri/dicalcium silicate	ZrO_2
iRoot, EndoSequence BC, Edge Endo, TotalFill BC sealer	Innovative BioCeramix Inc.; Brasseler USA	Organic liquid paste in syringe	Tri/dicalcium silicate	ZrO_2, Ta_2O_5
Endo CPM sealer	EGEO SRL Buenos Aires, Argentina	Powder/liquid	Tri/dicalcium silicate	Bi_2O_3, $BaSO_4$
MTA Fillapex	Ângelus Indústria de Produtos Odontlógicos Ltda	Dual paste	Disalicylate resins	Bi_2O_3
Bio-C		Organic liquid paste in syringe	Tri/dicalcium silicate	ZrO_2
Seal Plus BC	MK Life, Porto Alegre, RS, Brazil	Organic liquid paste in syringe	Tri/dicalcium silicate	ZrO_2

iRoot SP (EndoSequence BC Sealer), TotalFill Sealer, and Bio-C Sealer are premixed sealers. MTA Fillapex and Seal Plus BC are available as two paste syringes that are combined in a mixing tip. BioRoot RCS, Endo CPM Sealer, and MTA Plus sealers are powder/gel systems that the user can mix to the consistency desired. The available materials' manufacturers descriptions are vague. For example, MTA Fillapex has "natural or diluent resins," with "filler and thickening agents." However, what is believed to be in common is that tricalcium silicate powder is a component of all these sealers.

The MTA Fillapex and iRoot SP sealers have nonaqueous vehicles in which the powders are dispersed. For the tricalcium silicate particles to contribute to sealing, they must become hydrated in the tooth by exchange of the nonaqueous vehicle for water in the RCS. EndoCPM Sealer and MTA Plus sealer rely on mixing with water-based gels just before use without a nonaqueous resin. The proportions of the tricalcium silicate powders are not known in any of these materials but are most likely to be highest in MTA Plus because no inert liquid vehicle is included. The inert vehicle is the liquid medium in the pastes of MTA Fillapex and iRoot SP that does not react with the powder. The bioactivity of MTA-type materials has made the use of tricalcium silicate powders in sealers of interest. The possibility of overextension past the apex with a bioactive material leads to the supposition that healing in the periapical area would occur more readily with a tricalcium

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silicate product. However, two of the sealers have the tricalcium silicate powders dispersed in a nonreactive organic medium, which may detract from the potential benefit of the bioactive powder. To date, no peer-reviewed papers have been published to establish the benefits of tricalcium silicate powders in sealers in human clinical studies. All studies and reports were based on in vitro testing and in vivo animal models. Calcium carbonate is added to Endo CPM Sealer to reduce the pH from 12.5 to 10.0 after setting. By doing so, the manufacturer claimed that surface necrosis in contact with the material is reduced, thereby optimizing the conditions for the enzymatic action of alkaline phosphatase.[107]

The physical properties of some of these sealers have been published. Table 9.3 contains values that are known for comparison to the ISO 6876/ADA 57 methods and requirements and the AH Plus sealer. The flow, film thicknesses, solubility, dimensional stability, and radiopacity of the new sealers meet the ISO 6876 requirements. The flow of these sealers varies, but so do the values for AH Plus. The dimensional stability is excellent. The solubility of the sealers is higher than AH Plus, which may be attributed to the formation of partially soluble $Ca(OH)_2$ within the set tricalcium silicate. The radiopacities are less than AH Plus, which is known for its radiopacity.

Table 9.3

Physical Properties of Tricalcium Silicate Root Canal Sealers

Quality	ISO 6876-2001 ADA 57 Requirement	AH Plus*,†	MTA Fillapex†	EndoSequence BC Sealer	MTA Plus White, Gray‡
Flow (mm)	>20	21, 40	25, 29	23	31
Film thickness (μm)	<50	16	24	22	47
Solubility	<3%	0.1	1.1	2.9	1.9
Dimensional stability	−1% to +0.1%	−0.04	−0.7	+0.09	+0.05
Radiopacity	>3 mm Al	9	4	4	4.9, 5.5
Working time (hours)	NR	4:00, 5:30	0:45, 0:30	2:40	0:20
Setting time (hours)	NR	11:30, 5:10	2:30, 2:15	>24	10:00
Antimicrobial Efficacy	NR	§	§	§	§

*An epoxy resin-based sealer, included for comparative purposes.

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†Measured by two authors.

‡If one value is given, then white and gray versions of MTA Plus are equal.

§Antimicrobial effect against the Enterococcus faecalis.

MTA, Mineral trioxide aggregate; *NR*, no requirement.

The working and setting times of the iRoot SP and MTA Plus materials are longer than the MTA Fillapex sealer. iRoot SP has the longest setting time and requires diffusion of water from the dentinal tubules into the sealer to set.[188] In a test where water was added to the EndoSequence BC sealer (iRoot SP), the setting time was reduced to about 150 hours but the hardness was significantly diminished.[168] The EndoSequence BC sealer has been tested for strengthening teeth when used with glass ionomer-coated cones and has been found to be stronger in vitro.[102]

Biocompatibility tests have been performed with some of these sealers. EndoSequence BC sealer was more cytotoxic than AH Plus root canal sealer. The time to become non-cytotoxic as judged from mitochondrial enzymatic activity using succinic dehydrogenase was 5 weeks for EndoSequence BC sealer and 3 weeks for AH Plus sealer.[168] Another study reported that, after setting, the cytotoxicity of MTA Fillapex decreased and the sealer presented suitable bioactivity to stimulate hydroxyapatite nucleation.[61,245] A calcium silicate-based sealer possesses a higher modulus of elasticity than AH Plus sealer, unmodified and modified GP cones, and provided better adhesion and sealer penetration at the apical area.[206] A recent systematic review evaluated the biocompatibility of different bioceramic materials in both human and animal mesenchymal cells and reported that all tricalcium silicate materials have similar biological properties to those of MTA, including low cytotoxicity, promotion of cell adhesion and proliferation, and reduced inflammatory cytokines, cells, and mediations.[76] Thus, given the similarity, the choice of bioceramic material should be a professional's decision.

To date, no peer-reviewed papers have been published to establish the benefits of tricalcium silicate powders in sealers in human clinical studies. All studies and reports were based on in vitro testing and in vivo animal models. Despite the lack of well-designed long-term clinical trials, the premixed calcium silicate-based sealers show good physicochemical and biological properties in vitro, with results similar or better than conventional

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root canal sealer sealers, as observed in in vitro and in vivo animal studies.[268]

A concern associated with the use of tri/dicalcium silicate sealers is the potential difficulty in retreating a hard material.[126,150] It has to be remembered though that, similar to the recommendations for other root canal sealers, a tri/dicalcium sealer is supposed to be employed as a thin film instead of being used en bloc, for both single cone and lateral compaction techniques. For retreatment of root canals that had been filled with GP and tri/dicalcium silicate sealers, removal of the sealer remnants and the regaining of apical patency were no worse than with epoxy or resin-based sealers.[57,192]

Medicated sealers

Sealers containing paraformaldehyde are strongly contraindicated in endodontic treatment (Fig. 9.20). Although the lead and mercury components may have been removed from these zinc oxide–eugenol formulations over time, the severely toxic paraformaldehyde content has remained a constant. These sealers are not approved by the U.S. Food and Drug Administration[1] and are unacceptable under any circumstances in clinical treatment because of the severe and permanent toxic effects on periradicular tissues.[263] A paste containing 6.5% paraformaldehyde as well as lead and mercury was advocated for use by Sargenti[248] and originally marketed as N-2. Lead has been reported in distant organ systems when N2 is placed within the radicular space.[207] In another study the investigators reported the same results regarding systemic distribution of the paraformaldehyde component of N2.[32] Removal of the heavy metals resulted in a new formulation: RC2B. Other paraformaldehyde sealers include Endomethasone, SPAD, and Reibler's paste. The toxic in vivo effects of these materials on the pulp and periapical tissues have been demonstrated over time.[64,193]

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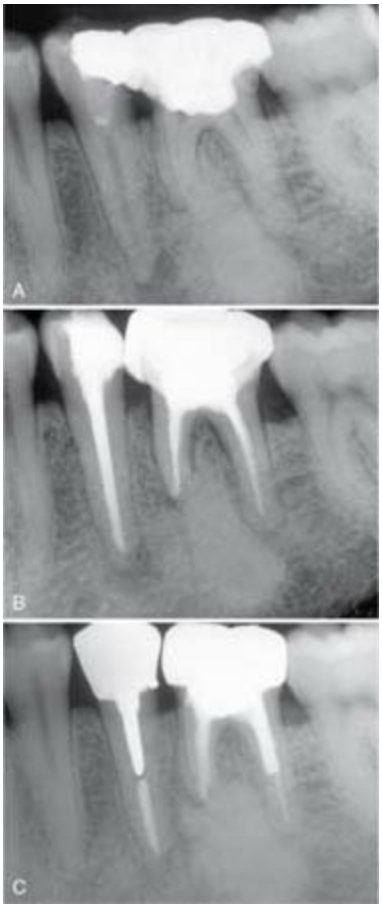

FIG. 9.20 Patient treated with Sargenti paste in her mandibular left second premolar and first molar. **A,** Pretreatment radiograph exhibits an osteolytic response associated with the premolar and a proliferative response associated with the molar. **B,** Posttreatment radiograph of the teeth. **C,** One-year follow-up radiograph exhibiting osseous regeneration apical to the second premolar.

A) Radiograph of teeth shows the first tooth with partial radiopaque crown, radiolucent neck and dark canal; second tooth shows an irregularly radiopaque crown and neck.

B) Radiograph of teeth shows the first tooth with partial radiopaque crown, radiolucent neck and dark canal; second tooth shows a partial radiopaque crown, neck and a vertical canal. A radiopaque outline of sealer is visible.

C) Radiograph of teeth shows the first tooth with radiopaque crown, neck and upper canal with radiolucent lower canal; second tooth shows a radiopaque crown, neck and a radiopaque upper canals. An radiopaque outline of sealer is visible.

In addition to the toxic nature of the material, clinicians employing the material place it with a lentulo spiral. Overextension has resulted in

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osteomyelitis and paresthesia,[88] where irreversible neurotoxicity has manifested as dysesthesia as a result of paraformaldehyde being forced through the apical foramen into the periapical tissues.[148] The reader is referred to Chapter 27 for further discussion about this harmful material and technique.

Sealer placement

Various methods of sealer placement have been advocated, including a master cone, lentulo spiral, a file and/or reamer, and ultrasonics. Investigators compared sealer placement using a file rotated counterclockwise, the lentulo spiral (Fig. 9.21), an ultrasonic file, and coating the master GP cone.[306] Placement did not differ with the various techniques. However, the investigators noted the most variation in sealer coating was in the apical area.[306] Another study compared sealer placement with a K-type file, the lentulo spiral, and using the master cone in curved canals. Results demonstrated no significant differences in the techniques after obturation. No technique covered more than 62.5% of the RCS.[118] Other investigators reported that ultrasonics produced the best sealer distribution when used circumferentially.[3,279] In the past, it has been presumed that instrumented RCS need to be thoroughly dried prior to obturation with root filling materials and sealers. Indeed, there were leakage studies reporting that a better apical seal might be achieved by drying the canals with 95% ethanol prior to obturation.[280] A recent study reported that, for some sealers (iRoot SP, AH Plus, Epiphany, and MTA Fillapex), better adhesion to the dentinal walls might be achieved by leaving the canals slightly moist. This can be accomplished by drying the RCS with low vacuum via a Luer adapter for 5 seconds followed by insertion of a paper point for 1 second.[188] Similar results were also reported with the use of the EndoREZ sealer.[324] This, however, does not mean that a RCS should remain totally flooded with water prior to obturation because such a level of residual moisture would adversely affect the adhesive of those root canal sealers to intraradicular dentin.[188]

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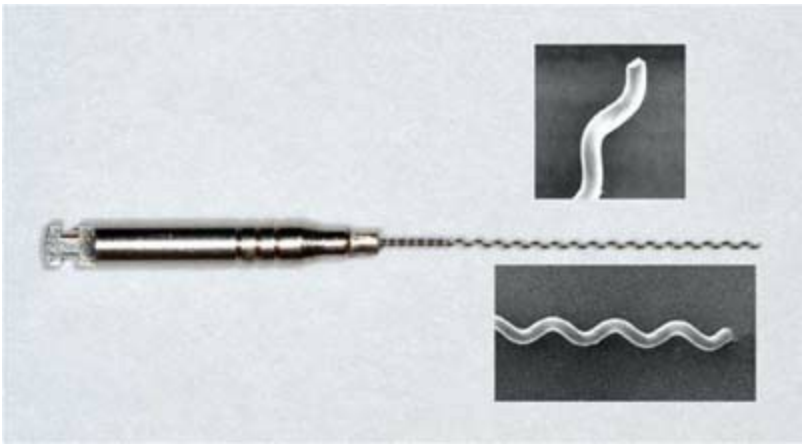

FIG. 9.21 Lentulo spiral used for sealer placement during obturation.

Close-up shows a lentulo spiral instrument with a metallic handle, a spiral body and an almost flat tip.

The method of obturation does not seem to affect the sealer distribution on the canal wall in the apical portion of the canal. However, lateral compaction results in better distribution in the midcoronal areas when compared with warm vertical compaction.[312] Another well-controlled study reported that none of five evaluated obturation techniques resulted in uniform sealer distribution along the entire length of the core obturation material.[130] Evidence indicates that the method of obturation affects the sealer penetration into tubules. This was exemplified by a study reporting that thermoplastic techniques produced deeper sealer penetration into tubules.[74] Removal of the smear layer enhances sealer penetration into the dentinal tubules.[152]

Core materials

Although a variety of core materials have been used in conjunction with a sealer/cement, the most common method of obturation involves GP as a core material. Regardless of the obturating technique, emphasis should be placed on the process of C&S the RCS. The materials and techniques described do not routinely create an impervious seal of the RCS. All materials leak to some extent.[4] The choice of the obturation technique(s) depends on the unique circumstances with which each case presents.

The properties of an ideal obturation material have been outlined by Grossman (Box 9.2).[110] Historically, a variety of materials have been

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employed to obturate the RCS. Solids, semisolid materials, and pastes have been employed. A common solid material used in the past was the silver cone.

Box 9.2

Properties of an Ideal Obturation Material

- Easily manipulated and provides ample working time
- Dimensionally stable with no shrinkage once inserted
- Seals the canal laterally and apically, conforming to its complex internal anatomy
- Nonirritating to the periapical tissues
- Impervious to moisture and nonporous
- Unaffected by tissue fluids—no corrosion or oxidization
- Inhibits bacterial growth
- Radiopaque and easily discernible on radiographs
- Does not discolor tooth structure
- Sterile
- Easily removed from the canal if necessary

Silver cones

Jasper introduced cones made of silver, which he claimed produced the same success rate as GP and were easier to use.[136] The rigidity provided by the silver cones made them easier to place and permitted more predictable length control; however, their inability to fill the irregularly shaped RCS permitted leakage (Fig. 9.22). Silver cones were believed to possess an oligodynamic property which, if true, would have resulted in the destruction of microbes within the RCS. Unfortunately, it was not true. Moreover, when silver points contact tissue fluids or saliva, they corrode.[38] The corrosion of such products has been found to be cytotoxic and produces pathosis or impedes periapical healing.[260]

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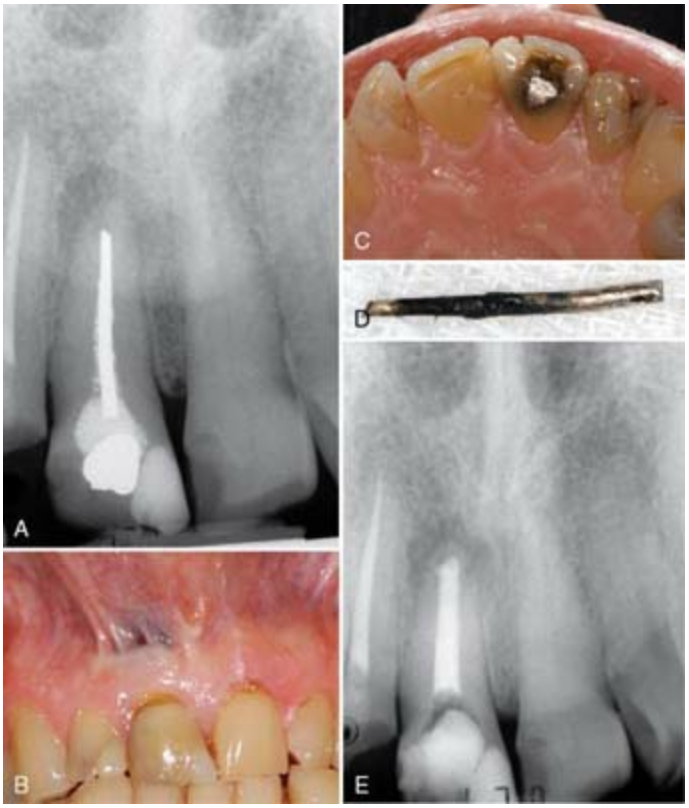

FIG. 9.22 Silver cones have been advocated for ease of placement and length control. **A,** Radiograph of a maxillary right central incisor obturated with a silver cone. **B,** Tissue discoloration indicating corrosion and leakage. **C,** Lingual view indicates coronal leakage. **D,** Corroded silver cone removed from the tooth. **E,** Posttreatment radiograph of the tooth.

A) Radiograph shows a tooth with two radiolucent patches on the crown. A radiopaque patch is visible on the central crown with a radiopaque canal. A radiopaque outline of the silver cone is visible.

B) Close-up of a frontal set of central incisors shows a black patch under labial frenulum.

C) Close-up of a posterior set of central incisors shows a part of the silver cone exposed from a thinned right incisor.

D) Close-up of a removed cylindrical cone with silver ends and black center.

E) Radiograph shows a tooth with segmented radiopaque crown, radiolucent neck and radiopaque canal.

With the introduction of rigid silver cones, it became possible to easily place them to length. This resulted in clinicians often failing to properly clean

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and shape RCS before obturation. Treatment failures were the result of leakage and failure to remove the irritants from the RCS. The use of silver cones today is considered to be below the standard of care in contemporary endodontic practice. For further information about this material and technique, the reader is referred to Chapter 27.

Gutta-percha

GP is the most popular core material used for obturation. Major advantages of GP are its plasticity, ease of manipulation, minimal toxicity, radiopacity, and ease of removal with heat or solvents. Disadvantages include its lack of adhesion to dentin and, when heated, shrinkage upon cooling. GP is the *trans*-isomer of polyisoprene, natural rubber, and exists in two crystalline forms, α and β.[108] In the unheated β phase, the material is a solid mass that is compactable. When heated, the material changes to the α phase and becomes pliable and tacky and can be made to flow when pressure is applied. A disadvantage to the α phase is that the material shrinks on setting.[257]

GP cones consist of approximately 20% GP, 65% zinc oxide, 10% radiopacifiers, and 5% plasticizers.[96] Attempts have been made to make GP more antimicrobial, but the clinical effectiveness of adding these materials has not been demonstrated. Moreover, to exert an antimicrobial pharmacological effect, the active ingredient must leach out of the GP, which could have a detrimental effect on long-term sealability.

Unlike rubber, room temperature GP cannot be compacted or made to flow. Compaction results in transmission of forces to the material and the canal wall equally and may result in root fracture. GP can be made to flow if it is modified by either heat or solvents such as chloroform. This permits adaptation to the irregularities of the canal walls.

The α form of GP melts when heated above 65°C. When cooled extremely slowly, the α form will recrystallize. Routine cooling results in the recrystallization of the β form. Although the mechanical properties for the two forms are the same, when α-phase GP is heated and cooled, it undergoes less shrinkage, making it more dimensionally stable for thermoplasticized techniques. The use of α-phase GP for obturation has increased as thermoplastic techniques have become more common.

GP cones are available in standardized and nonstandardized sizes.

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Standardized sizes conform to requirements contained in specification published by the International Organization of Standardization (ISO) or ADA American National Standards Institute (ADA ANSI). The nonstandard nomenclature refers to the dimensions of the tip and body (Fig. 9.23). A fine-medium cone has a fine tip with a medium body. Standardized cones are designed to match the taper of stainless steel and nickel titanium instruments (Figs. 9.24 and 9.25). A size 40, 0.04 cone has a tip of 0.4 mm and a taper of 0.04 mm per millimeter. Unfortunately, ISO and ADA ANSI standards allow tolerances and, coupled with less-than-completely accurate manufacturing processes, the actual cone size varies as does the tip and taper of the master apical file.[106]

FIG. 9.23 Nonstandard GP cones: extra fine, fine fine, fine, medium fine, fine medium, medium, large, and extra-large.

Seven gutta percha cones of different sizes with elongated bodies.

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FIG. 9.24 **A,** Standard GP cone sizes #15 to #40. **B,** Standard cones #.06, taper sizes #15 to #40. **C,** Standard cones ProTaper F1, F2, and F3.

A) Six gutta percha standard cones with elongated color-coded bodies; broader bases and narrower tips.

B) Six gutta percha standard cones with stout bodies; broader bases and narrower tips.

C) Three gutta percha standard cones with color-coded bodies; broader bases and narrower tips.

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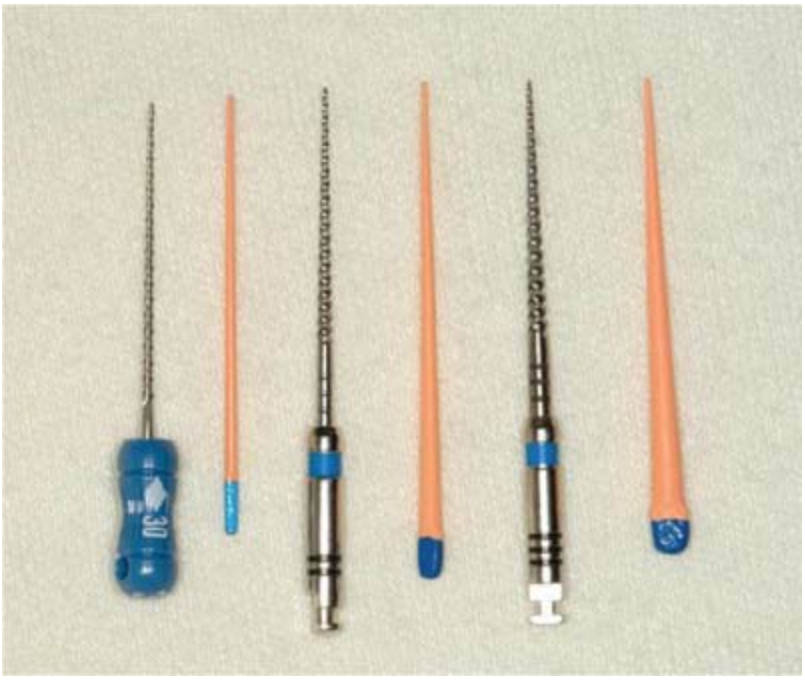

FIG. 9.25 Size #30 standard GP points exhibiting #.02, #.04, and #.06 tapers.

A set of three spirally textured tapers and three gutta percha points of different sizes.

Although GP points cannot be heat sterilized, a study reported that GP points can be sterilized by placing in 5.25% NaOCl for 1 minute. This study also reported that 2% glutaraldehyde, 2% chlorhexidine, and 70% ethyl alcohol were not effective in killing *Bacillus subtilis* spores, one of the hallmark organisms used for testing effective antimicrobial effectiveness.[270]

Activ GP

Activ GP (Brasseler USA) consists of GP cones impregnated on the external surface with glass ionomer (see Fig. 7.14). Single cones are used with a glass ionomer sealer. Available in 0.04 and 0.06 taper cones, the sizes are laser-verified to help ensure a more precise fit. The single cone technique is designed to provide a bond between the dentinal canal wall and the master cone. A bacterial leakage study comparing Activ GP/glass ionomer sealer, Resilon/Epiphany, and GP/AH Plus demonstrated no statistically significant differences in leakage at 65 days.[95]

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Custom cones

When the apical foramen is excessively large or the prepared RCS is large, a custom cone may need to be fabricated (Fig. 9.26). This permits the adaptation of the cone to the canal walls, reduces the potential for extrusion of the core material, and may improve the resultant seal.[26,180] The technique involves selection of a master cone and fitting that cone 2 to 4 mm short of the prepared length with frictional resistance. The cone is grasped with locking cotton pliers or a hemostat so that it can be placed into the RCS in the same spatial relationship each time. The tip of the GP cone is removed and the resultant new tip is softened in chloroform, eucalyptol, or halothane for 1 or 2 seconds, depending on the clinical requirements. Only the outer superficial portion of the cone is softened. The central core of the cone should remain semirigid. The cone is then placed back into the RCS to the working length. The process can be repeated until an adequate impression of the canal is obtained at the prepared length. A radiograph is exposed to verify proper fit and position. An alternative to solvents is softening with heat.[145]

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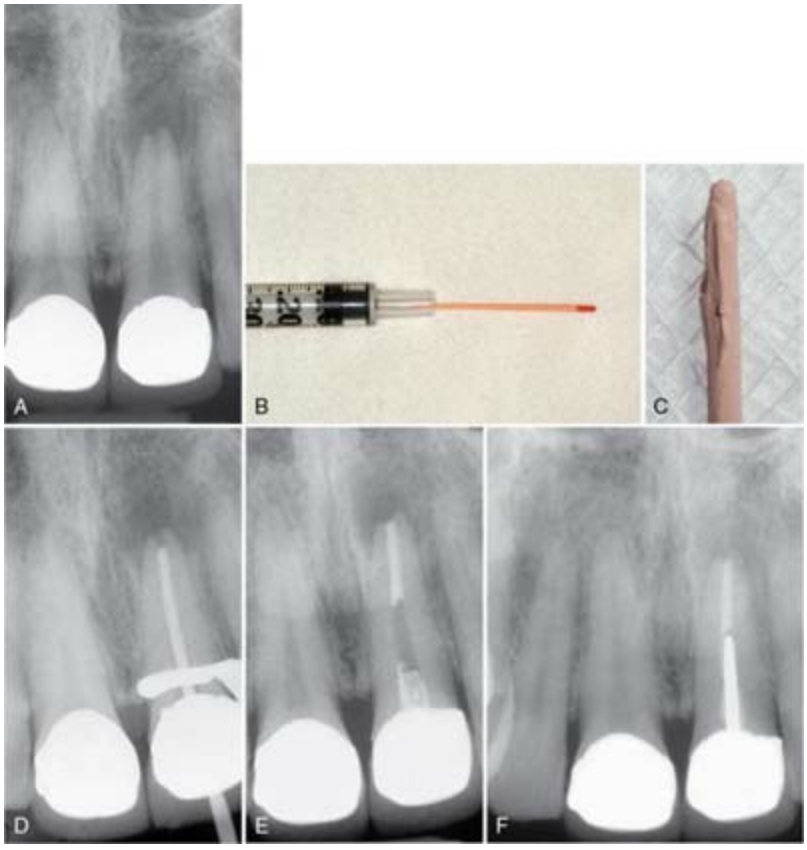

FIG. 9.26 Apical root resorption often results in an open apex requiring fabrication of a custom cone. **A,** Pretreatment radiograph of a maxillary left central incisor with pulp necrosis and chronic apical periodontitis. Apical root resorption is present. **B,** In fabricating a custom master cone, a GP point is fit several millimeters short before softening in solvent and tamping into place. **C,** Softening the apical 2 to 3 mm in chloroform that has been placed in a tuberculin syringe. **D,** The completed custom cone represents an impression of the apical portion of the RCS. **E,** The posttreatment radiograph with post space prepared. **F,** A 1-year follow-up radiograph demonstrating osseous regeneration.

A) Radiograph shows two teeth with white circular radiolucent patches over the crowns.

B) Close-up of a tubeculin syringe with a gutta percha cone tip.

C) Close-up of an irregular cone shows the shape of the apical portion of the root canal system.

D) Radiograph shows a tooth shows a radiopaque crown. An radiopaque outline of the dental file and clamp is visible.

E) Radiograph shows a tooth with a white circular radiolucent patch over the crown, radiopaque upper canal, dark middle canal and radiopaque lower canal.

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F) Radiograph shows a tooth with a radiopaque crown, radiopaque upper canal and radiolucent lower canal.

A large RCS may necessitate the custom fabrication of a large master cone before canal adaptation. This may be accomplished by heating two, or more, large GP cones and rolling the mass between two glass slabs until an appropriate size is obtained (Fig. 9.27). A spatula may also be used to shape the cone.

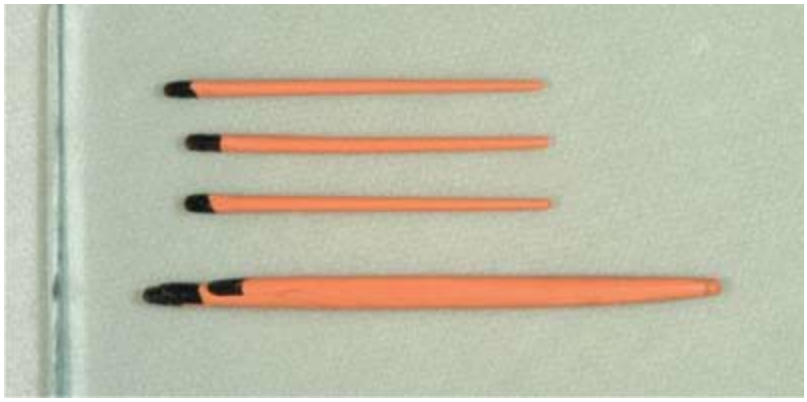

FIG. 9.27 For large RCS, several GP points can be heated and rolled together, using a spatula or two glass slabs.

A set of three smaller and one large gutta percha points.

Methods of obturation

To date, little evidence exists to support one method of obturation as being superior to another based on outcome assessment investigations.[14,195] The prospective Toronto studies have suggested that warm vertical compaction may be superior to lateral compaction.[73] However, definitive evidence is lacking.[218]

Lateral compaction

Lateral compaction is a common method for obturation (Figs. 9.28 and 9.29).[50] The technique can be used in most clinical situations and provides for predictable length control during compaction.[103] A disadvantage is that

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the technique may not fill canal irregularities[315] as well as warm vertical compaction or other thermoplastic techniques.[309] The procedure can be accomplished with any of the acceptable sealers.

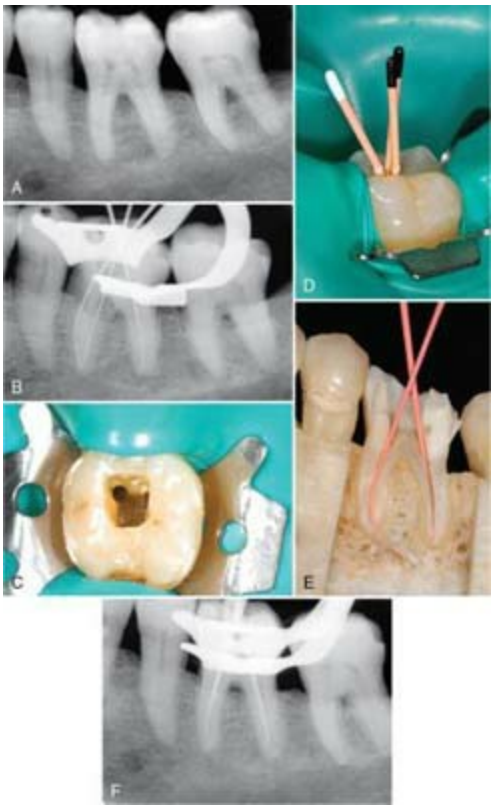

FIG. 9.28 Left first mandibular molar. **A,** Pretreatment radiograph. **B,** Working length radiograph. **C,** Coronal access opening, demonstrating the prepared mesiobuccal canal. **D,** Standardized master cones with coronal reference marked. **E,** Standard master cones fit to length as they exhibit minimal taper and permit deeper penetration of the spreader. **F,** Master cone radiograph.

A) Radiograph shows a tooth with a radiolucent crown, neck and pulp. A dark outline of canals is visible.

B) Radiograph shows a tooth with a radiolucent crown, neck and pulp. A radiopaque outline of three files and a clamp is visible.

C) Close-up of horizontal section of tooth held in a clamp shows an open crown exposing the canal.

D) Close-up of lateral section of a tooth held in a clamp shows an open crown with three gutta percha points inserted in the cavity.

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E) Close-up of cutaway section of a tooth shows two gutta percha points inserted in the canal cavity.

F) Radiograph shows a tooth with a radiopaque crown and canals with radiolucent pulp. An outline of clamp and gutta percha points are visible.

FIG. 9.29 Lateral compaction. **A,** Finger spreader in place. **B,** Fine-medium accessory cone placed in the space created by the spreader. **C,** Finger spreader placed in preparation, creating space for additional accessory cones. **D,** Additional cones are placed until the spreader does not penetrate past the coronal one third of the canal. The cones are then removed at the orifice with heat, and the coronal mass is vertically compacted with a plugger. **E,** Interim radiograph may be exposed to assess the quality of obturation. **F,** Posttreatment radiograph demonstrating adequate length, density, and taper. The gutta-percha is removed to the level of the orifice, and a coronal seal has been established with an adequate provisional restoration.

A) Close-up of lateral section of a tooth held in a clamp under a rubber dam shows an open crown with a gutta percha point and a taper inserted in the cavity.

B) Close-up of lateral section of a tooth held in a clamp under a rubber dam

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shows an open crown with two gutta percha points inserted in the cavity.

C) Close-up of lateral section of a tooth held in a clamp under a rubber dam shows an open crown with two gutta percha points and a taper inserted in the cavity.

D) Close-up of lateral section of a tooth held in a clamp under a rubber dam shows an open crown with four gutta percha points inserted in the cavity.

E) Radiograph shows a tooth with radiolucent crown and radiopaque canals. A radiopaque outline of clamp and gutta percha points are visible.

F) Radiograph shows a tooth with radiopaque crown, radiolucent neck and radiopaque canals.

After RCS preparation, a standard cone is selected that has a tip and taper consistent with the prepared canal diameter at the working length established by the master apical file. Standard cones generally have less taper when compared with nonstandard cones and will permit deeper spreader penetration, which will result in a better-quality resultant seal.[220] An alternative is to adapt an appropriately tapered nonstandard cone by cutting small increments from the tip. This “master cone” is measured and grasped with forceps so that the distance from the tip of the cone to the reference point on the forceps is equal to the prepared length. A reference point on the cone can be made by pinching the cone. The cone is placed into the RCS and, if an appropriate size is selected, there will be resistance to displacement or “tug back.” If the cone is loose, it can be adapted by removing small increments from the tip. If the master cone fails to go to the prepared length a smaller cone can be selected. Devices are available to cut cones accurately at a predetermined length (Tip Snip; SybronEndo). When the cone extends beyond the prepared length, a larger cone must be adapted or the existing cone shortened until there is resistance to displacement at the corrected working length.

The master cone placement is confirmed with a radiograph. The canal is irrigated and dried with paper points. Sealer is applied to the canal walls and a spreader is prefit so as to allow it to be inserted to within 1 to 2 mm of the working length. Appropriately sized accessory points are also selected to closely match the size of the spreader to be used. The correlation between spreader size and nonstandard cones is variable,[43,322]

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Finger spreaders may provide better tactile sensation and are less likely to induce fractures in the root when compared with the more traditional D-11T hand spreader.[164] In addition to the type of spreader, forces applied, and amount of dentin removed, spreader size may be a factor in root fracture, with large sizes inducing more stress.[225] Spreaders made from nickel titanium are available and provide increased flexibility,[30] reduced stress,[101] and deeper penetration when compared with stainless steel instruments.[258] The spreader should fit to within 1 to 2 mm of the prepared length, and, when introduced into the RCS with the master cone in place, it should be within 2 mm of the working length.[8] There appears to be a correlation between the establishment of a higher quality seal and the depth of spreader penetration.[8]

After the spreader has been placed to its maximum depth, it is removed by rotating it back-and-forth as it is withdrawn. An accessory cone is placed in the space vacated by the spreader. The process is repeated until the spreader no longer goes beyond the coronal one third of the canal. The excess GP is removed with heat and the coronal mass is compacted with an appropriate unheated plugger. Only light pressure is required during lateral compaction because the GP is not condensed, and because as little as 1.5 kg of pressure is capable of fracturing a root (Fig. 9.30). In addition to the force applied, investigators have noted that removal of excessive amounts of dentin during preparation is a significant factor in root fracture.[307]

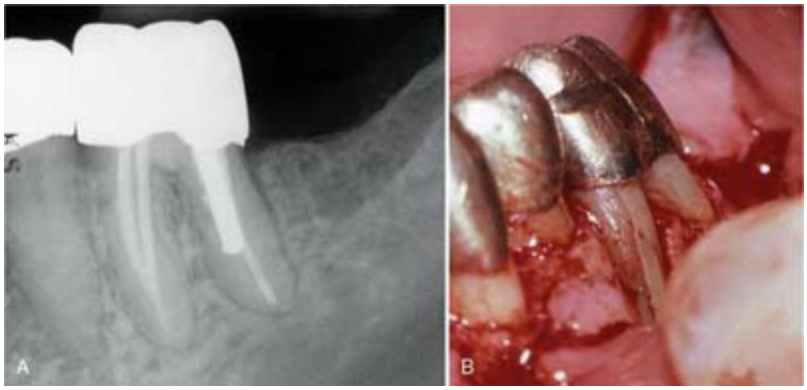

FIG. 9.30 Vertical root fractures can occur with excessive compaction forces. **A,** Follow-up radiograph of a mandibular left first molar. A deep isolated periodontal probing defect was associated with the buccal aspect of the mesiobuccal root. **B,** Flap reflection revealed a vertical root fracture.

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A) Radiograph shows a tooth with radiopaque crown, radiolucent segmented left canal and radiopaque upper canal and radiolucent lower canal.

B) Close-up of lateral section of teeth covered with silver caps with exposed roots.

A disadvantage to lateral compaction is that the process does not produce a homogeneous mass. The accessory and master cones are laminated and remain separate. It is envisaged that the space between each of the cones is filled with sealer to aid in establishment of a water-tight seal

The excess GP in the chamber is then seared off and vertically compacted with a heated plugger at the orifice or approximately 1 mm below the orifice in posterior teeth. In anterior teeth, the desired level is the cementoenamel junction on the facial surface to avoid aesthetic issues if the dentin should become stained. An alternative to lateral compaction with finger spreaders is the use of ultrasonics and a clinical study reported a 93% success rate.[321]

Warm vertical compaction

Schilder introduced warm vertical compaction of GP as a method of obturating the radicular space in three dimensions.[256] Preparation requirements for the technique include preparing an RCS with a continuously tapering funnel and keeping the apical foramen as small as possible.

Various ISO standardized instruments are also available (Fig. 9.31).

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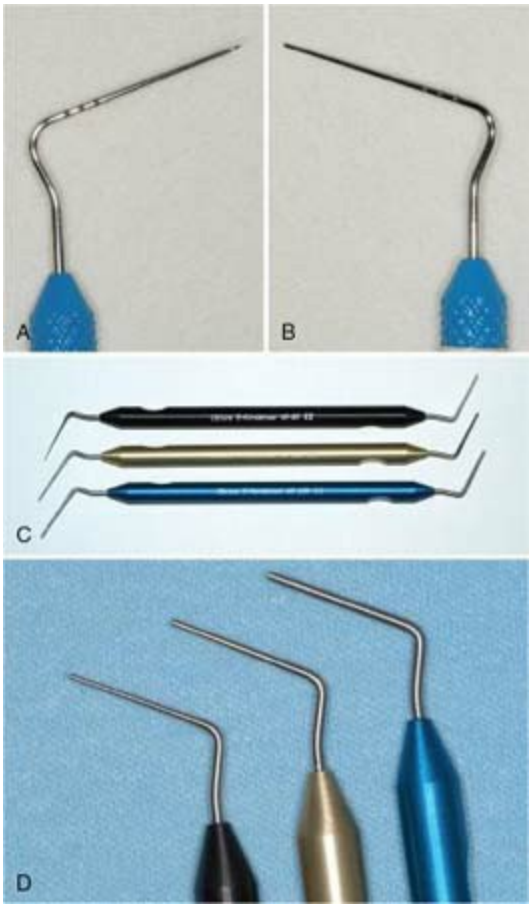

FIG. 9.31 Various pluggers are manufactured for compacting warm GP. **A,** ISO standard spreader. **B,** ISO standard plugger. **C,** Obtura S Kondensers. **D,** Obtura S-Kondensers.

A) Close-up of standard spreader shows an angular body with spirally textured surface and a pointed tip.

B) Close-up of standard plugger shows an angular body with spirally textured surface and a flat tip.

C) An image of a set of three double sided kondensers with angular pointed and flat tips.

D) Close-up of three double sided kondensers exposing the upper side with angular spirally textured flat tips.

Source: (***C,*** Courtesy Obtura Spartan, Earth City, MO.)

The technique involves fitting a master cone 0.5 to 2 mm short of the prepared working length with resistance to displacement (Fig. 9.32). This ensures that the cone diameter is larger than the prepared canal at the terminus. Nonstandard cones that closely replicate the canal taper are best

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because they permit the development of hydraulic pressure during compaction. After the adaptation of the master cone, it is removed and sealer is applied to the cone and the walls of the prepared canal. The cone is placed in the canal and the coronal portion is removed with a heated instrument. A heated spreader or plugger is used to remove portions of the coronal GP in successive increments and soften the remaining material in the canal. The Touch 'n Heat (Kerr) (Fig. 9.33) and System B (Kerr Dental) (Fig. 9.34) are alternatives to applying heat with a flame-heated instrument because they permit improved temperature control. A plugger is inserted into the RCS and the GP is compacted, forcing the plasticized material apically. The process is repeated until the apical portion has been reached. The coronal RCS is back-filled using small preheated pieces of GP. The sectional method consists of placing 3 to 4 mm sections of GP approximating the size of the canal into the root, applying heat, and compacting the mass with a plugger.

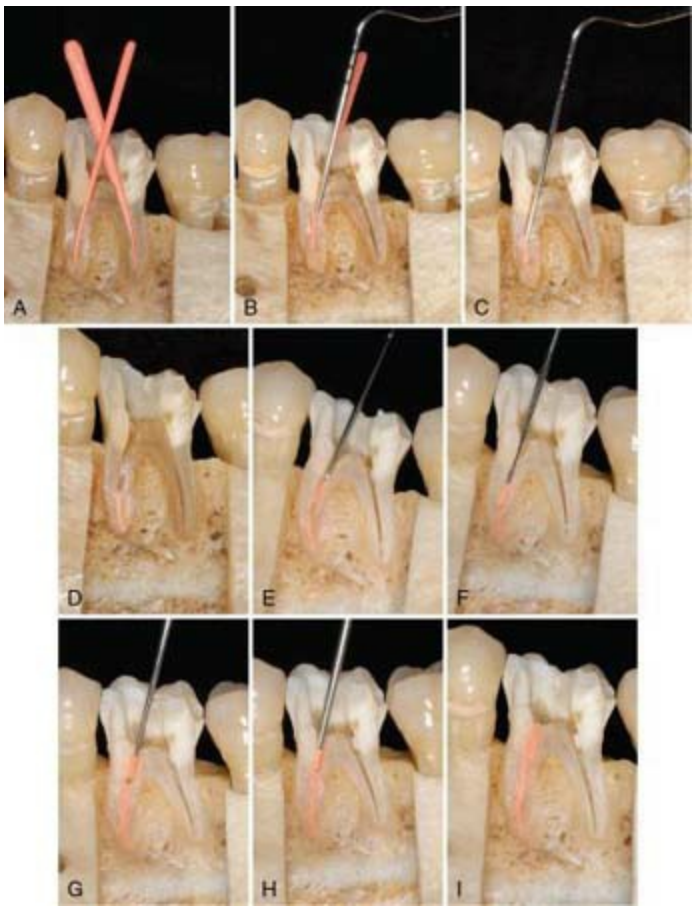

FIG. 9.32 Warm vertical compaction of GP employs heat and various

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condensers. **A,** Nonstandard cones are selected and fit short of the prepared length because they more closely replicate the prepared canal. **B,** Heated pluggers or spreaders are used to apply heat to the master cone and remove the excess coronal material. **C,** A room temperature plugger is used to compact the heated GP. **D,** Apical compaction is complete. **E,** A GP segment is placed in the canal, and heat is applied. **F,** The heated segment is compacted. **G,** The process is repeated for the coronal portion of the canal by placing and heating a segment of GP. **H,** A plugger is again used to compact the heated material. **I,** Completed obturation.

A) Close-up of lateral section of cutaway tooth shows an open crown with two gutta percha points inserted in two canals.

B) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha point and a spreader inserted in the left canal.

C) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha segment in the lower canal and a spreader inserted in the left upper canal.

D) Close-up of lateral section of cutaway tooth shows an open crown with a vertical gutta percha segment in the lower canal.

E) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha segment in the lower canal and a spreader in upper canal.

F) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha segment being compacted by a spreader inside the lower left canal.

G) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha segment being compacted by a spreader inside the upper left canal.

H) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha segment being compacted by a spreader inside the upper left canal.

I) Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha inserted into the left canal.

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FIG. 9.33 Touch 'n Heat unit.

A touch and heat device with a connected heat carrier pen with tip depicting a probe.

Source: (Courtesy SybronEndo, Orange, CA.)

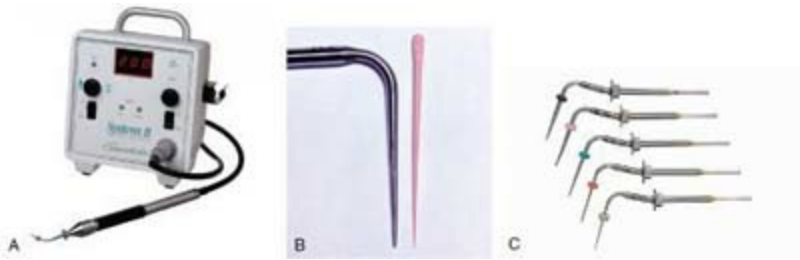

FIG. 9.34 Continuous wave obturation uses the System B unit. **A,** The System B unit. **B,** System B plugger with a nonstandard cone of similar taper. **C,** System B pluggers.

A) Close-up of a system B unit shows obturation unit connected to a safety handpiece.

B) Close-up of angular system B pointed plugger with a tapered cone.

C) Close-up of five angular system B pointed tips and pluggers.

Source: (Courtesy SybronEndo, Orange, CA.)

One study measured temperature changes in an RCS with warm vertical

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compaction.[34] The maximal temperatures occurred coronally and decreased apically. The authors reported that the maximal temperature in the canal was 118°C 8 mm from the apex. At 0 to 2 mm from the apical terminus, the maximal temperature had decreased to 44°C. Another study compared root surface temperatures for warm vertical obturation using the System B heat source, the Touch 'n Heat device, and a flame-heated carrier in maxillary and mandibular incisors and premolars 2 mm below the cementoenamel junction. System B and the Touch 'n Heat produced a surface temperature rise that was less than 10°C for all maxillary incisors and premolar teeth. The Touch 'n Heat produced a greater than 10°C rise in mandibular incisors. The flame-heated carrier produced temperature changes greater than 10°C in all experimental teeth. Because the critical level of root surface heat required to produce irreversible bone damage is believed to be greater than 10°C, the findings suggest that warm vertical compaction with the System B should not damage supporting periodontal structures. However, caution should be exercised with the Touch 'n Heat and flame-heated carriers.[161] The flame-heated spreader has no heat controls and the Touch 'n Heat controls only send heat to the tip and receive no feedback from the tip based on clinical conditions. The System B has a built-in computer that allows it to receive feedback from the tip and adjust the tip temperature based on the clinical conditions, which allow the tip to remain at the dialed-in temperature.

The potential for vertical root fracture is also present with warm vertical compaction.[33] The forces developed appear to be equal to lateral compaction. Investigators compared warm vertical compaction and lateral compaction as a function of time. Results indicated that the forces developed with the two techniques were not significantly different. In a follow-up study, the mean value for wedging with warm vertical compaction was 0.65 ± 0.07 kg, whereas for lateral compaction it was 0.8 ± 0.1 kg.

Warm thermoplastic techniques have the advantage of producing movement of the plasticized GP within the obturated RCS, creating a more homogeneous mass of resultant GP and filling irregularities and accessory canals better than lateral compaction.[311] This was illustrated in a study that reported a correlation between the quality of adaptation and the depth of heat application and canal size. Heat application close to the apical extent of the preparation produced the best results, and adaptation was better in small RCS

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when compared with wider ones.[310] However, thermoplasticized techniques resulted in more extrusion of obturating materials.[154] There appeared to be no consistent differences between the techniques in sealing the RCS.[311] Warm vertical compaction also influenced setting time and flow of sealers.[228]

Advantages of warm vertical compaction include filling of canal irregularities and accessory canals. Disadvantages include a slight risk of vertical root fracture because of compaction forces, less length control than with lateral compaction, and the potential for overextension of obturation materials into the periradicular tissues. Warm vertical compaction is also difficult in more curved RCS where the rigid pluggers may be unable to penetrate to the necessary depth. Occasionally, the RCS have to be enlarged and tapered more to enable rigid carriers to penetrate within 4 to 5 mm of the working length. Additional removal of RCS dentin can weaken the root, making it more susceptible to root fracture.

Continuous wave compaction technique

A variation of warm vertical compaction is the continuous wave compaction technique.[45] The increasing use of nickel titanium rotary preparation techniques and the fabrication of greater taper standard cones have resulted in more clinicians using thermoplasticizing techniques. The manufacturing of GP cones to mimic the tapered preparation permits the application of greater hydraulic force during compaction when appropriately tapered pluggers are used. The continuous wave compaction technique employs the System B connected to 0.04, 0.06, 0.08, 0.10, or 0.12 tapered stainless steel dead-soft pluggers (see Fig. 9.34). The 0.06 tapered plugger also approximates the fine nonstandard GP cone, while the 0.08 plugger approximates the fine-medium cone, the 0.10 plugger the medium cone, and the 0.12 plugger the medium-large cone. Pluggers are consistent with the ProFile GT instruments (DENTSPLY Sirona), and Autofit GP cones (Kerr Endo) are also available. In summary, after selecting an appropriate master cone, a series of pluggers from large to small are prefitted to finally reach within 5 to 7 mm of the prepared length (Fig. 9.35).[45]

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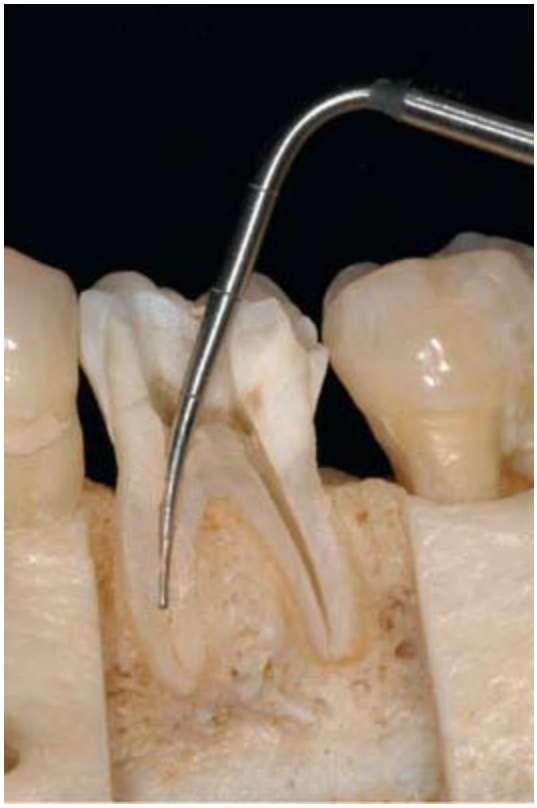

FIG. 9.35 System B plugger fit.

Close-up of lateral section of cutaway tooth shows an open crown with a system B plugger tip in the lower left canal.

The continuous wave and warm backfill technique cause significantly higher temperatures than other obturation techniques by virtue of the technique.[80] This occurs because the plugger of the System B unit is inserted into the canal orifice and activated to remove excess coronal material, while the series of compaction and removal of GP takes place until the plugger is moved rapidly (1 to 2 seconds) to within 3 mm of the binding point (Fig. 9.36). The continuous wave technique was reported to have higher temperatures at 3 mm and 6 mm from the apex than the warm backfill technique.[80]

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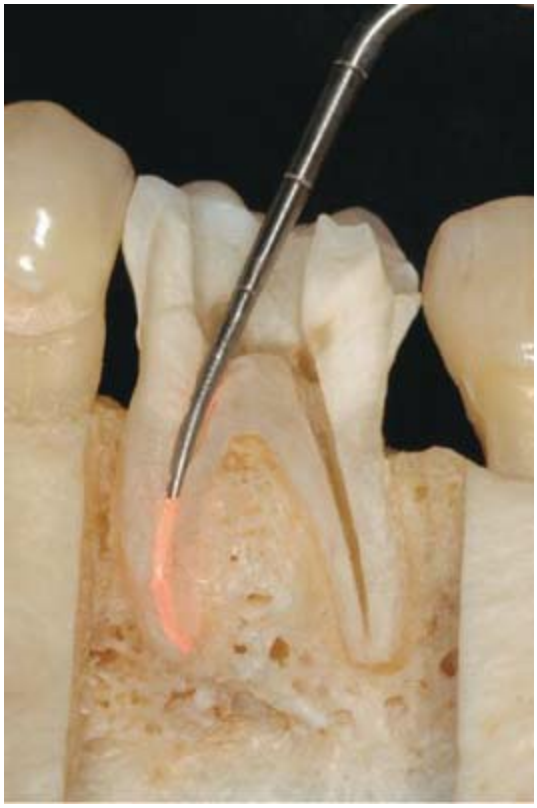

FIG. 9.36 System B activation and compaction.

Close-up of lateral section of cutaway tooth shows an open crown with a compacted gutta percha segment in the lower canal and a plugger tip inserted in the left upper canal.

It should be noted that with the continuous wave technique, the tip of the heated plugger is placed only to within 5 to 7 mm from the tip of the GP. The apical portion of the GP remains essentially a single-cone technique as the heat transfer does not take place in the apical 2 to 5 mm of the GP.[109]

In ovoid canals, where the canal configuration may prevent the generation of hydraulic forces, an accessory cone can be placed alongside the master cone before compaction. With type II canals, the master cones are placed in both canals before compaction. A hand plugger is used to stabilize the cone in one canal while the other is being obturated.

Filling the space left by the plugger may be accomplished by a thermoplastic injection technique (Obtura II or Ultrafil 3D [Coltène/Whaledent], Calamus [DENTSPLY Sirona Dental Specialties], Elements free [Kerr Dental], or HotShot [Discus Dental, Culver City, CA]) (Fig. 9.37)[138] or by fitting an accessory cone into the space with sealer,

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heating it, and compacting by short applications of heat and vertical pressure.

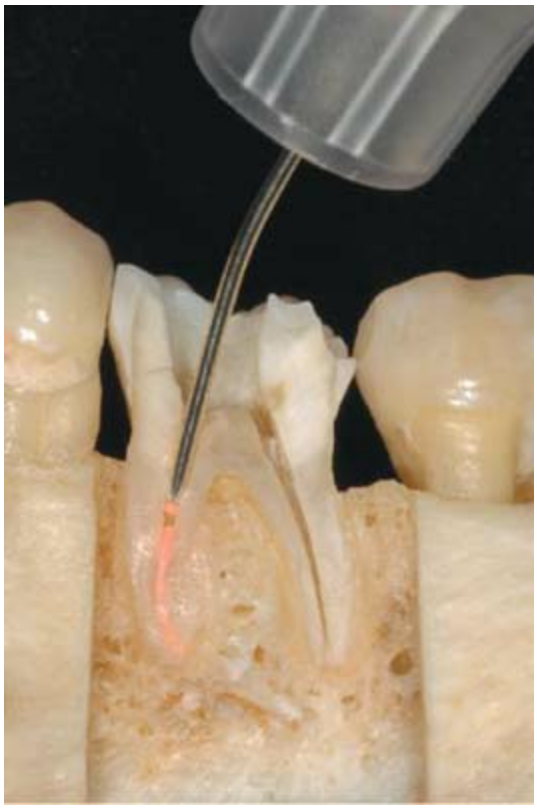

FIG. 9.37 Backfill by the Obtura II thermoplastic injection technique.

Close-up of lateral section of cutaway tooth shows an open crown with a compacted gutta percha segment in the lower canal and obituary two thermoplastic injection needle inserted in the left upper canal.

Thermoplastic injection techniques

Heating of GP outside the tooth and injecting the material into the canal is an additional variation of a thermoplastic technique (Fig. 9.38). The Obtura III (Fig. 9.39), Calamus (Fig. 9.40), Elements (Fig. 9.41), HotShot (Fig. 9.42), and Ultrafil 3D (Fig. 9.43) are available devices. The Obtura II system heats the GP to 160°C, whereas the Ultrafil 3D system employs a low-temperature GP that is heated to 90°C.

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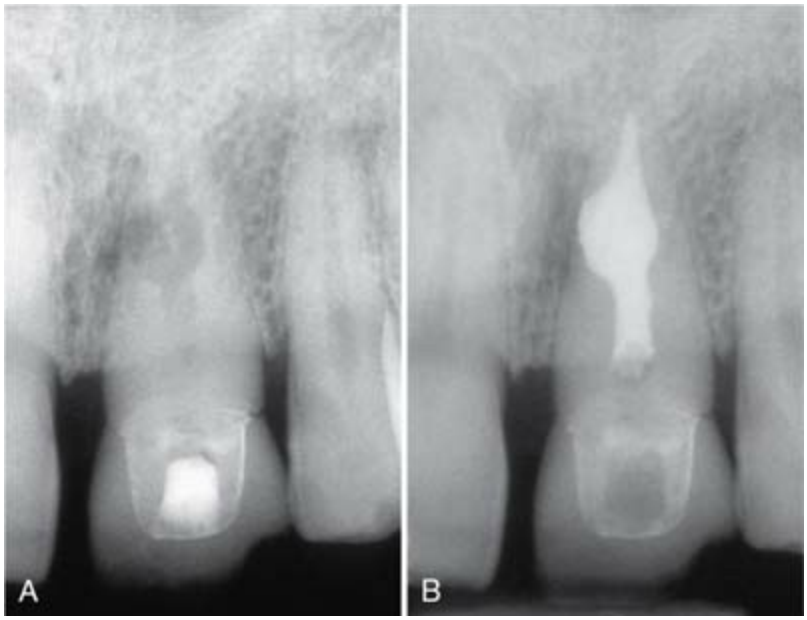

FIG. 9.38 Thermoplastic techniques are often used in cases with significant canal irregularities. **A,** A pretreatment radiograph of a maxillary central incisor exhibiting internal resorption. **B,** Posttreatment radiograph demonstrates a dense obturation of the resorptive defect with GP.

A) Radiograph shows a tooth with radiopaque square patch on the crown with an outline of radiolucent patch.

B) Radiograph shows a tooth with radiolucent outlined square patch on the crown with a radiopaque canal with an inflamed middle part.

FIG. 9.39 Obtura III unit with silver tips, GP plugs, and cleaning

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solution (Obtura Spartan, Earth City, MO).

An Obtura system shows a control unit, gutta percha plugs, two sizes of applicator needles and a hand piece.

FIG. 9.40 The Calamus thermoplastic unit (DENTSPLY Tulsa Dental Specialties, Tulsa, OK) for heating and injecting GP.

Close-up of the calamus thermoplastic unit shows a calamus dual console, pack and flow hand pieces, three heat pluggers, cartridge nut, cleaning brush, bending tool and a heat shield.

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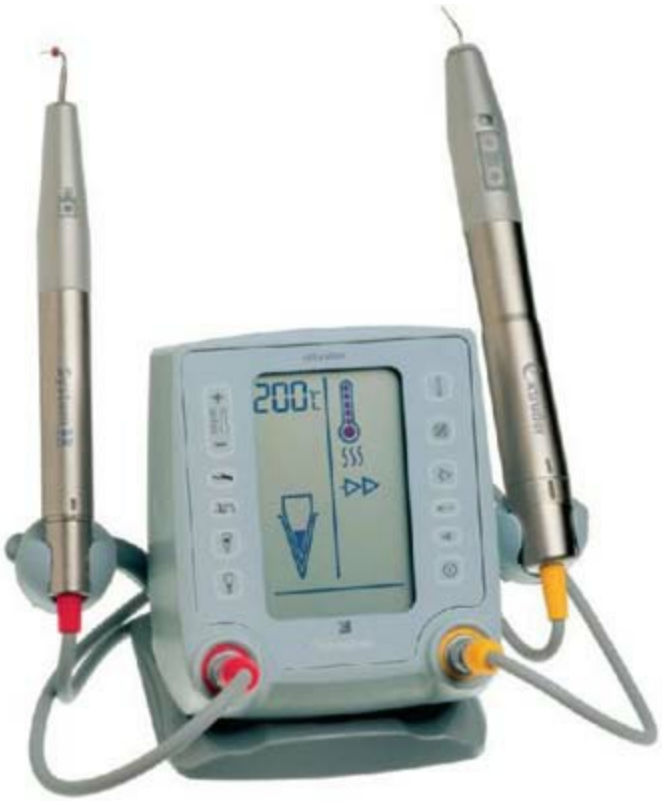

FIG. 9.41 The Elements obturation unit (Kerr Endo, Orange, CA) for injecting and compacting GP. Note the System B heat source.

Close-up of the elements obturation system shows a elements obturation unit with motor driven extruder hand pieces.

FIG. 9.42 The battery-powered HotShot unit (Discus Dental, Culver

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City, CA) for heating and injecting GP.

Close-up of the hotshot system with gun shaped hotshot unit with three plain textured needles with flat tips.

FIG. 9.43 The Ultrafil 3D system consists of an injection syringe, GP cannulas, and heating unit (Coltène/Whaledent, Cuyahoga Falls, OH).

Close-up of ultrafil three-dimensional system shows a heating unit, a gun-shaped injection syringe and gutta percha cannula tubes.

Obtura III

The Obtura III system (Obtura Spartan, Earth City, MO, USA) consists of a handheld "gun" that contains a chamber surrounded by a heating element into which pellets of GP are loaded (see Fig. 9.40). Silver needles (varying gauges of 20, 23, and 25) deliver the thermoplasticized material into the RCS. The control unit allows the operator to adjust the temperature and thus the viscosity of the GP. At 6 mm from the apex, one study reported that the highest internal temperature with the Obtura system was 27°C.[283]

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Canal preparation is similar for other obturation techniques. The apical terminus should be as small as possible to prevent overextension of GP. The technique requires the use of sealer and, once the canal is dried, the canal walls are coated with sealer using the last file used to length or a paper point. GP is preheated in the gun and the needle is positioned in the RCS so that it reaches within 3 to 5 mm of the apical preparation. GP is then gradually, passively injected by squeezing the trigger of the "gun." The needle backs out of the canal as the apical portion is filled. Room-temperature prefittted pluggers, dipped in alcohol, are used to compact the GP. A segmental technique may also be used, in which 3- to 4-mm segments of GP are sequentially injected and compacted. In either case, compaction should continue until the GP cools and solidifies to compensate for the contraction of the GP that takes place on cooling.

The difficulties with this system include lack of length control. Both overextension and underextension are common results. To overcome this drawback, a hybrid technique may be used in which the clinician begins obturating the RCS by a lateral compaction technique. When the master cone and several accessory cones have been placed so that the mass is firmly lodged in the apical portion of the canal, a hot plugger is introduced, searing the points off approximately 4 to 5 mm from the apex. Light vertical compaction is applied to restore the integrity of the apical plug of GP. The remainder of the canal is then filled with thermoplasticized GP injected as previously described, and the risk of overextension of GP is reduced.

Investigators studied, at 3, 6, and 12 months posttreatment, the healing rate of 236 teeth obturated with the Obtura system. Results indicated that 96% of the cases healed with the highest healing rate being in teeth filled flush with the radiographic apex (97%) when compared to overextensions (93%) and filling short (93%).[228]

Ultrafil 3D

Ultrafil 3D (Coltène/Whaledent) is a thermoplastic GP injection technique involving GP cannulas, a heating unit, and an injection syringe (see Fig. 9.43). The system employs three types of GP cannulas. Regular Set is a low-viscosity material that requires 30 minutes to set. Firm Set is also a low-viscosity material but differs in that it sets in 4 minutes. The manufacturer

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recommends compaction after the initial set with both materials. Endoset has a higher viscosity and does not flow as well. It is recommended for techniques employing compaction and sets in 2 minutes. The heater is preset at 90°C and does not require adjustment.

Each cannula has a 22-gauge stainless steel needle that measures 21 mm in length. The needles can be precurved. Cannulas can be disinfected but are not designed for heat sterilization procedures. Heating time varies but, for a cold unit, it takes 10 to 15 minutes. In a warm heater the recommended time is 3 minutes. After removing the cannula from the heater, the needle should be placed on the hot part of the heater for several seconds. The GP remains able to flow for 45 to 60 seconds depending on the viscosity.

Calamus

The Calamus flow obturation delivery system (DENTSPLY Sirona) is a thermoplastic device equipped with a cartridge system with 20- and 23-gauge needles (see Fig. 9.40). The unit permits control of temperature and the flow rate. Pluggers are also available for use with the system. The 360-degree activation switch allows great tactile sensation during use.

Elements

The Elements obturation unit (Kerr Endodontics) consists of a System B heat source and plugger as well as a hand piece extruder for delivering thermoplastic GP or RealSeal from a disposable cartridge (see Fig. 9.42). The cartridges come with 20-, 23-, and 25-gauge needles for GP and 20- and 23-gauges for RealSeal.

HotShot

The HotShot delivery system (Discus Dental) is a cordless thermoplastic device that has a heating range from 150°C to 230°C (see Fig. 9.43). The unit is cordless and can be used with GP. Needles are available in 20, 23, and 25 gauges.

Carrier-based gutta-percha

Thermafil, profile GT obturators, GT series X obturators, and

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ProTaper universal obturators

Guttacore and Thermafil (DENTSPLY Sirona Dental Specialties) were introduced as GP obturation materials with a solid core (Fig. 9.44). Originally manufactured with a metal core and a coating of GP, the carrier was heated over an open flame. The technique was popular because the central core provided a rigid mechanism to facilitate the placement of the GP. Advantages included ease of placement and the pliable properties of the GP. Disadvantages were that the metallic core made placement of a post challenging and that retreatment procedures were difficult. Additionally, the GP was often stripped from the carrier, leaving the carrier as the obturating material in the apical area of the canal. Reports have stated that carrier-based systems resulted in minimal gaps and voids in the resulting obturation mass.[325] Other reports have stated that there were no differences in healing rates between carrier-based systems and lateral compaction of GP.[117]

FIG. 9.44 Thermafil carrier and size verifier (DENTSPLY Sirona, Tulsa, OK).

A) Close-up of a thermafill carrier with a spiral body and a pointed tip.

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B) Close-up of a thin cylindrical verifier with a gutta percha surface.

Changes in the carrier systems include the development of a plastic core coated with α-phase GP (Fig. 9.45) and a heating device that controls the temperature (Fig. 9.46). Obturators are designed to correspond to ISO standard file sizes, variable tapered nickel titanium rotary files, and ProFile GT and GT Series X nickel titanium rotary files (DENTSPLY Sirona). Size verifiers are available to aid in selection of the appropriate carrier and should fit passively at the working length (see Fig. 9.45).

FIG. 9.45 GT obturator and instrument (DENTSPLY Sirona, Tulsa, OK).

A) Close-up of a thin cylindrical GT obturator with a gutta percha surface.

B) Close-up of a thermafill carrier with a laterally textured upper body and a spiral lower body with a pointed tip.

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FIG. 9.46 The Thermafil oven with carrier in place (DENTSPLY Tulsa Dental Specialties, Tulsa, OK).

A thermafill obturation oven with a central radiopaque plastic core with two obturations over the top for heating gutta percha.

As with all techniques, sealer is required. Grossman formulation sealers and resin sealers consistent with AH-26 and AH Plus are acceptable. However, Tubli-Seal and Wach's paste are not recommended.

The carrier is placed in the heating device. When the carrier is heated to the appropriate temperature, the clinician has approximately 10 seconds to retrieve it and insert it into the RCS (Fig. 9.47). This is accomplished without rotation or twisting. Evidence suggests that the insertion rate affects the obturation. The fill length and obturation of irregularities increase with increasing insertion rates, and a rapid insertion rate enhances obturation.[165]

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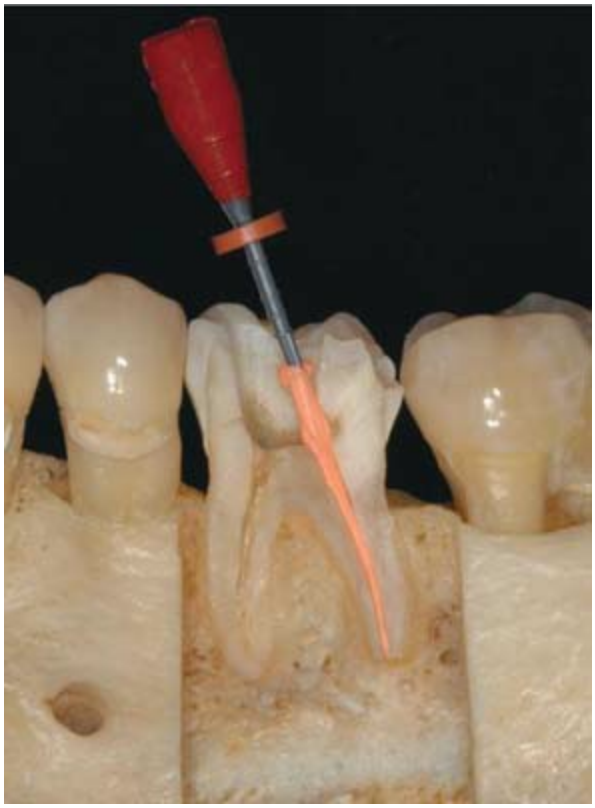

FIG. 9.47 Thermafil carrier placed in a distal canal.

Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha around a thermafill carrier inserted in the right canal.

Vertical compaction of the coronal GP can then be accomplished. When necessary, GP can be added, heat softened, and compacted. An advantage to this technique is the potential for movement of GP into the lateral and accessory canals (Fig. 9.48);[308] however, extrusion of material beyond the apical extent of the root anatomical apex is a disadvantage.[154,197]

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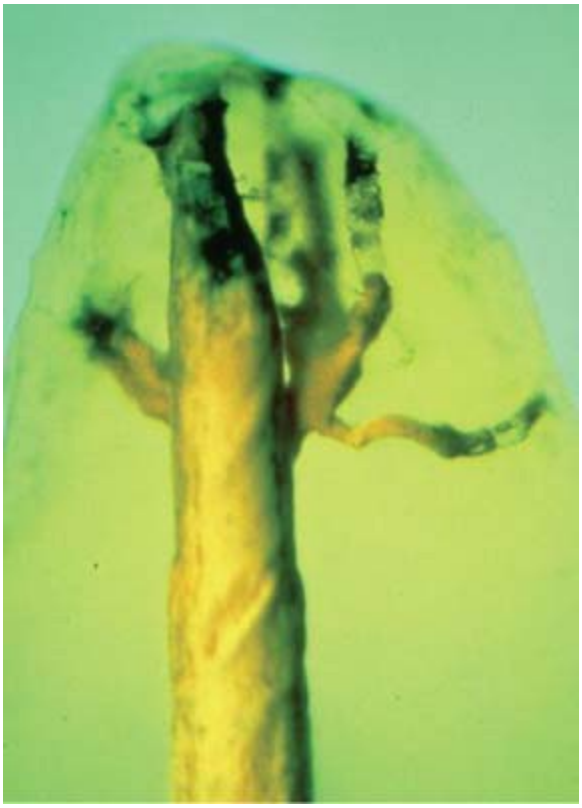

FIG. 9.48 Apical obturation of accessory canals by the Thermafil technique.

Micrograph of a lower segment of root shows a branched tip with ruptures around the outlines of the root.

Source: (Courtesy DENTSPLY Sirona, Tulsa, OK. All rights owned by and used with permission from DENTSPLY International, Inc.)

Pro-Post drills (DENTSPLY Sirona) are recommended if post space is required for restoration of the tooth. The unique eccentric cutting tip keeps the instrument centered in the canal while friction softens and removes the GP and plastic carrier.

When retreatment is necessary, the plastic carrier has a groove along its length to provide a potential access point for placement of a file to remove the carrier. Chloroform and hand files can be used to remove the GP surrounding the carrier. Rotary #.04 and #.06 nickel titanium files may also be used to remove the obturation materials. Retreatment rotary nickel titanium files are available in three different sizes to facilitate removal of GP and the carrier.

The plastic carriers are composed of two nontoxic materials. Sizes #20 to

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#40 are manufactured from a liquid crystal plastic. Sizes #40 to #90 are composed of polysulfone polymer. Both have similar physical characteristics, with the polysulfone carriers being susceptible to dissolution in chloroform.

GuttaCore (DENTSPLY Sirona), the latest generation of core carriers, uses cross-linked GP as the carrier of the outer thermoplasticized GP. This supposedly makes retreatment simpler as the clinician can simply drill through the carrier to gain access into the canal space. A recent in vitro study comparing the time required to remove GuttaCore, Thermafil Plus, and thermoplasticized GP from moderately curved RCS with ProTaper retreatment files reported that GuttaCore was removed in significantly less time when compared with the other two techniques.[25] The survival and healing rates after NS RCT with carrier-based obturation technique seem to be comparable to those previously reported for other obturation techniques.[223]

SuccessFil

SuccessFil (Coltène/Whaledent) is a carrier-based system associated with Ultrafil 3D (Fig. 9.49); however, the GP used in this technique comes in a syringe. Titanium or radiopaque plastic carriers are inserted into the syringe to the measured length of the canal. The GP is expressed on the carrier with the amount and shape determined by the rate of withdrawal from the syringe. Sealer is lightly coated on the canal walls, and the carrier with GP is placed into the RCS to the prepared length. The GP can be compacted around the carrier with various pluggers depending on the canal morphology. This is followed by severing of the carrier slightly above the orifice with a bur.

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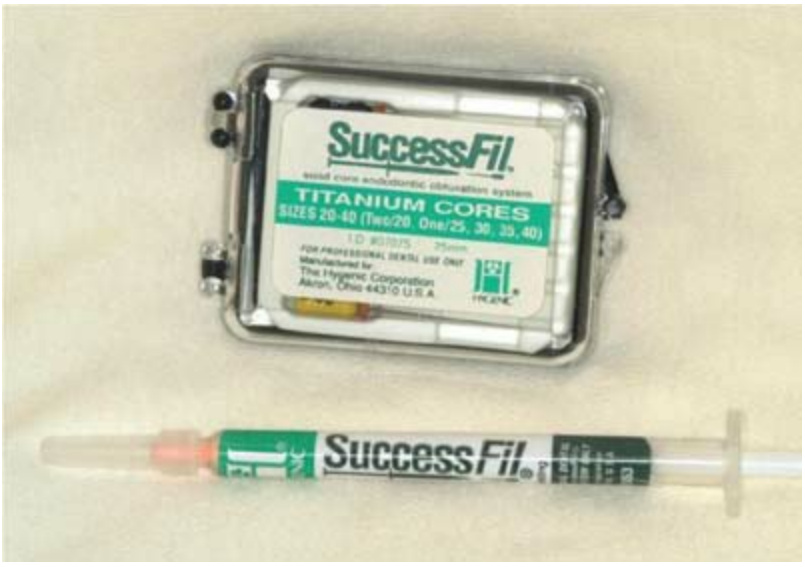

FIG. 9.49 SuccessFil is an additional carrier system (Coltène/Whaledent, Cuyahoga Falls, OH).

Close-up shows a package of titanium cores and a syringe with a plastic barrel, plunger and a cap

Simplifill

Simplifill (Kerr Endo) is GP manufactured for use after canal preparation with LightSpeed instruments (Fig. 9.50). The carrier has an apical plug with 5 mm of GP. The technique involves fitting a carrier that is consistent with the master apical rotary file to within 1 to 3 mm of the prepared length (Fig. 9.51). The apical GP plug can be modified by clipping the end in 1 mm increments to obtain an appropriate fit if the plug is too small. Once the cone is fitted, it is withdrawn and sealer applied to the canal walls. AH Plus is recommended. The Simplifill carrier is slowly advanced to the prepared length. This may require firm pressure. With the plug at the corrected working length, the handle is quickly rotated a minimum of four complete terms in a counterclockwise direction to separate the shaft from the apical GP. The coronal space can then be filled with GP using lateral compaction or a warm thermoplastic technique. When using lateral compaction, it is recommended that the first cone be the same size as the Simplifill carrier. This sectional technique is efficient and leakage potential is similar to that of other common techniques.[247]

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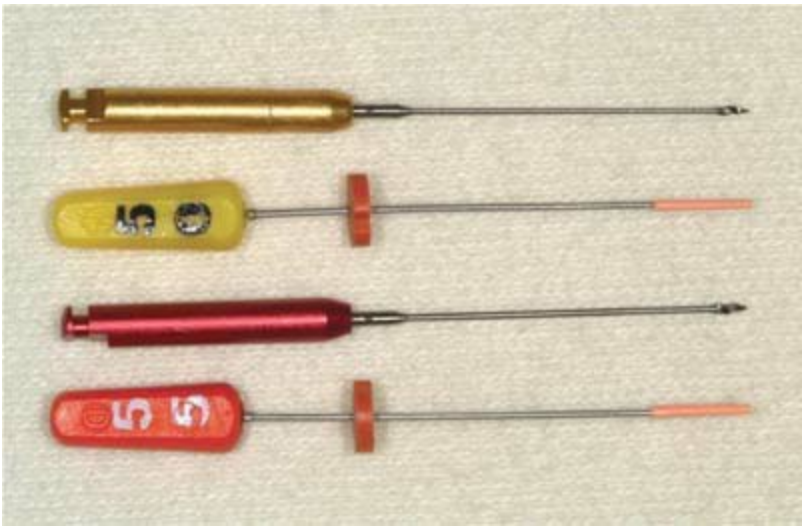

FIG. 9.50 Simplifill carrier and LightSpeed file (LightSpeed Technology, San Antonio, TX/Discus Dental, Culver City, CA).

Close-up of two simplifill carriers with gutta percha tips and two light speed files with axially flattened and pointed tips.

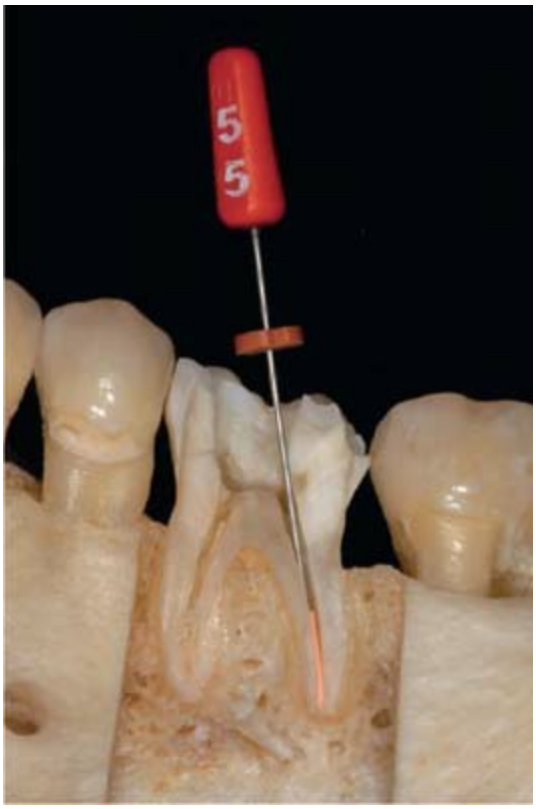

FIG. 9.51 Simplifill fitted to 1 to 3 mm from the prepared length.

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Close-up of lateral section of cutaway tooth shows an open crown with a gutta percha around a light speed file inserted in the right canal.

Thermomechanical compaction

McSpadden introduced the McSpadden Compactor, with flutes similar to a Hedström file, but in reverse. When activated in a slow-speed hand piece, the instrument would generate friction, soften the GP, and move it apically. Rotary compactors similar in design have been developed and advocated. To increase flexibility, the instrument is available in nickel titanium.

The technique requires fitting a GP master cone short of the prepared length and applying sealer. A compactor is selected on the basis of the size of the final RCS and inserted alongside the GP cone 3 to 4 mm from the prepared length. The hand piece is activated, and the friction of the rotating instrument heats the GP. The pliable mass is compacted apically and laterally as the device is withdrawn from the canal.

Advantages include simplicity of the armamentarium, the ability to fill canal irregularities,[251] and time. Disadvantages include possible extrusion of material, instrument fracture,[200] gouging canal walls, the inability to use the technique in curved canals, and possible excessive heat generation.[252] Microseal condensers (Kerr Dental) and the Gutta Condenser (DENTSPLY Sirona) are variations of this product.

Solvent techniques

GP can be plasticized with solvents, for example, chloroform, eucalyptol, or xylol. Disadvantages include shrinkage caused by evaporation of the solvent, voids, the inability to achieve predictable length control of the obturating material, and irritation of periradicular tissues. Obturation techniques using solvents have been abandoned and replaced with materials and methods that exhibit minimum shrinkage.

Pastes

Pastes fulfill some of the criteria outlined by Grossman[110] and can adapt to the complex internal canal anatomy. However, the flow characteristic can result in extrusion or incomplete obturation. The inability to control the

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material is a distinct disadvantage, and, when extrusion occurs, it is possible that it can be corrected only by surgical intervention. In addition, pastes are sometimes used as a substitute for complete C&S procedures, and the addition of paraformaldehyde results in severe toxicity.

Immediate obturation

Apical barriers may be necessary in cases with immature apical development, cases with external apical root resorption, and cases where instrumentation extends beyond the confines of the RCS. Dentin chips, $Ca(OH)_2$, demineralized dentin, lyophilized bone, tricalcium phosphate, hydroxyapatite, and collagen have been advocated for placement as a barrier in RCS exhibiting an open apex. The barriers are designed to permit obturation without extrusion of materials into the periradicular tissues but are often incomplete and do not seal the RCS.[242]

Dentin chips appear to confine materials to the RCS during instrumentation/obturation and may encourage development of a biologic seal.[242] A concern with this technique is contamination of the dentin with bacteria or other nonhost materials, because investigators found that infected dentin adversely affected healing.[208]

Calcium hydroxide has also been extensively used as a common apical barrier. $Ca(OH)_2$ has been shown to induce an apical barrier in apexification procedures. Calcifications similar to dentin plugs have been noted at the apical foramen.[208] Calcium hydroxide has the advantage of being free of bacterial contamination and may provide a better, although imperfect, apical seal.[303]

MTA (ProRoot MTA; DENTSPLY Sirona) has been successfully employed as an apical barrier material before obturation as an alternative to apexification, inducing cementum deposition and bone formation (Fig. 9.52).[264]

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FIG. 9.52 Mineral trioxide aggregate is available as ProRoot MTA. This material is advocated for use in apexification, repair of root perforations, repair of root resorption, root-end fillings, and pulp capping.

Close-up of mineral trioxide aggregate used for treating root canals in a sachet pack.

Source: (Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK, USA. All rights owned by and used with permission from DENTSPLY International Inc.)

After C&S procedures, the RCS is dried and a small increment of MTA is placed to the radiographic apex and the location verified through a radiograph. If the material is overextended, it can be easily removed by irrigating with sterile saline. If it is short of the radiographic apex, it can be compacted with prefitted pluggers to move it to the radiographic apex. The material is compacted into the apical portion of the root to form a barrier. After the material sets, any thermoplasticized technique can be used to backfill with GP without concern of overextension (Fig. 9.53). Hand compaction of MTA is superior to direct placement of ultrasonic activation.[9] However, indirect ultrasonic placement of a 4 mm apical plug of MTA improved the seal.[317]

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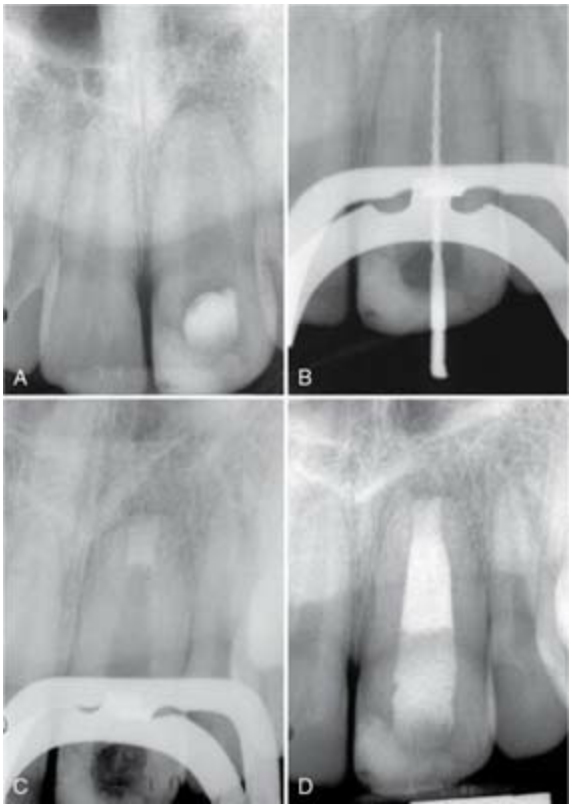

FIG. 9.53 Immediate obturation employs a barrier technique to prevent extrusion when the apex is open. This case involves a maxillary left central incisor with pulp necrosis caused by trauma. **A,** Pretreatment radiograph demonstrates a large canal with an open apex. **B,** Working length is established and the RCS is prepared. **C,** Mineral trioxide plug is placed. **D,** The canal is obturated with GP.

A) Radiograph shows a tooth with radiopaque square patch on the crown with radiolucent tooth surface.

B) Radiograph shows a tooth with a black square patch on the crown. A radiopaque outline of a working length file and clamp is visible.

C) Radiograph shows a tooth with a black patch on the crown. A radiopaque outline of mineral trioxide plug is visible.

D) Radiograph shows a tooth with a radiolucent crown, radiopaque neck and radiopaque canal.

MTA is biocompatible and capable of inducing hard tissue formation.

Clinical evidence supports the use of bioactive endodontic cements like MTA.[292] In a study comparing $Ca(OH)_2$ with MTA in 15 children, each having paired immature teeth with necrotic pulps, two teeth treated with

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$Ca(OH)_2$ exhibited pathosis on recall examination whereas all the teeth treated with MTA were clinically and radiographically successful.[87] A prospective clinical study found the success rate of MTA barriers and immediate obturation in 43 cases to be 81%.[269] In another study, 85% of 20 teeth with immature apical development were considered healed and 5% were considered healing after immediate barrier placement.[127] The time required for healing is comparable to traditional apexification with $Ca(OH)_2$, and treatment time is reduced.[226]

Another option for treating immature teeth with pulp necrosis involves regenerative endodontic techniques (see Chapter 12). Advantages include continued increasing thickness of the canal walls, continued apical root development, and apical closure.

Coronal orifice seal

No matter which technique is used to obturate the RCS, coronal microleakage can occur within a short time through seemingly well-obturated canals,[293] potentially causing infection of the periapical area. Early research efforts focused on the quality of the seal in the apical part of the RCS to prevent percolation of apical fluids. However, contemporary research efforts have identified the greater importance of maintaining a coronal seal to prevent bacterial leakage.[137] Cavit (Premier Dental Products, 3M ESPE) has traditionally been advocated as an acceptable material. One study demonstrated that the placement of 3.5 mm of Cavit or Super EBA cement (Bosworth, Skokie, IL, USA) decreased bacterial leakage by 85% and 65%, respectively, when compared with unsealed controls, which all leaked at 45 days.[224] Another method to slow leakage through the obturated RCS, should failure of the coronal restoration occur, is to cover the floor of the pulp chamber with a lining of bonded material after the removal of excess GP and sealer to the canal oriface.[162] Investigators found that this procedure resulted in none of the experimental RCS demonstrating bacterial leakage at 60 days.[60] A material with a different color than dentin may be used for identification in case subsequent placement of a post is desired.

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Future technology and nanodiamond-embedded gutta-percha

Ongoing clinical trials are researching nanodiamond-embedded GP with some promising progress report and no adverse events.[159,160]

These studies might be the foundation for the continued clinical research of nanodiamond biomaterial and their application in endodontics.

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References

1. *FDA explains status of N2 material*, J Am Dent Assoc 1992;123: 236-237.
2. American Association of Endodontists. Guide to Clinical Endodontics 2013; Chicago https://www.aae.org/specialty/clinical-resources/guide-clinical-endodontics/.
3. Aguirre AM, el-Deeb ME, Aguirre R. *The effect of ultrasonics on sealer distribution and sealing of root canals*, J Endod 1997;23: 759-764.
4. Ainley JE. *Fluorometric assay of the apical seal of root canal fillings*, Oral Surg Oral Med Oral Pathol 1970;29: 753-762.
5. Aktener BO, Cengiz T, Piskin B. *The penetration of smear material into dentinal tubules during instrumentation with surface-active reagents: a scanning electron microscopic study*, J Endod 1989;15: 588-590.
6. al-Khatib ZZ, Baum RH, Morse DR, et al: *The antimicrobial effect of various endodontic sealers*, Oral Surg Oral Med Oral Pathol 1990;70: 784-790.
7. Allan NA, Walton RC, Schaeffer MA. *Setting times for endodontic sealers under clinical usage and in vitro conditions*, J Endod 2001;27: 421-423.
8. Allison DA, Michelich RJ, Walton RE. *The influence of master cone adaptation on the quality of the apical seal*, J Endod 1981;7: 61-65.
9. Aminoshariae A, Hartwell GR, Moon PC. *Placement of mineral trioxide aggregate using two different techniques*, J Endod 2003;29: 679-682.
10. Aminoshariae A, Kulild J. *Master apical file size - smaller*

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www.konkur.in

or larger: a systematic review of microbial reduction, Int Endod J 2015;48: 1007-1022.

11. Aminoshariae A, Kulild JC. *Master apical file size - smaller or larger: a systematic review of healing outcomes*, Int Endod J 2015;48: 639-647.
12. Aminoshariae A, Kulild JC, Syed A. *Cone-beam computed tomography compared with intraoral radiographic lesions in endodontic outcome studies: a systematic review*, J Endod 2018;44: 1626-1631.
13. Anderson AC, Hellwig E, Vespermann R, et al: *Comprehensive analysis of secondary dental root canal infections: a combination of culture and culture-independent approaches reveals new insights*, PLoS One 2012;7: e49576.
14. Aqrabawi JA. *Outcome of endodontic treatment of teeth filled using lateral condensation versus vertical compaction (Schilder's technique)*, J Contemp Dent Pract 2006;7: 17-24.
15. Arruda MEF, Neves MAS, Diogenes A, et al: *Infection control in teeth with apical periodontitis using a triple antibiotic solution or calcium hydroxide with chlorhexidine: a randomized clinical trial*, J Endod 2018;44: 1474-1479.
16. Arslan D, Guneser MB, Dincer AN, et al: *Comparison of smear layer removal ability of QMix with different activation techniques*, J Endod 2016;42: 1279-1285.
17. Augsburger RA, Peters DD. *Radiographic evaluation of extruded obturation materials*, J Endod 1990;16: 492-497.
18. Awawdeh L, Al-Qudah A, Hamouri H, et al: *Outcomes of vital pulp therapy using mineral trioxide aggregate or biodentine: a prospective randomized clinical trial*, J Endod 2018;44: 1603-1609.

Telegram: @dental_k

www.konkur.in

19. Barkhordar RA. *Evaluation of antimicrobial activity in vitro of ten root canal sealers on Streptococcus sanguis and Streptococcus mutans*, Oral Surg Oral Med Oral Pathol 1989;68: 770-772.
20. Barkhordar RA, Watanabe LG, Marshall GW, et al: *Removal of intracanal smear by doxycycline in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84: 420-423.
21. Barthel CR, Zimmer S, Trope M. *Relationship of radiologic and histologic signs of inflammation in human root-filled teeth*, J Endod 2004;30: 75-79.
22. Baumgartner JC, Brown CM, Mader CL, et al: *A scanning electron microscopic evaluation of root canal debridement using saline, sodium hypochlorite, and citric acid*, J Endod 1984;10: 525-531.
23. Baumgartner JC, Cuenin PR. *Efficacy of several concentrations of sodium hypochlorite for root canal irrigation*, J Endod 1992;18: 605-612.
24. Baumgartner JC, Mader CL. *A scanning electron microscopic evaluation of four root canal irrigation regimens*, J Endod 1987;13: 147-157.
25. Beasley RT, Williamson AE, Justman BC, et al: *Time required to remove guttacore, thermafil plus, and thermoplasticized gutta-percha from moderately curved root canals with protaper files*, J Endod 2013;39: 125-128.
26. Beatty RG, Zakariasen KL. *Apical leakage associated with three obturation techniques in large and small root canals*, Int Endod J 1984;17: 67-72.
27. Behrend GD, Cutler CW, Gutmann JL. *An in-vitro study of smear layer removal and microbial leakage along root-canal fillings*, Int Endod J 1996;29: 99-107.
28. Belli S, Ozcan E, Derinbay O, et al: *A comparative*

Telegram: @dental_k

www.konkur.in

evaluation of sealing ability of a new, self-etching, dual-curable sealer: hybrid root SEAL (MetaSEAL), Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: e45-e52.

29. Bergmans L, Moisiadis P, De Munck J, et al: *Effect of polymerization shrinkage on the sealing capacity of resin fillers for endodontic use*, J Adhes Dent 2005;7: 321-329.
30. Berry KA, Loushine RJ, Primack PD, et al: *Nickel-titanium versus stainless-steel finger spreaders in curved canals*, J Endod 1998;24: 752-754.
31. Blaskovic-Subat V, Maricic B, Sutalo J. *Asymmetry of the root canal foramen*, Int Endod J 1992;25: 158-164.
32. Block RM, Lewis RD, Hirsch J, et al: *Systemic distribution of N2 paste containing 14C paraformaldehyde following root canal therapy in dogs*, Oral Surg Oral Med Oral Pathol 1980;50: 350-360.
33. Blum JY, Esber S, Micallef JP. *Analysis of forces developed during obturations. Comparison of three gutta-percha techniques*, J Endod 1997;23: 340-345.
34. Blum JY, Parahy E, Machtou P. *Warm vertical compaction sequences in relation to gutta-percha temperature*, J Endod 1997;23: 307-311.
35. Boiesen J, Brodin P. *Neurotoxic effect of two root canal sealers with calcium hydroxide on rat phrenic nerve in vitro*, Endod Dent Traumatol 1991;7: 242-245.
36. Bolles JA, He J, Svoboda KK, et al: *Comparison of Vibringe, EndoActivator, and needle irrigation on sealer penetration in extracted human teeth*, J Endod 2013;39: 708-711.
37. Bouillaguet S, Wataha JC, Tay FR, et al: *Initial in vitro biological response to contemporary endodontic sealers*, J Endod 2006;32: 989-992.

Telegram: @dental_k

www.konkur.in

38. Brady JM, del Rio CE. *Corrosion of endodontic silver cones in humans: a scanning electron microscope and X-ray microprobe study*, J Endod 1975;1: 205-210.
39. Brannstrom M. *Smear layer: pathological and treatment considerations*, Oper Dent Suppl 1984;3: 35-42.
40. Briseno BM, Willershausen B. *Root canal sealer cytotoxicity on human gingival fibroblasts. 1. Zinc oxide-eugenol-based sealers*, J Endod 1990;16: 383-386.
41. Briseno BM, Willershausen B. *Root canal sealer cytotoxicity on human gingival fibroblasts: 2. Silicone- and resin-based sealers*, J Endod 1991;17: 537-540.
42. Briseno BM, Willershausen B. *Root canal sealer cytotoxicity with human gingival fibroblasts. III. Calcium hydroxide-based sealers*, J Endod 1992;18: 110-113.
43. Briseno Marroquin B, Wolter D, Willershausen-Zonnchen B. *Dimensional variability of nonstandardized greater taper finger spreaders with matching gutta-percha points*, Int Endod J 2001;34: 23-28.
44. Brodin P, Roed A, Aars H, et al: *Neurotoxic effects of root filling materials on rat phrenic nerve in vitro*, J Dent Res 1982;61: 1020-1023.
45. Buchanan LS. *Continuous wave of condensation technique*, Endod Prac 1998;1: 7-10 13–6, 18 passim.
46. Burch JG, Hulen S. *The relationship of the apical foramen to the anatomic apex of the tooth root*, Oral Surg Oral Med Oral Pathol 1972;34: 262-268.
47. Burkovski A, Karl M. *Lack of evidence for the necessity of root canal obturation*, Quintessence Int 2018;2–8:.
48. Bystrom A, Sundqvist G. *Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy*, Scand J Dent Res 1981;89: 321-328.
49. Bystrom A, Sundqvist G. *The antibacterial action of*

Telegram: @dental_k

www.konkur.in

sodium hypochlorite and EDTA in 60 cases of endodontic therapy, Int Endod J 1985;18: 35-40.

50. Cailleteau JG, Mullaney TP. *Prevalence of teaching apical patency and various instrumentation and obturation techniques in United States dental schools*, J Endod 1997;23: 394-396.
51. Calt S, Serper A. *Time-dependent effects of EDTA on dentin structures*, J Endod 2002;28: 17-19.
52. Camargo RV, Silva-Sousa YTC, Rosa R, et al: *Evaluation of the physicochemical properties of silicone- and epoxy resin-based root canal sealers*, Braz Oral Res 2017;31: e72.
53. Cameron JA. *The synergistic relationship between ultrasound and sodium hypochlorite: a scanning electron microscope evaluation*, J Endod 1987;13: 541-545.
54. Camilleri J, Pitt Ford TR. *Mineral trioxide aggregate: a review of the constituents and biological properties of the material*, Int Endod J 2006;39: 747-754.
55. Camps J, About I. *Cytotoxicity testing of endodontic sealers: a new method*, J Endod 2003;29: 583-586.
56. Card SJ, Sigurdsson A, Orstavik D, et al: *The effectiveness of increased apical enlargement in reducing intracanal bacteria*, J Endod 2002;28: 779-783.
57. Carpenter MT, Sidow SJ, Lindsey KW, et al: *Regaining apical patency after obturation with gutta-percha and a sealer containing mineral trioxide aggregate*, J Endod 2014;40: 588-590.
58. Carratu P, Amato M, Riccitiello F, et al: *Evaluation of leakage of bacteria and endotoxins in teeth treated endodontically by two different techniques*, J Endod 2002;28: 272-275.
59. Chailertvanitkul P, Saunders WP, MacKenzie D. *The*

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www.konkur.in

effect of smear layer on microbial coronal leakage of gutta-percha root fillings, Int Endod J 1996;29: 242-248.

60. Chailertvanitkul P, Saunders WP, Saunders EM, et al: *An evaluation of microbial coronal leakage in the restored pulp chamber of root-canal treated multirooted teeth*, Int Endod J 1997;30: 318-322.
61. Chang SW, Lee SY, Kang SK, et al: *In vitro biocompatibility, inflammatory response, and osteogenic potential of 4 root canal sealers: Sealapex, Sankin apatite root sealer, MTA Fillapex, and iRoot SP root canal sealer*, J Endod 2014;40: 1642-1648.
62. Chugal NM, Clive JM, Spangberg LS. *Endodontic treatment outcome: effect of the permanent restoration*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 576-582.
63. Cobankara FK, Altinoz HC, Ergani O, et al: *In vitro antibacterial activities of root-canal sealers by using two different methods*, J Endod 2004;30: 57-60.
64. Cohler CM, Newton CW, Patterson SS, et al: *Studies of Sargenti's technique of endodontic treatment: short-term response in monkeys*, J Endod 1980;6: 473-478.
65. Collado-Gonzalez M, Tomas-Catala CJ, Onate-Sanchez RE, et al: *Cytotoxicity of GuttaFlow Bioseal, GuttaFlow2, MTA Fillapex, and AH plus on human periodontal ligament stem cells*, J Endod 2017;43: 816-822.
66. Cotton TP, Schindler WG, Schwartz SA, et al: *A retrospective study comparing clinical outcomes after obturation with Resilon/Epiphany or Gutta-Percha/Kerr sealer*, J Endod 2008;34: 789-797.
67. Cruz-Filho AM, Sousa-Neto MD, Savioli RN, et al: *Effect of chelating solutions on the microhardness of root canal lumen dentin*, J Endod 2011;37: 358-362.

Telegram: @dental_k

www.konkur.in

68. Cuesta A, Zea-Garcia JD, Londono-Zuluaga D, et al: *Multiscale understanding of tricalcium silicate hydration reactions*, Sci Rep 2018;8: 8544.
69. Cunningham WT, Martin H. *A scanning electron microscope evaluation of root canal debridement with the endosonic ultrasonic synergistic system*, Oral Surg Oral Med Oral Pathol 1982;53: 527-531.
70. Dai L, Khechen K, Khan S, et al: *The effect of QMix, an experimental antibacterial root canal irrigant, on removal of canal wall smear layer and debris*, J Endod 2011;37: 80-84.
71. Darvell BW, Wu RC. *"MTA"—an hydraulic silicate cement: review update and setting reaction*, Dent Mater 2011;27: 407-422.
72. de Chevigny C, Dao TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study—phase 4: initial treatment*, J Endod 2008;34: 258-263.
73. de Chevigny C, Dao TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study—phases 3 and 4: orthograde retreatment*, J Endod 2008;34: 131-137.
74. De Deus GA, Gurgel-Filho ED, Maniglia-Ferreira C, et al: *The influence of filling technique on depth of tubule penetration by root canal sealer: a study using light microscopy and digital image processing*, Aust Endod J 2004;30: 23-28.
75. de Lucena JM, Decker EM, Walter C, et al: *Antimicrobial effectiveness of intracanal medicaments on Enterococcus faecalis: chlorhexidine versus octenidine*, Int Endod J 2013;46: 53-61.
76. de Oliveira NG, de Souza Araujo PR, da Silveira MT, et al: *Comparison of the biocompatibility of calcium silicate-based materials to mineral trioxide aggregate: systematic*

Telegram: @dental_k

www.konkur.in

review, Eur J Dent 2018;12: 317-326.

77. Delivanis PD, Mattison GD, Mendel RW. *The survivability of F43 strain of Streptococcus sanguis in root canals filled with gutta-percha and Procosol cement*, J Endod 1983;9: 407-410.
78. Desai S, Chandler N. *Calcium hydroxide-based root canal sealers: a review*, J Endod 2009;35: 475-480.
79. Diamond A, Carrel R. *The smear layer: a review of restorative progress*, J Pedod 1984;8: 219-226.
80. Donnermeyer D, Schafer E, Burklein S. *Real-time intracanal temperature measurement during different obturation techniques*, J Endod 2018;44: 1832-1836.
81. Dow PR, Ingle JI. *Isotope determination of root canal failure*, Oral Surg Oral Med Oral Pathol 1955;8: 1100-1104.
82. Drake DR, Wiemann AH, Rivera EM, et al: *Bacterial retention in canal walls in vitro: effect of smear layer*, J Endod 1994;20: 78-82.
83. Dummer PM, Kelly T, Meghji A, et al: *An in vitro study of the quality of root fillings in teeth obturated by lateral condensation of gutta-percha or Thermafil obturators*, Int Endod J 1993;26: 99-105.
84. Dummer PM, Lyle L, Rawle J, et al: *A laboratory study of root fillings in teeth obturated by lateral condensation of gutta-percha or Thermafil obturators*, Int Endod J 1994;27: 32-38.
85. Dummer PM, McGinn JH, Rees DG. *The position and topography of the apical canal constriction and apical foramen*, Int Endod J 1984;17: 192-198.
86. Economides N, Liolios E, Kolokuris I, et al: *Long-term evaluation of the influence of smear layer removal on the sealing ability of different sealers*, J Endod 1999;25: 123-

Telegram: @dental_k

www.konkur.in

125.
87. El-Meligy OA, Avery DR. *Comparison of apexification with mineral trioxide aggregate and calcium hydroxide*, Pediatr Dent 2006;28: 248-253.
88. Erisen R, Yucel T, Kucukay S. *Endomethasone root canal filling material in the mandibular canal. A case report*, Oral Surg Oral Med Oral Pathol 1989;68: 343-345.
89. Esberard RM, Carnes DL Jr, Del Rio CE. *pH changes at the surface of root dentin when using root canal sealers containing calcium hydroxide*, J Endod 1996;22: 399-401.
90. Estrela C, Couto GS, Bueno MR, et al: *Apical foramen position in relation to proximal root surfaces of human permanent teeth determined by using a new cone-beam computed tomographic software*, J Endod 2018;44: 1741-1748.
91. Evans M, Davies JK, Sundqvist G, et al: *Mechanisms involved in the resistance of Enterococcus faecalis to calcium hydroxide*, Int Endod J 2002;35: 221-228.
92. Farzaneh M, Abitbol S, Friedman S. *Treatment outcome in endodontics: the Toronto study. Phases I and II: Orthograde retreatment*, J Endod 2004;30: 627-633.
93. Farzaneh M, Abitbol S, Lawrence HP, et al: *Treatment outcome in endodontics-the Toronto study. Phase II: initial treatment*, J Endod 2004;30: 302-309.
94. Felippe WT, Ruschel MF, Felippe GS, et al: *SEM evaluation of the apical external root surface of teeth with chronic periapical lesion*, Aust Endod J 2009;35: 153-157.
95. Fransen JN, He J, Glickman GN, et al: *Comparative assessment of ActiV GP/glass ionomer sealer, Resilon/Epiphany, and gutta-percha/AH plus obturation: a bacterial leakage study*, J Endod 2008;34: 725-727.
96. Friedman CE, Sandrik JL, Heuer MA, et al: *Composition*

Telegram: @dental_k

www.konkur.in

and physical properties of gutta-percha endodontic filling materials, J Endod 1977;3: 304-308.

97. Friedman S, Abitbol S, Lawrence HP. *Treatment outcome in endodontics: the Toronto study. Phase 1: initial treatment*, J Endod 2003;29: 787-793.
98. Friedman S, Lost C, Zarrabian M, et al: *Evaluation of success and failure after endodontic therapy using a glass ionomer cement sealer*, J Endod 1995;21: 384-390.
99. Gencoglu N, Samani S, Gunday M. *Dentinal wall adaptation of thermoplasticized gutta-percha in the absence or presence of smear layer: a scanning electron microscopic study*, J Endod 1993;19: 558-562.
100. Gettleman BH, Messer HH, ElDeeb ME. *Adhesion of sealer cements to dentin with and without the smear layer*, J Endod 1991;17: 15-20.
101. Gharai SR, Thorpe JR, Strother JM, et al: *Comparison of generated forces and apical microleakage using nickel-titanium and stainless steel finger spreaders in curved canals*, J Endod 2005;31: 198-200.
102. Ghoneim AG, Lutfy RA, Sabet NE, et al: *Resistance to fracture of roots obturated with novel canal-filling systems*, J Endod 2011;37: 1590-1592.
103. Gilhooly RM, Hayes SJ, Bryant ST, et al: *Comparison of lateral condensation and thermomechanically compacted warm alpha-phase gutta-percha with a single cone for obturating curved root canals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 89-94.
104. Gillen BM, Looney SW, Gu LS, et al: *Impact of the quality of coronal restoration versus the quality of root canal fillings on success of root canal treatment: a systematic review and meta-analysis*, J Endod 2011;37: 895-902.

Telegram: @dental_k

www.konkur.in

105. Givol N, Rosen E, Bjorndal L, et al: *Medico-legal aspects of altered sensation following endodontic treatment: a retrospective case series*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 126-131.
106. Goldberg F, Gurfinkel J, Spielberg C. *Microscopic study of standardized gutta-percha points*, Oral Surg Oral Med Oral Pathol 1979;47: 275-276.
107. Gomes-Filho JE, Watanabe S, Bernabe PF, et al: *A mineral trioxide aggregate sealer stimulated mineralization*, J Endod 2009;35: 256-260.
108. Goodman A, Schilder H, Aldrich W. *The thermomechanical properties of gutta-percha. II. The history and molecular chemistry of gutta-percha*, Oral Surg Oral Med Oral Pathol 1974;37: 954-961.
109. Goodman A, Schilder H, Aldrich W. *The thermomechanical properties of gutta-percha. Part IV. A thermal profile of the warm gutta-percha packing procedure*, Oral Surg Oral Med Oral Pathol 1981;51: 544-551.
110. Grossman. Endodontics ed 11 1988; Lea & Febiger Philadelphia.
111. Grossman LI. *An improved root canal cement*, J Am Dent Assoc 1958;56: 381-385.
112. Guignes P, Faure J, Maurette A. *Relationship between endodontic preparations and human dentin permeability measured in situ*, J Endod 1996;22: 60-67.
113. Gutierrez JH, Aguayo P. *Apical foraminal openings in human teeth. Number and location*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 769-777.
114. Gutierrez JH, Brizuela C, Villota E. *Human teeth with periapical pathosis after overinstrumentation and overfilling of the root canals: a scanning electron*

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www.konkur.in

microscopic study, Int Endod J 1999;32: 40-48.
115. Gutmann JL. *Adaptation of injected thermoplasticized gutta-percha in the absence of the dentinal smear layer*, Int Endod J 1993;26: 87-92.
116. Haapasalo HK, Siren EK, Waltimo TM, et al: *Inactivation of local root canal medicaments by dentine: an in vitro study*, Int Endod J 2000;33: 126-131.
117. Hale R, Gatti R, Glickman GN, et al: *Comparative analysis of carrier-based obturation and lateral compaction: a retrospective clinical outcomes study*, Int J Dent 2012;2012: 954675.
118. Hall MC, Clement DJ, Dove SB, et al: *A comparison of sealer placement techniques in curved canals*, J Endod 1996;22: 638-642.
119. Hansrani V. *Assessing root canal fillings on a radiograph—an overview*, Br Dent J 2015;219: 481-483.
120. Hauman CH, Love RM. *Biocompatibility of dental materials used in contemporary endodontic therapy: a review. Part 2. Root-canal-filling materials*, Int Endod J 2003;36: 147-160.
121. Haznedaroglu F, Ersev H. *Tetracycline HCl solution as a root canal irrigant*, J Endod 2001;27: 738-740.
122. Heard F, Walton RE. *Scanning electron microscope study comparing four root canal preparation techniques in small curved canals*, Int Endod J 1997;30: 323-331.
123. Heling I, Chandler NP. *The antimicrobial effect within dentinal tubules of four root canal sealers*, J Endod 1996;22: 257-259.
124. Heling I, Gorfil C, Slutzky H, et al: *Endodontic failure caused by inadequate restorative procedures: review and treatment recommendations*, J Prosthet Dent 2002;87: 674-678.

Telegram: @dental_k

www.konkur.in

125. Herrera DR, Santos ZT, Tay LY, et al: *Efficacy of different final irrigant activation protocols on smear layer removal by EDTA and citric acid*, Microsc Res Tech 2013;76: 364-369.
126. Hess D, Solomon E, Spears R, et al: *Retreatability of a bioceramic root canal sealing material*, J Endod 2011;37: 1547-1549.
127. Holden DT, Schwartz SA, Kirkpatrick TC, et al: *Clinical outcomes of artificial root-end barriers with mineral trioxide aggregate in teeth with immature apices*, J Endod 2008;34: 812-817.
128. Holland GR. *Periapical response to apical plugs of dentin and calcium hydroxide in ferret canines*, J Endod 1984;10: 71-74.
129. Hottel TL, el-Refai NY, Jones JJ. *A comparison of the effects of three chelating agents on the root canals of extracted human teeth*, J Endod 1999;25: 716-717.
130. Hugh CL, Walton RE, Facer SR. *Evaluation of intracanal sealer distribution with 5 different obturation techniques*, Quintessence Int 2005;36: 721-729.
131. Hulsmann M, Heckendorff M, Lennon A. *Chelating agents in root canal treatment: mode of action and indications for their use*, Int Endod J 2003;36: 810-830.
132. Iqbal MK, Johansson AA, Akeel RF, et al: *A retrospective analysis of factors associated with the periapical status of restored, endodontically treated teeth*, Int J Prosthodont 2003;16: 31-38.
133. Iqbal MK, Ku J. *Instrumentation and obturation of the apical third of root canals: addressing the forgotten dimension*, Compend Contin Educ Dent 2007;28: 314-320 quiz 21, 32.
134. Jafari F, Jafari S. *Importance and methodologies of*

Telegram: @dental_k

www.konkur.in

endodontic microleakage studies: A systematic review, J Clin Exp Dent 2017;9: e812- e819.

135. Jafari F, Jafari S, Etesamnia P. *Genotoxicity, bioactivity and clinical properties of calcium silicate based sealers: a literature review*, Iran Endod J 2017;12: 407-413.
136. Jasper. *Adaptation and tolerance of silver point canal filling*, J Dent Res 1941;4: 355.
137. Jenkins S, Kulild J, Williams K, et al: *Sealing ability of three materials in the orifice of root canal systems obturated with gutta-percha*, J Endod 2006;32: 225-227.
138. Johnson BT, Bond MS. *Leakage associated with single or multiple increment backfill with the Obtura II gutta-percha system*, J Endod 1999;25: 613-614.
139. Johnson WT, Zakariasen KL. *Spectrophotometric analysis of microleakage in the fine curved canals found in the mesial roots of mandibular molars*, Oral Surg Oral Med Oral Pathol 1983;56: 305-309.
140. Jung S, Sielker S, Hanisch MR, et al: *Cytotoxic effects of four different root canal sealers on human osteoblasts*, PLoS One 2018;13: e0194467.
141. Kakehashi S, Stanley HR, Fitzgerald RJ. *The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral Pathol 1965;20: 340-349.
142. Kaur A, Shah N, Logani A, et al: *Biotoxicity of commonly used root canal sealers: A meta-analysis*, J Conserv Dent 2015;18: 83-88.
143. Kazemi RB, Safavi KE, Spangberg LS. *Dimensional changes of endodontic sealers*, Oral Surg Oral Med Oral Pathol 1993;76: 766-771.
144. Keinan D, Moshonov J, Smidt A. *Is endodontic re-treatment mandatory for every relatively old temporary*

Telegram: @dental_k

www.konkur.in

restoration? A narrative review, J Am Dent Assoc 2011;142: 391-396.

145. Kerezoudis NP, Valavanis D, Prountzos F. *A method of adapting gutta-percha master cones for obturation of open apex cases using heat*, Int Endod J 1999;32: 53-60.
146. Kersten HW, Wesselink PR, Thoden van Velzen SK. *The diagnostic reliability of the buccal radiograph after root canal filling*, Int Endod J 1987;20: 20-24.
147. Kim YK, Grandini S, Ames JM, et al: *Critical review on methacrylate resin-based root canal sealers*, J Endod 2010;36: 383-399.
148. Kleier DJ, Averbach RE. *Painful dysesthesia of the inferior alveolar nerve following use of a paraformaldehyde-containing root canal sealer*, Endod Dent Traumatol 1988;4: 46-48.
149. Koch MJ. *Formaldehyde release from root-canal sealers: influence of method*, Int Endod J 1999;32: 10-16.
150. Kok D, Rosa RA, Barreto MS, et al: *Penetrability of AH plus and MTA fillapex after endodontic treatment and retreatment: a confocal laser scanning microscopy study*, Microsc Res Tech 2014;77: 467-471.
151. Krastl G, Allgayer N, Lenherr P, et al: *Tooth discoloration induced by endodontic materials: a literature review*, Dent Traumatol 2013;29: 2-7.
152. Kuci A, Alacam T, Yavas O, et al: *Sealer penetration into dentinal tubules in the presence or absence of smear layer: a confocal laser scanning microscopic study*, J Endod 2014;40: 1627-1631.
153. Kuttler Y. *Microscopic investigation of root apexes*, J Am Dent Assoc 1955;50: 544-552.
154. Kytridou V, Gutmann JL, Nunn MH. *Adaptation and sealability of two contemporary obturation techniques in*

Telegram: @dental_k

www.konkur.in

the absence of the dentinal smear layer, Int Endod J 1999;32: 464-474.

155. Langeland K. *Root canal sealants and pastes*, Dent Clin North Am 1974;18: 309-327.
156. Langeland K, Olsson B, Pascon EA. *Biological evaluation of Hydron*, J Endod 1981;7: 196-204.
157. Law A, Messer H. *An evidence-based analysis of the antibacterial effectiveness of intracanal medicaments*, J Endod 2004;30: 689-694.
158. Lawson MS, Loushine B, Mai S, et al: *Resistance of a 4-META-containing, methacrylate-based sealer to dislocation in root canals*, J Endod 2008;34: 833-837.
159. Lee DK, Kee T, Liang Z, et al: *Clinical validation of a nanodiamond-embedded thermoplastic biomaterial*, Proc Natl Acad Sci U S A 2017;114: E9445- E9454.
160. Lee DK, Kim SV, Limansubroto AN, et al: *Nanodiamond-gutta percha composite biomaterials for root canal therapy*, ACS Nano 2015;9: 11490-11501.
161. Lee FS, Van Cura JE, BeGole E. *A comparison of root surface temperatures using different obturation heat sources*, J Endod 1998;24: 617-620.
162. Leonard JE, Gutmann JL, Guo IY. *Apical and coronal seal of roots obturated with a dentine bonding agent and resin*, Int Endod J 1996;29: 76-83.
163. Leonardo MR, Bezerra da Silva LA, Filho MT, et al: *Release of formaldehyde by 4 endodontic sealers*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88: 221-225.
164. Lertchirakarn V, Palamara JE, Messer HH. *Patterns of vertical root fracture: factors affecting stress distribution in the root canal*, J Endod 2003;29: 523-528.
165. Levitan ME, Himel VT, Luckey JB. *The effect of*

Telegram: @dental_k

www.konkur.in

insertion rates on fill length and adaptation of a thermoplasticized gutta-percha technique, J Endod 2003;29: 505-508.

166. Lim ES, Park YB, Kwon YS, et al: *Physical properties and biocompatibility of an injectable calcium-silicate-based root canal sealer: in vitro and in vivo study*, BMC Oral Health 2015;15: 129.
167. Lim TS, Wee TY, Choi MY, et al: *Light and scanning electron microscopic evaluation of Glyde File Prep in smear layer removal*, Int Endod J 2003;36: 336-343.
168. Loushine BA, Bryan TE, Looney SW, et al: *Setting properties and cytotoxicity evaluation of a premixed bioceramic root canal sealer*, J Endod 2011;37: 673-677.
169. Lynch CD, Burke FM, Ni Riordain R, et al: *The influence of coronal restoration type on the survival of endodontically treated teeth*, Eur J Prosthodont Restor Dent 2004;12: 171-176.
170. Mader CL, Baumgartner JC, Peters DD. *Scanning electron microscopic investigation of the smeared layer on root canal walls*, J Endod 1984;10: 477-483.
171. Mai S, Kim YK, Arola DD, et al: *Differential aggressiveness of ethylenediamine tetraacetic acid in causing canal wall erosion in the presence of sodium hypochlorite*, J Dent 2010;38: 201-206.
172. Malkhassian G, Manzur AJ, Legner M, et al: *Antibacterial efficacy of MTAD final rinse and two percent chlorhexidine gel medication in teeth with apical periodontitis: a randomized double-blinded clinical trial*, J Endod 2009;35: 1483-1490.
173. Malueg LA, Wilcox LR, Johnson W. *Examination of external apical root resorption with scanning electron microscopy*, Oral Surg Oral Med Oral Pathol Oral Radiol

Telegram: @dental_k

www.konkur.in

Endod 1996;82: 89-93.
174. Mandal P, Zhao J, Sah SK, et al: *In vitro cytotoxicity of guttaflow 2 on human gingival fibroblasts*, J Endod 2014;40: 1156-1159.
175. Manfredi M, Figini L, Gagliani M, et al: *Single versus multiple visits for endodontic treatment of permanent teeth*, Cochrane Database Syst Rev 2016;12: Cd005296.
176. Manzur A, Gonzalez AM, Pozos A, et al: *Bacterial quantification in teeth with apical periodontitis related to instrumentation and different intracanal medications: a randomized clinical trial*, J Endod 2007;33: 114-118.
177. Marquis VL, Dao T, Farzaneh M, et al: *Treatment outcome in endodontics: the Toronto Study. Phase III: initial treatment*, J Endod 2006;32: 299-306.
178. McComb D, Smith DC. *A preliminary scanning electron microscopic study of root canals after endodontic procedures*, J Endod 1975;1: 238-242.
179. Mendes AT, Silva PBD, So BB, et al: *Evaluation of physicochemical properties of new calcium silicate-based sealer*, Braz Dent J 2018;29: 536-540.
180. Mente J, Werner S, Koch MJ, et al: *In vitro leakage associated with three root-filling techniques in large and extremely large root canals*, J Endod 2007;33: 306-309.
181. Mergoni G, Percudani D, Lodi G, et al: *Prevalence of Candida species in endodontic infections: systematic review and meta-analysis*, J Endod 2018;44: 1616-1625.e9.
182. Messing JJ. *An investigation of the sealing properties of some root filling materials*, J Br Endod Soc 1970;4: 18-22.
183. Mjor IA, Smith MR, Ferrari M, et al: *The structure of dentine in the apical region of human teeth*, Int Endod J 2001;34: 346-353.

Telegram: @dental_k

www.konkur.in

184. Mohammadi Z, Dummer PM. *Properties and applications of calcium hydroxide in endodontics and dental traumatology*, Int Endod J 2011;44: 697-730.
185. Molander A, Warfvinge J, Reit C, et al: *Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical periodontitis: a randomized clinical trial*, J Endod 2007;33: 1145-1148.
186. Moller AJ, Fabricius L, Dahlen G, et al: *Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys*, Scand J Dent Res 1981;89: 475-484.
187. Moshonov J, Trope M, Friedman S. *Retreatment efficacy 3 months after obturation using glass ionomer cement, zinc oxide-eugenol, and epoxy resin sealers*, J Endod 1994;20: 90-92.
188. Nagas E, Uyanik MO, Eymirli A, et al: *Dentin moisture conditions affect the adhesion of root canal sealers*, J Endod 2012;38: 240-244.
189. Nair PN. *Abusing technology? Culture-difficult microbes and microbial remnants*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 569-570.
190. Nair PN, Henry S, Cano V, et al: *Microbial status of apical root canal system of human mandibular first molars with primary apical periodontitis after "one-visit" endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 231-252.
191. Naito T. *Better success rate for root canal therapy when treatment includes obturation short of the apex*, Evid Based Dent 2005;6: 45.
192. Neelakantan P, Grotra D, Sharma S. *Retreatability of 2 mineral trioxide aggregate-based root canal sealers: a*

Telegram: @dental_k

www.konkur.in

cone-beam computed tomography analysis, J Endod 2013;39: 893-896.

193. Newton CW, Patterson SS, Kafrawy AH. *Studies of Sargenti's technique of endodontic treatment: six-month and one-year responses*, J Endod 1980;6: 509-517.
194. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2: tooth survival*, Int Endod J 2011;44: 610-625.
195. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature - part 1. Effects of study characteristics on probability of success*, Int Endod J 2007;40: 921-939.
196. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature — Part 2. Influence of clinical factors*, Int Endod J 2008;41: 6-31.
197. Nino-Barrera JL, Gamboa-Martinez LF, Laserna-Zuluaga H, et al: *Factors associated to apical overfilling after a thermoplastic obturation technique - Calamus(R) or Guttacore(R): a randomized clinical experiment*, Acta Odontol Latinoam 2018;31: 45-52.
198. Niu LN, Jiao K, Wang TD, et al: *A review of the bioactivity of hydraulic calcium silicate cements*, J Dent 2014;42: 517-533.
199. Nygaard-Østby. *Chelation in root canal cleansing and widening of root canals*, Odontol Tidskr 1957;65: 3.
200. O'Neill KJ, Pitts DL, Harrington GW. *Evaluation of the apical seal produced by the McSpadden compactor and the lateral condensation with a chloroform-softened primary cone*, J Endod 1983;9: 190-197.
201. Oksan T, Aktener BO, Sen BH, et al: *The penetration of*

Telegram: @dental_k

www.konkur.in

root canal sealers into dentinal tubules. A scanning electron microscopic study, Int Endod J 1993;26: 301-305.

202. Onay EO, Ungor M, Unver S, et al: *An in vitro evaluation of the apical sealing ability of new polymeric endodontic filling systems*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: e49- e54.
203. Ordinola-Zapata R, Bramante CM, Minotti PG, et al: *Antimicrobial activity of triantibiotic paste, 2% chlorhexidine gel, and calcium hydroxide on an intraoral-infected dentin biofilm model*, J Endod 2013;39: 115-118.
204. Orstavik D, Haapasalo M. *Disinfection by endodontic irrigants and dressings of experimentally infected dentinal tubules*, Endod Dent Traumatol 1990;6: 142-149.
205. Orstavik D, Nordahl I, Tibballs JE. *Dimensional change following setting of root canal sealer materials*, Dent Mater 2001;17: 512-519.
206. Osiri S, Banomyong D, Sattabanasuk V, et al: *Root reinforcement after obturation with calcium silicate-based sealer and modified Gutta-percha cone*, J Endod 2018;44: 1843-1848.
207. Oswald RJ, Cohn SA. *Systemic distribution of lead from root canal fillings*, J Endod 1975;1: 59-63.
208. Oswald RJ, Friedman CE. *Periapical response to dentin filings. A pilot study*, Oral Surg Oral Med Oral Pathol 1980;49: 344-355.
209. Ozkurt-Kayahan Z, Barut G, Ulusoy Z, et al: *Influence of post space preparation on the apical leakage of calamus, single-cone and cold lateral condensation obturation techniques: a computerized fluid filtration study*, J Prosthodont 2017.
210. Ozok AR, van der Sluis LW, Wu MK, et al: *Sealing ability of a new polydimethylsiloxane-based root canal*

Telegram: @dental_k

www.konkur.in

filling material, J Endod 2008;34: 204-207.
211. Pallares A, Faus V, Glickman GN. *The adaptation of mechanically softened gutta-percha to the canal walls in the presence or absence of smear layer: a scanning electron microscopic study*, Int Endod J 1995;28: 266-269.
212. Pameijer CH, Zmener O. *Resin materials for root canal obturation*, Dent Clin North Am 2010;54: 325-344.
213. Paredes-Vieyra J, Enriquez FJ. *Success rate of single- versus two-visit root canal treatment of teeth with apical periodontitis: a randomized controlled trial*, J Endod 2012;38: 1164-1169.
214. Parirokh M, Torabinejad M, Dummer PMH. *Mineral trioxide aggregate and other bioactive endodontic cements: an updated overview - part I: vital pulp therapy*, Int Endod J 2018;51: 177-205.
215. Pashley DH. *Smear layer: overview of structure and function*, Proc Finn Dent Soc Suppl 1, 1992;88: 215-224.
216. Pashley EL, Birdsong NL, Bowman K, et al: *Cytotoxic effects of NaOCl on vital tissue*, J Endod 1985;11: 525-528.
217. Patel S, Dawood A, Whaites E, et al: *New dimensions in endodontic imaging: part 1. Conventional and alternative radiographic systems*, Int Endod J 2009;42: 447-462.
218. Peng L, Ye L, Tan H, et al: *Outcome of root canal obturation by warm gutta-percha versus cold lateral condensation: a meta-analysis*, J Endod 2007;33: 106-109.
219. Perez F, Rochd T, Lodter JP, et al: *In vitro study of the penetration of three bacterial strains into root dentine*, Oral Surg Oral Med Oral Pathol 1993;76: 97-103.
220. Perez Heredia M, Clavero Gonzalez J, Ferrer Luque CM, et al: *Apical seal comparison of low-temperature thermoplasticized Gutta-percha technique and lateral*

Telegram: @dental_k

www.konkur.in

condensation with two different master cones, Med Oral Patol Oral Cir Bucal 2007;12: E175- E179.

221. Peters DD. *Two-year in vitro solubility evaluation of four Gutta-percha sealer obturation techniques*, J Endod 1986;12: 139-145.
222. Peters LB, van Winkelhoff AJ, Buijs JF, et al: *Effects of instrumentation, irrigation and dressing with calcium hydroxide on infection in pulpless teeth with periapical bone lesions*, Int Endod J 2002;35: 13-21.
223. Pirani C, Friedman S, Gatto MR, et al: *Survival and periapical health after root canal treatment with carrier-based root fillings: five-year retrospective assessment*, Int Endod J Suppl 3, 2018;51: e178- e188.
224. Pisano DM, DiFiore PM, McClanahan SB, et al: *Intraorifice sealing of gutta-percha obturated root canals to prevent coronal microleakage*, J Endod 1998;24: 659-662.
225. Piskin B, Aydin B, Sarikanat M. *The effect of spreader size on fracture resistance of maxillary incisor roots*, Int Endod J 2008;41: 54-59.
226. Pradhan DP, Chawla HS, Gauba K, et al: *Comparative evaluation of endodontic management of teeth with unformed apices with mineral trioxide aggregate and calcium hydroxide*, J Dent Child (Chic) 2006;73: 79-85.
227. Qian W, Shen Y, Haapasalo M. *Quantitative analysis of the effect of irrigant solution sequences on dentin erosion*, J Endod 2011;37: 1437-1441.
228. Qu W, Bai W, Liang YH, et al: *Influence of warm vertical compaction technique on physical properties of root canal sealers*, J Endod 2016;42: 1829-1833.
229. Radovic I, Monticelli F, Goracci C, et al: *Self-adhesive resin cements: a literature review*, J Adhes Dent 2008;10:

Telegram: @dental_k

www.konkur.in

251-258.
230. Rasimick BJ, Shah RP, Musikant BL, et al: *Radiopacity of endodontic materials on film and a digital sensor*, J Endod 2007;33: 1098-1101.
231. Ricucci D. *Apical limit of root canal instrumentation and obturation, part 1. Literature review*, Int Endod J 1998;31: 384-393.
232. Ricucci D, Bergenholtz G. *Bacterial status in root-filled teeth exposed to the oral environment by loss of restoration and fracture or caries—a histobacteriological study of treated cases*, Int Endod J 2003;36: 787-802.
233. Ricucci D, Grondahl K, Bergenholtz G. *Periapical status of root-filled teeth exposed to the oral environment by loss of restoration or caries*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 354-359.
234. Ricucci D, Langeland K. *Apical limit of root canal instrumentation and obturation, part 2. A histological study*, Int Endod J 1998;31: 394-409.
235. Ricucci D, Rocas IN, Alves FR, et al: *Apically extruded sealers: fate and influence on treatment outcome*, J Endod 2016;42: 243-249.
236. Ricucci D, Siqueira JF Jr. *Fate of the tissue in lateral canals and apical ramifications in response to pathologic conditions and treatment procedures*, J Endod 2010;36: 1-15.
237. Rocas IN, Siqueira JF Jr. *Identification of bacteria enduring endodontic treatment procedures by a combined reverse transcriptase-polymerase chain reaction and reverse-capture checkerboard approach*, J Endod 2010;36: 45-52.
238. Rodriguez-Lozano FJ, Collado-Gonzalez M, Tomás-Catalá CJ, et al: *GuttaFlow Bioseal promotes spontaneous*

Telegram: @dental_k

www.konkur.in

differentiation of human periodontal ligament stem cells into cementoblast-like cells, Dent Mater 2019;35: 114-124.

239. Sabeti M, Kermani V, Sabeti S, et al: *Significance of human cytomegalovirus and Epstein-Barr virus in inducing cytokine expression in periapical lesions*, J Endod 2012;38: 47-50.
240. Sabeti MA, Nekofar M, Motahhary P, et al: *Healing of apical periodontitis after endodontic treatment with and without obturation in dogs*, J Endod 2006;32: 628-633.
241. Sabins RA, Johnson JD, Hellstein JW. *A comparison of the cleaning efficacy of short-term sonic and ultrasonic passive irrigation after hand instrumentation in molar root canals*, J Endod 2003;29: 674-678.
242. Safavi K, Horsted P, Pascon EA, et al: *Biological evaluation of the apical dentin chip plug*, J Endod 1985;11: 18-24.
243. Safavi KE, Dowden WE, Langeland K. *Influence of delayed coronal permanent restoration on endodontic prognosis*, Endod Dent Traumatol 1987;3: 187-191.
244. Saini HR, Tewari S, Sangwan P, et al: *Effect of different apical preparation sizes on outcome of primary endodontic treatment: a randomized controlled trial*, J Endod 2012;38: 1309-1315.
245. Salles LP, Gomes-Cornelio AL, Guimaraes FC, et al: *Mineral trioxide aggregate-based endodontic sealer stimulates hydroxyapatite nucleation in human osteoblast-like cell culture*, J Endod 2012;38: 971-976.
246. Santoro V, Lozito P, Donno AD, et al: *Extrusion of endodontic filling materials: medico-legal aspects. Two cases*, Open Dent J 2009;3: 68-73.
247. Santos MD, Walker WA 3rd, Carnes DL Jr. *Evaluation of apical seal in straight canals after obturation using the*

Telegram: @dental_k

www.konkur.in

Lightspeed sectional method, J Endod 1999;25: 609-612.

248. Sargenti A. *The Sargenti N-2 method*, Dent Surv 1978;54: 55-58.
249. Sathorn C, Parashos P, Messer H. *Antibacterial efficacy of calcium hydroxide intracanal dressing: a systematic review and meta-analysis*, Int Endod J 2007;40: 2-10.
250. Sathorn C, Parashos P, Messer HH. *How useful is root canal culturing in predicting treatment outcome?*, J Endod 2007;33: 220-225.
251. Saunders EM. *The effect of variation in thermomechanical compaction techniques upon the quality of the apical seal*, Int Endod J 1989;22: 163-168.
252. Saunders EM. *In vivo findings associated with heat generation during thermomechanical compaction of gutta-percha. 2. Histological response to temperature elevation on the external surface of the root*, Int Endod J 1990;23: 268-274.
253. Scarfe WC, Levin MD, Gane D, et al: *Use of cone beam computed tomography in endodontics*, Int J Dent 2009;2009: 634567.
254. Scelza MF, Teixeira AM, Scelza P. *Decalcifying effect of EDTA-T, 10% citric acid, and 17% EDTA on root canal dentin*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 234-236.
255. Schaeffer MA, White RR, Walton RE. *Determining the optimal obturation length: a meta-analysis of literature*, J Endod 2005;31: 271-274.
256. Schilder H. *Filling root canals in three dimensions*, Dent Clin North Am 1967; 723-744.
257. Schilder H, Goodman A, Aldrich W. *The thermomechanical properties of gutta-percha. Part V. Volume changes in bulk gutta-percha as a function of*

Telegram: @dental_k

www.konkur.in

temperature and its relationship to molecular phase transformation, Oral Surg Oral Med Oral Pathol 1985;59: 285-296.

258. Schmidt KJ, Walker TL, Johnson JD, et al: *Comparison of nickel-titanium and stainless-steel spreader penetration and accessory cone fit in curved canals*, J Endod 2000;26: 42-44.
259. Segura JJ, Calvo JR, Guerrero JM, et al: *The disodium salt of EDTA inhibits the binding of vasoactive intestinal peptide to macrophage membranes: endodontic implications*, J Endod 1996;22: 337-340.
260. Seltzer S, Green DB, Weiner N, et al: *A scanning electron microscope examination of silver cones removed from endodontically treated teeth*, Oral Surg Oral Med Oral Pathol 1972;33: 589-605.
261. Sen BH, Piskin B, Demirci T. *Observation of bacteria and fungi in infected root canals and dentinal tubules by SEM*, Endod Dent Traumatol 1995;11: 6-9.
262. Sen BH, Wesselink PR, Turkun M. *The smear layer: a phenomenon in root canal therapy*, Int Endod J 1995;28: 141-148.
263. Serper A, Ucer O, Onur R, et al: *Comparative neurotoxic effects of root canal filling materials on rat sciatic nerve*, J Endod 1998;24: 592-594.
264. Shabahang S, Torabinejad M. *Treatment of teeth with open apices using mineral trioxide aggregate*, Pract Periodontics Aesthet Dent 2000;12: 315-320; quiz 22.
265. Shahravan A, Haghdoost AA, Adl A, et al: *Effect of smear layer on sealing ability of canal obturation: a systematic review and meta-analysis*, J Endod 2007;33: 96-105.
266. Shipper G, Orstavik D, Teixeira FB, et al: *An evaluation*

Telegram: @dental_k

www.konkur.in

of microbial leakage in roots filled with a thermoplastic synthetic polymer-based root canal filling material (Resilon), J Endod 2004;30: 342-347.

267. Siboni F, Taddei P, Zamparini F, et al: *Properties of BioRoot RCS, a tricalcium silicate endodontic sealer modified with povidone and polycarboxylate*, Int Endod J Suppl 2, 2017;50: e120- e136.
268. Silva Almeida LH, Moraes RR, Morgental RD, et al: *Are premixed calcium silicate-based endodontic sealers comparable to conventional materials? A systematic review of in vitro studies*, J Endod 2017;43: 527-535.
269. Simon S, Rilliard F, Berdal A, et al: *The use of mineral trioxide aggregate in one-visit apexification treatment: a prospective study*, Int Endod J 2007;40: 186-197.
270. Siqueira JF Jr, da Silva CH, Cerqueira MdD, et al: *Effectiveness of four chemical solutions in eliminating Bacillus subtilis spores on gutta-percha cones*, Endod Dent Traumatol 1998;14: 124-126.
271. Siqueira JF Jr, Guimaraes-Pinto T, Rocas IN. *Effects of chemomechanical preparation with 2.5% sodium hypochlorite and intracanal medication with calcium hydroxide on cultivable bacteria in infected root canals*, J Endod 2007;33: 800-805.
272. Siqueira JF Jr, Paiva SS, Rocas IN. *Reduction in the cultivable bacterial populations in infected root canals by a chlorhexidine-based antimicrobial protocol*, J Endod 2007;33: 541-547.
273. Siqueira JF Jr, Rocas IN. *Diversity of endodontic microbiota revisited*, J Dent Res 2009;88: 969-981.
274. Sjogren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int

Telegram: @dental_k

www.konkur.in

Endod J 1997;30: 297-306.
275. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498-504.
276. Sonat B, Dalat D, Gunhan O. *Periapical tissue reaction to root fillings with Sealapex*, Int Endod J 1990;23: 46-52.
277. Southard DW, Rooney TP. *Effective one-visit therapy for the acute periapical abscess*, J Endod 1984;10: 580-583.
278. Spangberg LS, Barbosa SV, Lavigne GD. *AH 26 releases formaldehyde*, J Endod 1993;19: 596-598.
279. Stamos DE, Gutmann JL, Gettleman BH. *In vivo evaluation of root canal sealer distribution*, J Endod 1995;21: 177-179.
280. Stevens RW, Strother JM, McClanahan SB. *Leakage and sealer penetration in smear-free dentin after a final rinse with 95% ethanol*, J Endod 2006;32: 785-788.
281. Su Y, Wang C, Ye L. *Healing rate and post-obturation pain of single- versus multiple-visit endodontic treatment for infected root canals: a systematic review*, J Endod 2011;37: 125-132.
282. Swartz DB, Skidmore AE, Griffin JA Jr. *Twenty years of endodontic success and failure*, J Endod 1983;9: 198-202.
283. Sweatman TL, Baumgartner JC, Sakaguchi RL. *Radicular temperatures associated with thermoplasticized gutta-percha*, J Endod 2001;27: 512-515.
284. Tagger M, Tagger E, Kfir A. *Release of calcium and hydroxyl ions from set endodontic sealers containing calcium hydroxide*, J Endod 1988;14: 588-591.
285. Tanomaru-Filho M, Jorge EG, Guerreiro Tanomaru JM, et al: *Radiopacity evaluation of new root canal filling materials by digitalization of images*, J Endod 2007;33: 249-251.

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286. Tanomaru Filho M, Leonardo MR, Silva LA, et al: *Inflammatory response to different endodontic irrigating solutions*, Int Endod J 2002;35: 735-739.
287. Tay FR, Loushine RJ, Monticelli F, et al: *Effectiveness of resin-coated gutta-percha cones and a dual-cured, hydrophilic methacrylate resin-based sealer in obturating root canals*, J Endod 2005;31: 659-664.
288. Tay FR, Pashley DH. *Monoblocks in root canals: a hypothetical or a tangible goal*, J Endod 2007;33: 391-398.
289. Taylor JK1, Jeansonne BG, Lemon RR. *Coronal leakage: effects of smear layer, obturation technique, and sealer*, J Endod 1997;23: 508-512.
290. Tewary S, Luzzo J, Hartwell G. *Endodontic radiography: who is reading the digital radiograph?*, J Endod 2011;37: 919-921.
291. Thakur S, Emil J, Paulaian B. *Evaluation of mineral trioxide aggregate as root canal sealer: A clinical study*, J Conserv Dent 2013;16: 494-498.
292. Torabinejad M, Parirokh M, Dummer PMH. *Mineral trioxide aggregate and other bioactive endodontic cements: an updated overview - part II: other clinical applications and complications*, Int Endod J 2018;51: 284-317.
293. Torabinejad M, Ung B, Kettering JD. *In vitro bacterial penetration of coronally unsealed endodontically treated teeth*, J Endod 1990;16: 566-569.
294. Trope M, Chow E, Nissan R. *In vitro endotoxin penetration of coronally unsealed endodontically treated teeth*, Endod Dent Traumatol 1995;11: 90-94.
295. Uranga A, Blum JY, Esber S, et al: *A comparative study of four coronal obturation materials in endodontic*

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treatment, J Endod 1999;25: 178-180.

296. Urban K, Neuhaus J, Donnermeyer D, et al: *Solubility and pH value of 3 different root canal sealers: a long-term investigation*, J Endod 2018;44: 1736-1740.
297. Vassiliadis L, Liolios E, Kouvas V, et al: *Effect of smear layer on coronal microleakage*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 315-320.
298. Vera J, Siqueira JF Jr, Ricucci D, et al: *One- versus two-visit endodontic treatment of teeth with apical periodontitis: a histobacteriologic study*, J Endod 2012;38: 1040-1052.
299. Vouzara T, Koulaouzidou E, Ziouti F, et al: *Combined and independent cytotoxicity of sodium hypochlorite, ethylenediaminetetraacetic acid and chlorhexidine*, Int Endod J 2016;49: 764-773.
300. Walker TL, del Rio CE. *Histological evaluation of ultrasonic debridement comparing sodium hypochlorite and water*, J Endod 1991;17: 66-71.
301. Walsh RM, He J, Schweitzer J, et al: *Bioactive endodontic materials for everyday use: a review*, Gen Dent 2018;66: 48-51.
302. Wang CS, Arnold RR, Trope M, et al: *Clinical efficiency of 2% chlorhexidine gel in reducing intracanal bacteria*, J Endod 2007;33: 1283-1289.
303. Weisenseel JA Jr, Hicks ML, Pelleu GB Jr. *Calcium hydroxide as an apical barrier*, J Endod 1987;13: 1-5.
304. Wennberg A, Orstavik D. *Adhesion of root canal sealers to bovine dentine and gutta-percha*, Int Endod J 1990;23: 13-19.
305. White RR, Goldman M, Lin PS. *The influence of the smeared layer upon dentinal tubule penetration by plastic filling materials*, J Endod 1984;10: 558-562.

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306. Wiemann AH, Wilcox LR. *In vitro evaluation of four methods of sealer placement*, J Endod 1991;17: 444-447.
307. Wilcox LR, Roskelley C, Sutton T. *The relationship of root canal enlargement to finger-spreader induced vertical root fracture*, J Endod 1997;23: 533-534.
308. Wolcott J, Himel VT, Powell W, et al: *Effect of two obturation techniques on the filling of lateral canals and the main canal*, J Endod 1997;23: 632-635.
309. Wu M, van der Sluis LW, Wesselink PR. *A preliminary study of the percentage of gutta-percha-filled area in the apical canal filled with vertically compacted warm gutta-percha*, Int Endod J 2002;35: 527-535.
310. Wu MK, Fan B, Wesselink PR. *Leakage along apical root fillings in curved root canals, Part I: effects of apical transportation on seal of root fillings*, J Endod 2000;26: 210-216.
311. Wu MK, Kast'akova A, Wesselink PR. *Quality of cold and warm gutta-percha fillings in oval canals in mandibular premolars*, Int Endod J 2001;34: 485-491.
312. Wu MK, Ozok AR, Wesselink PR. *Sealer distribution in root canals obturated by three techniques*, Int Endod J 2000;33: 340-345.
313. Wu MK, Shemesh H, Wesselink PR. *Limitations of previously published systematic reviews evaluating the outcome of endodontic treatment*, Int Endod J 2009;42: 656-666.
314. Wu MK, Wesselink PR. *Endodontic leakage studies reconsidered. Part I. Methodology, application and relevance*, Int Endod J 1993;26: 37-43.
315. Wu MK, Wesselink PR. *A primary observation on the preparation and obturation of oval canals*, Int Endod J 2001;34: 137-141.

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316. Yamada RS, Armas A, Goldman M, et al: *A scanning electron microscopic comparison of a high volume final flush with several irrigating solutions: Part 3*, J Endod 1983;9: 137-142.
317. Yeung P, Liewehr FR, Moon PC. *A quantitative comparison of the fill density of MTA produced by two placement techniques*, J Endod 2006;32: 456-459.
318. Youngson CC, Nattress BR, Manogue M, et al: *In vitro radiographic representation of the extent of voids within obturated root canals*, Int Endod J 1995;28: 77-81.
319. Zhang K, Tay FR, Kim YK, et al: *The effect of initial irrigation with two different sodium hypochlorite concentrations on the erosion of instrumented radicular dentin*, Dent Mater 2010;26: 514-523.
320. Zielinski TM, Baumgartner JC, Marshall JG. *An evaluation of Guttaflow and gutta-percha in the filling of lateral grooves and depressions*, J Endod 2008;34: 295-298.
321. Zmener O, Banegas G. *Clinical experience of root canal filling by ultrasonic condensation of gutta-percha*, Endod Dent Traumatol 1999;15: 57-59.
322. Zmener O, Hilu R, Scavo R. *Compatibility between standardized endodontic finger spreaders and accessory gutta-percha cones*, Endod Dent Traumatol 1996;12: 237-239.
323. Zmener O, Pameijer CH. *Clinical and radiographic evaluation of a resin-based root canal sealer: 10-year recall data*, Int J Dent 2012;2012: 763248.
324. Zmener O, Pameijer CH, Serrano SA, et al: *Significance of moist root canal dentin with the use of methacrylate-based endodontic sealers: an in vitro coronal dye leakage study*, J Endod 2008;34: 76-79.

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325. Zogheib C, Naaman A, Sigurdsson A, et al: *Comparative micro-computed tomographic evaluation of two carrier-based obturation systems*, Clin Oral Investig 2013;17: 1879-1883.

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10: Nonsurgical retreatment

Robert S. Roda, Bradley H. Gettleman, Scott C. Johnson

CHAPTER OUTLINE

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Nonsurgical root canal therapy has become a routine procedure in modern dentistry. Recent technical and scientific advances in endodontics have resulted in the retention of millions of teeth that would otherwise be lost. Even as recent advances in surgical and prosthetic restorative care have made tooth replacement less onerous than in the past, it is universally accepted that a natural tooth with a good prognosis is a superior choice to loss and replacement.

Unfortunately, not all treatments result in optimum long-term healing. Given the large numbers of treatments performed, the very small rate of unsuccessful outcomes translates into relatively large numbers of patients requiring further treatment. Dental clinicians should be able to diagnose persistent or reintroduced endodontic disease and be aware of the options for treatment. If they wish to approach treating these teeth, they should have the appropriate armamentarium and be capable of performing these very specialized techniques at the highest level (Fig. 10.1). Also, clinicians must always have a scientifically sound, evidence-based rationale for every treatment decision that is made so that they may best serve the patients who entrust them with their care. The purpose of this chapter is to provide information to allow the reader to maximize the likelihood of success in the treatment of persistent endodontic disease.

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FIG. 10.1 Some of the armamentarium needed to perform retreatment at the highest level.

A set of medicinal kits, Gonon universal post extractor kit with an extracting plier, five trephines, five corresponding tubular taps, torque bar, tube spacers and rubber bumpers of varying diameters, Thomas screw post dental kit, IRS kit with core drills etcetera are kept on a blue platform.

Etiology of posttreatment disease

In the past, undesirable outcomes of endodontic therapy were described as failures. Clinicians quote failure rates based on published success/failure studies. Using the words “success” and “failure” may be a holdover from a time when clinicians felt they needed to congratulate themselves on their successes and blame themselves for the failures of their treatment endeavors. This thought process does not reflect reality and can be potentially destructive. There are many instances where treatments performed at the highest level of clinical competence result in an undesirable outcome, and there are other instances where a procedure is performed well below a scientifically acceptable standard and yet provides long-term success.[232] We must begin to dissect the science from emotion and ego, and this separation may start with nomenclature. Friedman states that “most patients can relate to the concept of disease-treatment-healing, whereas failure, apart from being a negative and relative term, does not imply the necessity to pursue

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treatment."[73] He has suggested using the term *posttreatment disease* to describe those cases that would previously have been referred to as treatment failures. This will be the term used in the remainder of this chapter to describe persistent or reintroduced endodontic disease.

Approximately 22.3 million root canal procedures were performed in 2006[59] and, with success rates varying between 86% and 98%,[74,75] it has proven to be a very reliable treatment option. Conversely, the incidence of posttreatment disease, although small, translates into a very large number of cases where further treatment is needed. When faced with such a situation, the clinician must determine the etiology of the persistent pathosis and devise a rationale and strategy for treatment.

There are many causes for "failure" of initial endodontic therapy that have been described in the endodontic literature (Fig. 10.2). These include iatrogenic procedural errors such as poor access cavity design, untreated canals (both major and accessory),[298] canals that are poorly cleaned and obturated,[43,125] complications of instrumentation (ledges, perforations, or separated instruments),[259] and overextensions of root filling materials.[183] Coronal leakage[145,162,224,264,278] has also been blamed for posttreatment disease as has persistent intra- and extracanal infection[185,205,244,260] and radicular cysts.[181] These etiologies may be obvious at the time of diagnosing the diseased, root-filled tooth, or they may remain uncertain until the completion of successful therapy. Occasionally, the cause of posttreatment disease may take years to become discernable or may ultimately never be known. The most important causative factors for the clinician, however, are those related to treatment planning and determination of prognosis. In order to treatment plan effectively, the clinician may place the etiological factors into four groups (Fig. 10.3).[259]

1. Persistent or reintroduced intraradicular microorganisms
2. Extraradicular infection
3. Foreign body reaction
4. True cysts

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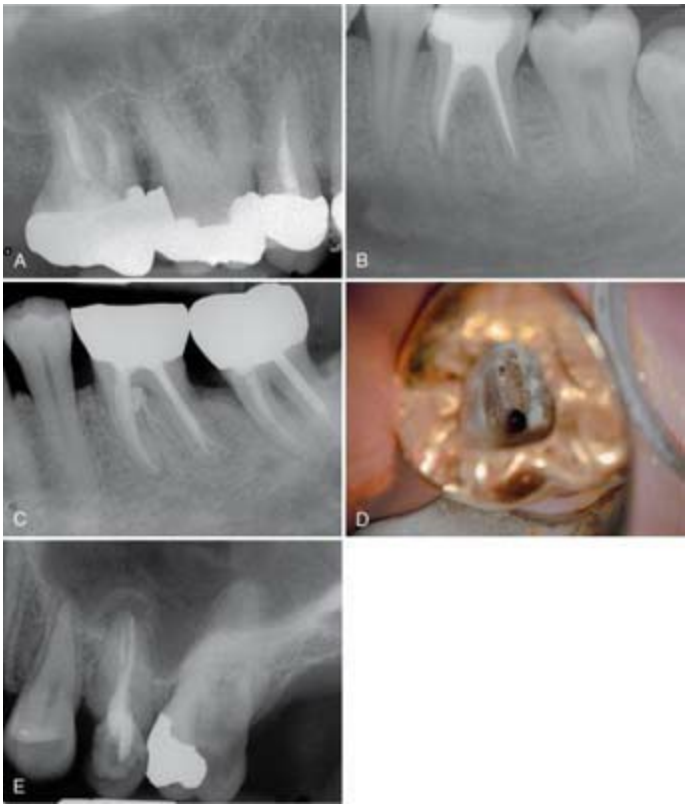

FIG. 10.2 Clinical presentations of posttreatment disease. **A,** Canals that are poorly cleaned, shaped, and obturated. **B,** Mesial canal with apical transport, ledge, and zip perforation. **C,** Strip perforation of the mesial root. **D,** Missed MB2 canal in an upper molar. **E,** Suspected coronal leakage of bacteria and a separated file.

A) Radiograph of three teeth shows radiopaque crown with white irregular patches.

B) Radiograph of tooth shows radiopaque crown and root canals.

C) Radiograph of two teeth show radiopaque crown and root canals with slightly slanting root tips.

D) Close-up view of a horizontal section of an upper molar shows a golden ring shaped circumference around the tooth.

E) Radiograph of three teeth shows first molar with white radiopaque patch on the crown and root canal of the other tooth is radiolucent.

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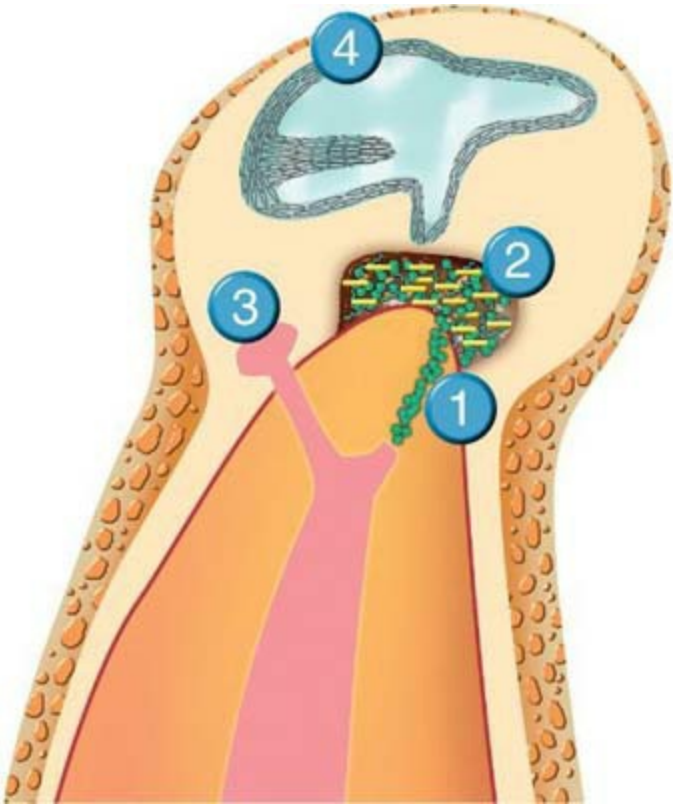

FIG. 10.3 The causes of posttreatment disease. *1*, Intraradicular microorganisms. *2*, Extraradicular infection. *3*, Foreign body reaction. *4*, True cysts.

A cutaway diagram of a molar shows the causes for posttreatment disease labeled from top to bottom numbered as 4, 2, 3 and 1.

Source: (Redrawn with permission from Sundqvist G, Figdor D: Endodontic treatment of apical periodontitis. In Orstavik D, Pitt-Ford TR, editors: *Essential endodontology, prevention and treatment of apical periodontitis*, London, 1998, Blackwell, 1998, p. 260. Courtesy Dentsply Endodontics.)

1. *Persistent or reintroduced intraradicular microorganisms.* When the root canal space and dentinal tubules are contaminated with microorganisms or their by-products, and if these pathogens can contact the periradicular tissues, apical periodontitis ensues. As stated earlier, inadequate cleaning, shaping, obturation, and final restoration of an endodontically diseased tooth can lead to posttreatment disease. If initial endodontic therapy does not render the canal space free of bacteria, or if the obturation does not adequately entomb those that may remain, or if new microorganisms can reenter the cleaned and sealed canal space, then posttreatment disease can and

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usually does occur. In fact, it has been asserted that persistent or reintroduced microorganisms are the major cause of posttreatment disease.[182] Many iatrogenic treatment complications, such as creation of a ledge or separation of an instrument, result in persistence of bacteria in the canal system. It is not the complication itself, however that results in persistent disease; rather, it is the inability to remove or entomb the microorganisms present that creates the pathologic state. Although infected root canals of endodontically untreated teeth generally contain a polymicrobial, predominantly anaerobic flora,[258] cultures of infected, previously root-filled teeth produce very few or even one single species. The infecting flora are predominantly gram positive, not anaerobic, and a very commonly isolated species is *Enterococcus faecalis*,[80,206] which has been shown to be very resistant to canal disinfection regimens.[21,37] One recent study, however, hints at greater diversity in the microflora of infected, root filled teeth than had been detected before and may show a correlation between increasing diversity and specific clinical features.[219] Interestingly, if the previous root canal treatment is done so poorly that the canal space contains no obturating material in the apical half of the root canal space, its flora is more typical of the untreated necrotic infected pulp than that of classic "failed" root canal therapy.[259] Though posttreatment disease has been primarily blamed on bacteria in the root canal system, fungi, notably *Candida albicans*, are frequently found in persistent endodontic infections and may be responsible for the recalcitrant lesion.[242]

2. *Extraradicular infection.* Occasionally, bacterial cells can invade the periradicular tissues either by direct spread of infection from the root canal space, via contaminated periodontal pockets that communicate with the apical area,[240] extrusion of infected dentin chips,[112] or by contamination with overextended, infected endodontic instruments.[288] Usually, the host response will destroy these organisms, but some microorganisms are able to resist the immune defenses and persist in the periradicular tissues, sometimes by producing an extracellular matrix or protective plaque.[279] It has also been shown[185,244,260] that two species of microorganisms, *Actinomyces israelii* and *Propionibacterium propionicum*, can exist in the periapical tissues and may prevent healing after root canal therapy.

3. *Foreign body reaction.* Occasionally, persistent endodontic disease occurs in the absence of discernable microorganisms and has been attributed

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to the presence of foreign material in the periradicular area. Several materials have been associated with inflammatory responses including lentil beans[239] and cellulose fibers from paper points.[139] In the seemingly endless debate about which endodontic obturation technique is superior, there has been much discussion about the effect of overextended root canal filling materials upon apical healing. Outcomes assessments generally show that filling material extrusion (root filling flush to the radiographic apex or gross overextension) leads to a lower incidence of healing.[72,243] Many of these cases involved not only overextension but also inadequate canal preparation and compaction of the root filling whereby persistent bacteria remaining in the canal space could leak out. Gutta-percha and sealers are usually well tolerated by the apical tissues, and if the tissues have not been inoculated with microorganisms by vigorous overinstrumentation, then healing in the presence of overextended filling materials can still occur.[72,79,152]

4. *True cysts.* Cysts form in the periradicular tissues when retained embryonic epithelium begins to proliferate due to the presence of chronic inflammation. The epithelial cell rests of Malassez are the source of the epithelium, and cyst formation may be an attempt to help separate the inflammatory stimulus from the surrounding bone.[204] The incidence of periapical cysts has been reported to be 15% to 42% of all periapical lesions,[181,251] and determining whether periapical radiolucency is a cyst or the more common periapical granuloma cannot be done radiographically.[28] There are two types of periapical cysts: the periapical true cyst and the periapical pocket cyst. True cysts have a contained cavity or lumen within a continuous epithelial lining, whereas, with pocket cysts, the lumen is open to the root canal of the affected tooth. True cysts, due to their self-sustaining nature, probably do not heal following nonsurgical endodontic therapy[125,184] and usually require surgical enucleation (Fig. 10.4).

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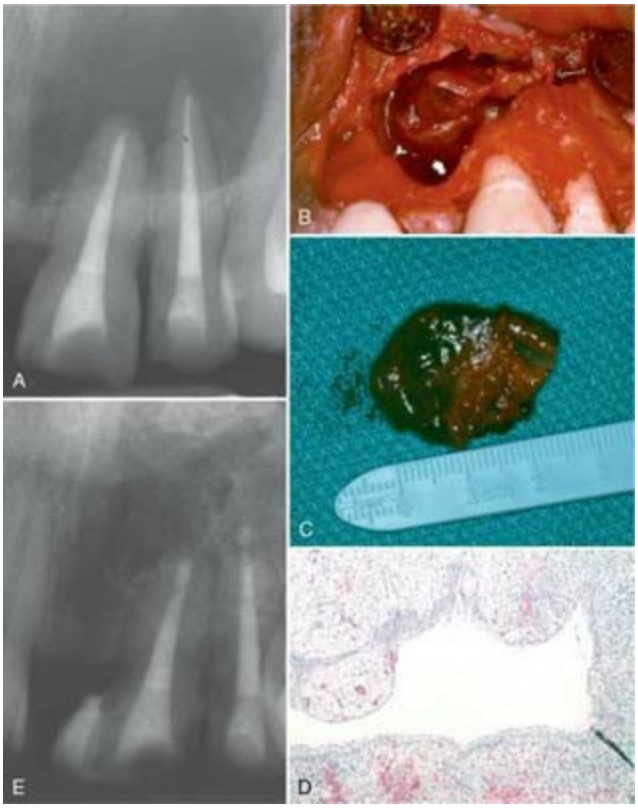

FIG. 10.4 **A,** Apparently good nonsurgical retreatment with large persistent lesion. **B,** Surgical exposure of apical lesion in situ. **C,** Large lesion removed in toto. **D,** Histopathologic section confirming cystic nature of the lesion. **E,** Four-year postoperative film showing apical scar formation due to the large size of the lesion. The teeth were asymptomatic and in function.

A) Radiograph of two teeth show radiolucent root canals.

B) A surgically exposed gum shows a lesion formed over two upper teeth with a wire passing through it.

C) Radiograph of three teeth shows radiopaque root canal and root tip of first two teeth and radiolucent crown of the third tooth.

D) An oval-shaped lesion approximately 3 centimeter wide with a ruler next to it.

E) Scan of the gum shows a large irregular patch in the center.

When a patient presents with posttreatment disease, clinical decision-making depends upon determining the cause of the persistent disease and then assessing how best to treat the pathologic condition. In the following section, the reader will be presented with a rationale and methods for

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performing endodontic diagnosis, which will allow for the greatest likelihood of a successful outcome.

Diagnosis of posttreatment disease

It has been stated that "there may be different ways of treating a disease, however there can be but one correct diagnosis."[11] The proper diagnosis is probably the most important portion of any endodontic procedure. This is not as bold of a statement as one may first suspect when consideration is given to what the patient may undergo if treatment is performed based on an incorrect diagnosis (Fig. 10.5). For the clinician to make a correct diagnosis, they must rule out nonodontogenic etiology, perform all of the appropriate tests, properly interpret the patient's responses to these tests, derive at a definitive diagnosis, and decide on treatment options. When performing a diagnosis in endodontic cases where there is no history of previous endodontic therapy, both a pulpal and periradicular diagnosis are necessary. In cases of persistent disease, the diagnosis may not be as straightforward as the clinician may be dealing with partially treated pulp canals and missed canals as well as many other types of problems associated with the previous treatment. These must be included in the diagnostic description for each case.

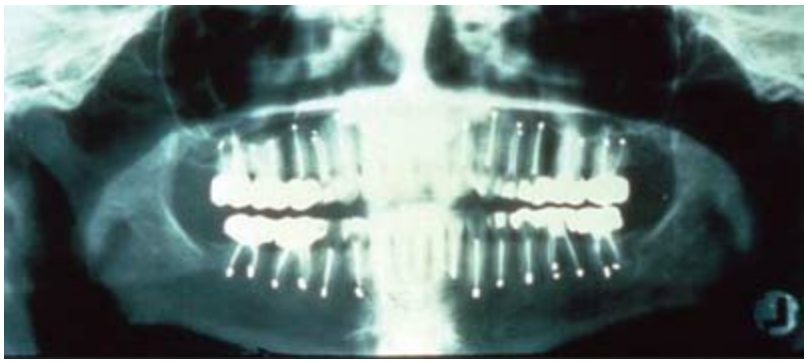

FIG. 10.5 This patient was misdiagnosed for years and underwent unnecessary endodontic therapy. The actual cause of the patient's complaint was nondental pain.

Radiograph of frontal upper and lower set of teeth. The crowns, root canals and root tips with tiny dots like attachments are radiopaque while the pulp of the teeth is radiolucent.

Source: (Courtesy Dr. Ramesh Kuba.)

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Endodontic diagnosis has been thoroughly discussed in an earlier chapter and the reader is referred there for further details on these procedures. The diagnostic method requires collecting subjective information, developing objective findings, and using these to arrive at a diagnosis and plan of treatment.

The subjective information is collected by questioning the patient and then actively listening to the responses. Of particular interest in cases of suspected posttreatment disease is whether the patient recalls the use of aseptic techniques during the previous endodontic therapy. If a rubber dam was not used, for example, and this can be confirmed with a call to the previous clinician, nonsurgical retreatment will almost certainly be necessary since the canals can be assumed to be contaminated regardless of how esthetically pleasing the previously filled case may appear on the radiograph. The diagnostician should be careful to avoid or to minimize communicating to the patient any negative feelings he or she has toward the previous treatment, however bad it may seem. This approach allows the patient to become more comfortable with the current clinician and the proposed corrective treatment. An irate patient is an irate patient, and negativity will color his or her emotional state, level of trust, and ability to accept current or future treatment plans. If the patient asks a direct question about the previous treatment, an honest answer is necessary, but avoid the temptation to imply superiority by disparaging the former clinician. To state the situation honestly and correctly without being inflammatory, use a sentence such as, “Well, it may be that your previous dentist (endodontist) had some difficulty with that tooth. Let’s see if we can figure out what could have been the problem.”

Following a thorough review of the patient’s health history, the next step is to gather all the objective information needed to obtain an accurate diagnosis. This information will include the clinical and radiographic examination. The clinical examination should include a visual extraoral and intraoral examination, and a thorough periodontal evaluation. Visual examination is greatly aided by magnification and illumination, which can allow the clinician to identify significant conditions invisible to the naked eye, such as very fine fractures on root surfaces (Fig. 10.6). Exposed dentin from recession and narrow probing defects may be the result of an endodontic infection draining through the sulcus; however, they are sometimes indicative

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of vertical root fracture.[46] The presence of occlusal wear facets indicates the presence of occlusal trauma that may complicate diagnosis and treatment outcome by predisposing the tooth to fracture,[101] and has been associated with posttreatment disease.[126]

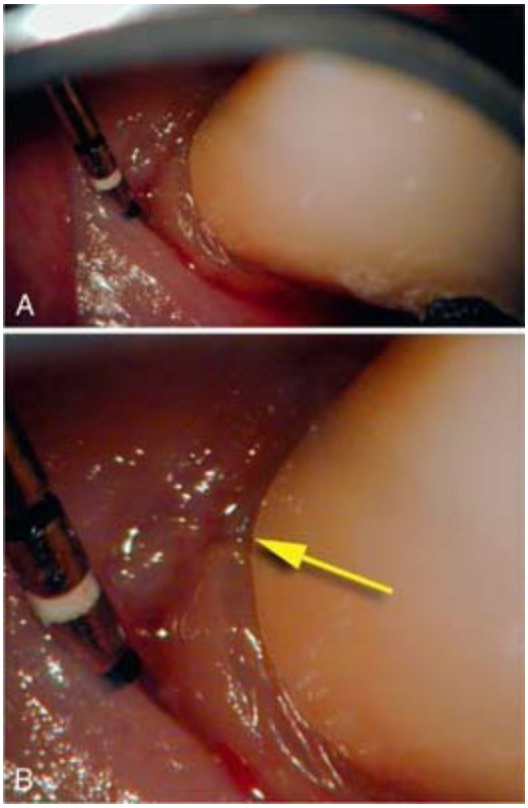

FIG. 10.6 **A,** Buccal aspect of a premolar with posttreatment disease. **B,** Higher magnification reveals a vertical fracture *(arrow)*.

A) Close-up view of a gum and a tooth with dark patches around it. Water is sprayed into the mouth using a water spray nozzle.

B) Close-up view of a gum shows an arrow pointing to a vertical line depicting fractured tooth on the root surface.

Radiographic assessment is obligatory. Even though radiographs may be a critical aid to the clinician, they should never be the sole support for a conclusive diagnosis. They are only one piece of the puzzle in determining endodontic etiology.[67] In cases with previous endodontic therapy, radiographs are very useful in the evaluation of caries, defective restorations, periodontal health, the quality of the obturation, existence of missed canals,

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impediments to instrumentation, periradicular pathosis, perforations, fractures,[267] resorption, and canal anatomy. Radiographs should be properly exposed and have a sharp, clear image. They should include the tooth and surrounding tissues, and multiple angulated images should be used to determine endodontic etiologies using the buccal object moves most rule (Fig. 10.7).[90] Bitewing radiographs are useful in determining periodontal bone height and to look for caries or fractures. All sinus tracts should be traced with a cone of gutta-percha followed by a radiograph to localize their origin.[124]

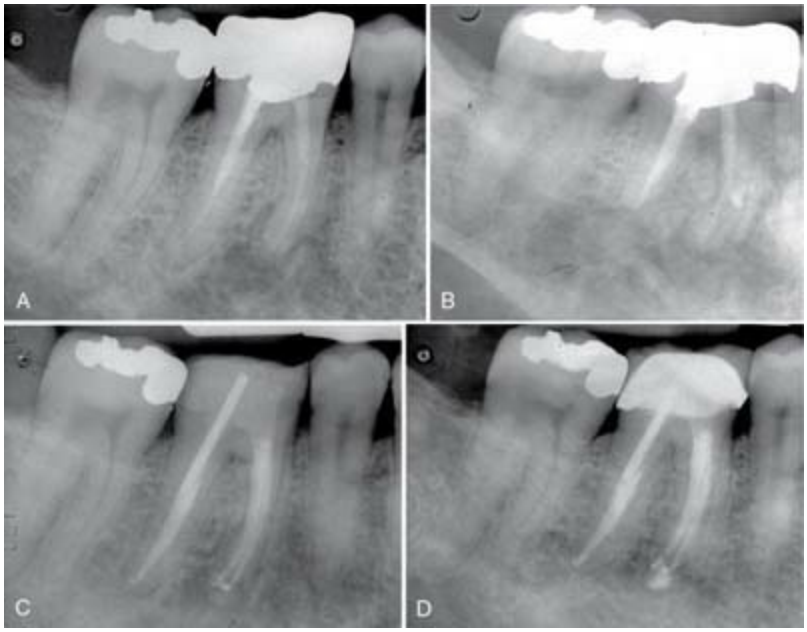

FIG. 10.7 **A,** Posttreatment disease. Previous endodontic therapy performed 3 years previously. **B,** Distal angle radiograph reveals asymmetry indicating the presence of an untreated mesiobuccal canal. **C,** Immediate postobturation film showing treated MB canal. **D,** Fourteen-month postoperative view. The patient was asymptomatic.

A) Radiograph of teeth show white radiopaque patch on the entire crown of first molar and radiopaque white patch on a part of crown of second molar. The pulp is radiolucent.

B) Radiograph of teeth shows white radiopaque patch on the entire crown of first molar and a part of a canal and radiopaque white patch on a part of the crown of second molar. The pulp is partially radiolucent.

C) Radiograph of teeth shows radiolucent root tips of the first molar and white radiopaque patch on a part of crown of second molar.

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D) Radiograph of teeth shows radiopaque crown and root canal of the first molar and radiopaque white patch on a part of crown of second molar.

Recently, cone-beam computed tomography (CBCT) has been introduced into endodontics, and its usefulness in the management of endodontic retreatment is unquestioned.[209] It has provided a quantum leap in our ability to determine the causes of posttreatment apical periodontitis by giving the clinician, for the first time, the ability to easily, safely, and inexpensively visualize the tooth and surrounding structures in three dimensions. Several recent studies have shown that if clinicians are given all the collected diagnostic data (including periapical radiographs) for a patient, and they make a treatment plan, if they are then given a CBCT volume of the same patient, they change the treatment plan in up to 62% of the cases.[58,177,210] This indicates the unprecedented diagnostic power of CBCT technology. CBCT use in endodontics is discussed in detail in another chapter, but when faced with a tooth needing retreatment, it is especially helpful. The CBCT allows the clinician to determine the true size, extent, and position of periapical and resorptive lesions as well as giving added information about tooth fractures, missed canals, root canal anatomy, and the nature of the alveolar bone topography around teeth.[48] CBCT technology has greatly enhanced presurgical diagnosis and treatment planning, since the relationship of adjacent anatomical structures such as the maxillary sinus and inferior alveolar nerve to the root apices can be clearly visualized. This helps the clinician to decide on when to perform endodontic retreatment surgically or nonsurgically. CBCT is more accurate than periapical radiography in the diagnosis of apical periodontitis, and it can reveal the details of the lesions and adjacent structures thus providing enhanced clinical diagnosis and treatment planning.[48,150,197,198]

There are many manufacturers and brands of CBCT machines on the market today, but the most useful ones for endodontic retreatment are those that produce the clearest image with the highest resolution.[172] These would be the small field-of-view (FOV) machines that image a small volume and use the smallest picture element (voxel) dimensions available. Radiation exposure to the patient with these machines is in the range of 23 to 488 μSv[157] which is very small, but the ALARA (as low as reasonably

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achievable) principle applies and so its use in every diagnostic case is not encouraged. In a joint position statement in 2010, The American Association of Endodontists and the American Association of Oral and Maxillofacial Radiologists state that “CBCT should only be used when the question for which imaging is required cannot be answered adequately by lower dose conventional dental radiography or alternate imaging modalities.”[1] When managing posttreatment disease, however, almost every case will benefit from the use of three-dimensional (3D) imaging and this has been recommended in the 2016 update to the AAE/AAOMR guidelines.[2]

One major cause of posttreatment apical periodontitis is untreated canals, and the CBCT gives an unprecedented ability to discover those (Fig. 10.8). In a recent study, endodontist evaluators failed to identify at least one root canal system in approximately 4 of 10 teeth when using images obtained by conventional digital radiography compared to images from cone-beam computed tomography.[169] Another recent study showed that significantly more MB2 canals were located when a preoperative CBCT image was available.[257]

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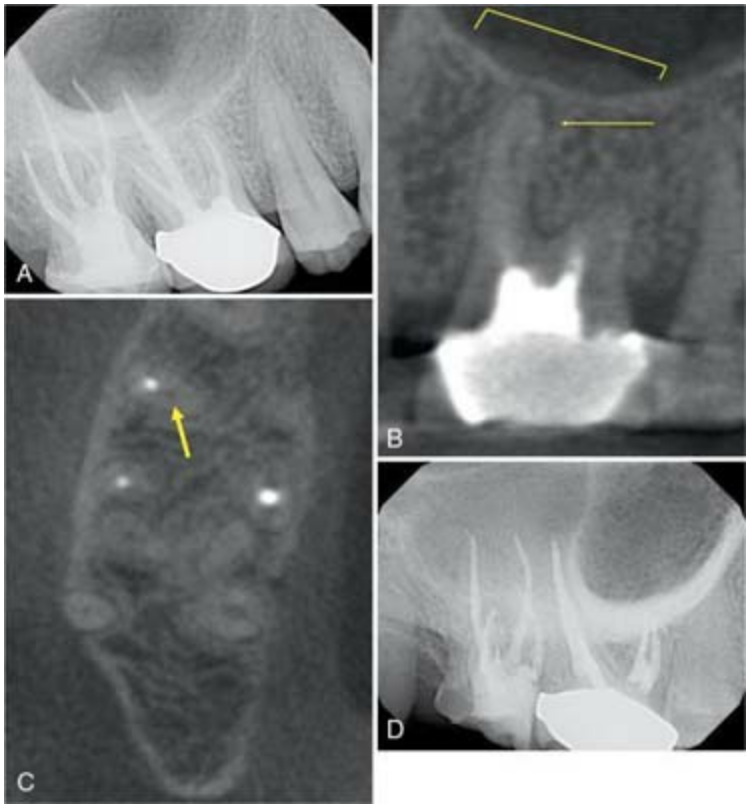

FIG. 10.8 **A,** Preoperative radiograph of symptomatic tooth #3. **B,** Sagittal slice of tooth #3 showing periodontal ligament thickening and associated sinus mucosal thickening. **C,** Axial slice showing untreated MB2 canal *(arrow)*. **D,** Treated case.

A) Radiograph shows circular dark patch at the root tips of first molar. The crown is radiopaque while the root tips are partially radiolucent.

B) Radiograph of the sagittal view of tooth shows black outlines formed around the gum and pulp of the tooth. The crown is radiolucent with a radiopaque neck.

C) Radiograph of horizontal section of canal shows three radiopaque dots where an arrow points to one of the dots.

D) Radiograph shows circular dark patch at the root tips of first molar. The crown and the root canal of the tooth are radiopaque.

Avoiding treatment that will lead to a predictable failure is beneficial for both the clinician and the patient. The ability to obtain 3D images of teeth will give the clinician help in avoiding these mishaps. The diagnosis of root fractures frequently frustrates the clinician, as a definitive diagnosis is often difficult and treating these teeth has a high likelihood of a poor outcome.

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While visualizing root fractures in teeth with root fillings is still not predictable using CBCT,[51,57,107,136,265] the patterns of bone loss indicative of root fracture can sometimes be seen[308] and this helps the clinician to infer their presence. The prognosis for the treatment of root resorption is directly related to the extent of the resorption, and this usually cannot be determined accurately using conventional radiography. Using small FOV CBCT, however, the extent of the lesions and the prognosis can be determined,[63] usually saving the patient from an exploratory procedure that may be doomed to fail (Fig. 10.9).

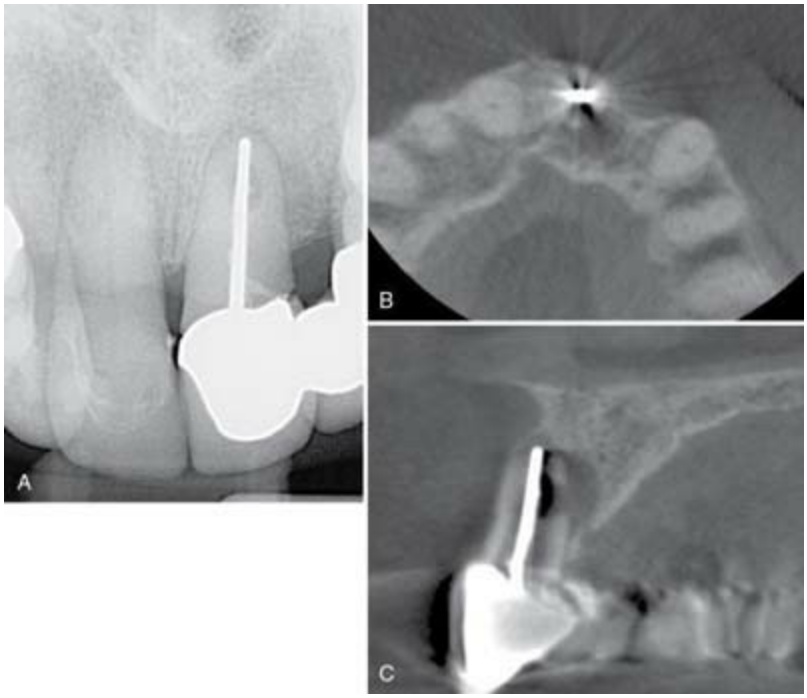

FIG. 10.9 **A,** Preoperative radiograph showing suspected small area of resorption associated with an endodontically treated tooth filled with a silver cone. **B,** Axial slice showing how beam hardening artifact from the metallic root filling obscures the image. **C,** Sagittal view showing very large palatally oriented external root resorption. The prognosis for retention of this tooth was poor and the patient elected to extract it.

A) Radiograph of teeth with a radiopaque white outline of the silver cone.

B) A horizontal section of upper palate shows posterior view of teeth with a beam of light over a black patch in a molar.

C) Radiograph of sagittal view of set of teeth with radiopaque white outline of the silver cone. Part of the head of the silver cone is radiolucent.

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The use of CBCT for preoperative assessment also has allowed for the possibility of treating only those roots in a multirooted tooth that show disease.[192] This concept of selective root retreatment has been advocated to minimize needed retreatment procedures and limit therapy to only those roots that show evidence of disease. This method, it is argued, gives nonsurgical retreatment a more selective approach, much like apical surgery where only diseased roots are treated. More research would need to be done in this area to validate this interesting development.

Though most clinicians believe that CBCT is not necessary for every patient treated, there are many retreatment situations where the additional information gained (relative to conventional radiography) is extremely valuable. Specific protocols for use are being developed,[2] but they are continuously being updated, so the authors recommend that the clinician use available guidelines and their own best judgment when deciding when to use this new technology.

Comparative testing is the next procedure performed in order to collect objective information about the pulpal and periradicular status. Most useful are the periradicular tests that include percussion, bite, and palpation.[287] These will allow the diagnostician to begin developing a sense of the status of the periradicular tissues. These tests are of great importance anytime an endodontic diagnosis is needed. However, they are of even greater importance when evaluating teeth that have been previously treated with endodontic therapy due to the lack of significant and consistent evidence that can be gained from pulp vitality tests in these cases. If a tooth exhibits percussion tenderness, it may be due to persistent endodontic disease, but recent trauma or occlusal trauma may also cause this finding[101] as can periodontal disease.[287]

Pulp vitality tests are often of little value when examining teeth with previous endodontic therapy. However, if the patient’s chief complaint reveals the need for these tests, they must be performed. When there is vital tissue remaining in the canals of a previously root-filled tooth, either by way of a completely missed canal or from an improperly cleaned canal, patients may complain of sensitivity to heat or cold.[101] Pulp vitality tests should then be performed to assess the situation. They are also useful in testing adjacent

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and opposing nonendodontically treated teeth to rule out those as etiologies for poorly localized pain. Once the tissue is removed from the pulp chamber after root canal therapy, the results of these tests should almost always be negative, even with radicular pulp remaining. Thus a negative response with previously treated teeth is not necessarily conclusive; whereas a positive response usually means there is responsive pulp tissue remaining in the tooth.[101] Care is always warranted in interpreting pulp test results, however, since false positive and negative results may occur.[231] As with cold tests, the same limits apply to heat tests as far as the reasons for false results and accuracy relative to retreatment cases.

The remaining pulp vitality tests, electric pulp test, test cavity, and direct mechanical dentin stimulation, are of even lesser value than thermal testing when evaluating teeth that have already received endodontic therapy. These are usually precluded by the existing restoration or endodontic therapy.

When all diagnostic information is collected, a diagnosis must be developed. It is important to record the diagnosis in the patient's record so that anyone reading the record can discern the clinician's rationale for treatment. The pulpal diagnosis will usually be recorded as previous endodontic treatment, but the periradicular diagnosis will vary depending on the clinical picture presented. In the case of previous endodontic treatment, however, a brief note about the suspected etiology of the persistent disease is warranted.

Treatment planning

Once the diagnosis is complete, the cause of the persistent disease will usually become apparent. At this point in the clinical process, information must be given to the patient by the clinician as to what treatment options are available and the likely outcomes of each choice. The patient is then allowed to decide based upon his or her own perceptions of the options not by the clinician's opinion as to what is "best" for the patient. The reader is reminded, however, that if the cause of the posttreatment condition remains unknown despite thorough diagnostic workup, then any decision results in an empirical, "trial and error" type of treatment. This approach should be avoided if possible, and prior to definitive treatment, consultation with an

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endodontist or other colleague is in order. This consultation may be as simple as a brief conversation or even referral of the patient, but a second opinion is extremely useful in these situations. In most instances due to the interdisciplinary nature of modern dentistry, consultation with other clinicians who are treating the patient becomes a necessity to enhance the potential for successful treatment outcomes.

Occasionally, a patient will have persistent symptoms that mimic posttreatment disease, but these symptoms are the result of nonendodontic conditions such as occlusal trauma, concurrent periodontal disease, or nondental pain conditions. Appropriate diagnostic procedures should allow the clinician to sift through these options and treat accordingly.

The patient harboring true endodontic posttreatment disease has four basic options for treatment, which are as follows:

1. Do nothing
2. Extract the tooth
3. Nonsurgical retreatment
4. Surgical retreatment

The first option is to do nothing with the condition and allow it to take its course (Fig. 10.10). This approach is sometimes a useful, short-term option if the etiology of the condition remains unknown and the clinician feels that another diagnostic sampling would help with diagnosis. Even though most clinicians would find this approach to be a less-than-desirable long-term course of action, the decision belongs to the patient. The clinician is bound, however, to ensure that the patient has complete information about what will happen if nothing is done. The events in the progression of the disease and a reasonable timeline are necessary, and the conversation needs to be thoroughly documented in the patient record to avoid possible subsequent accusations of abandonment. The question of whether the clinician is required to follow up with the patient or dismiss the patient from the practice is one that each clinician must make based upon the clinician’s experience, judgment, and knowledge of the patient.

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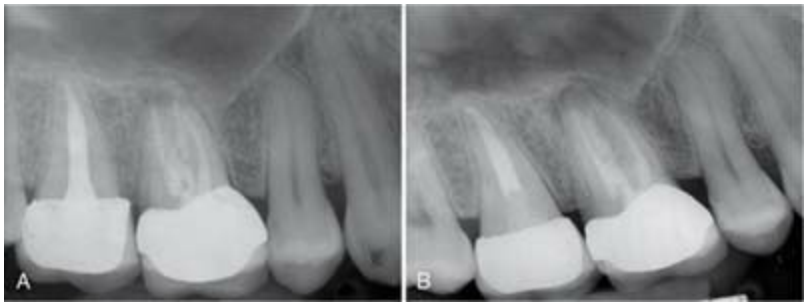

FIG. 10.10 **A,** Radiograph indicating presence of asymptomatic persistent apical periodontitis 7 years after initial treatment. The patient elected no treatment at that time. **B,** Six-year follow-up. Lesion has enlarged and the tooth has become symptomatic.

A) Radiograph of two teeth show a radiopaque crown and root canal and a radiopaque crown and radiolucent root canal.

B) Radiograph of two teeth show a black patch around the gum. The teeth in the right shows a radiopaque crown and radiolucent root canal and teeth in left shows radiopaque crown and radiolucent part of root canal.

Extraction of the tooth is usually considered a viable option. Recent advances in both prosthetic reconstruction techniques and in dental implantology have made extraction and replacement a more desirable option in certain cases where previously “heroic” (read: expensive with an unknown prognosis) methods were needed to “save” the tooth. This alternative, however, provides results that are inferior, more expensive, and much more time consuming than preserving the natural tooth. The average titanium root-form implant restoration can take up to 6 months to finish not counting preimplant site preparation, which can add months more. Despite published long-term success rates for dental implants,[5] postimplant disease does occur[5,95,96] (Fig. 10.11) and can leave the patient with very few options. The cost of implant treatment is high and may not be covered under dental benefit plans, so the net financial impact on the patient can be large. Implant esthetics can be inferior to that of natural teeth in the esthetic zone of the mouth, and there are patients that are just not candidates for implant procedures.[5] Fixed partial dentures are another replacement alternative with a very long history of successful use, but there are negative outcomes possible also. Most concerning to the endodontist is the likelihood that retainer fabrication procedures will result in endodontic disease of the abutment teeth[171] that may potentially occur at a rate of up to 10% (Fig. 10.12).[166,284]

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Removable partial dentures are a less desirable option for the patient because they are generally less comfortable, usually require a long period of patient adaptation, and frequently result in damage to adjacent oral tissues (tooth, gingiva, and mucosa) if not meticulously cleaned. Due to these factors, patient compliance with removable dentures is relatively low, and their use is declining. Occasionally, a patient will choose to have a tooth extracted and not pursue replacement. This decision is usually disastrous for the patient, but there are a few situations where this choice is a reasonable alternative. Diseased maxillary second molars with no opposing tooth, or with an opposing tooth in Class I or Class III occlusion that articulates with another tooth, may be extracted without concern for future inappropriate movement of the remaining teeth, which can be so occlusally and periodontally damaging. In most instances, however, removal of a tooth will result in the need for replacement, and unless the tooth is hopelessly nonrestorable, retaining the tooth with endodontic procedures is better for the patient.

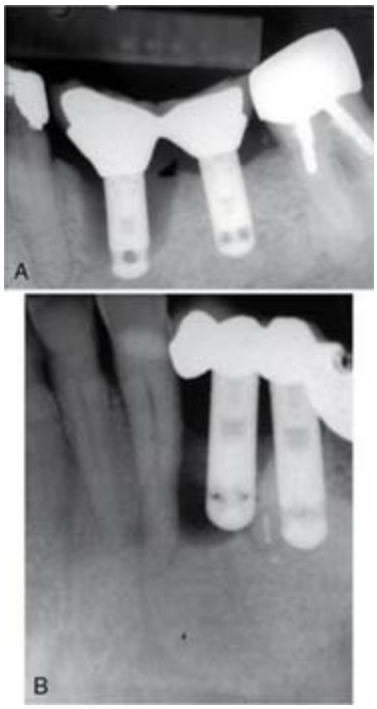

FIG. 10.11 **A,** Classic peri-implantitis. The implant needed removal. **B,** Another peri-implantitis. Note the endodontically treated root tip apical to the implant that may have contributed to the persistent disease. Perhaps apicoectomy should have been performed.

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A) Radiograph of teeth shows black outline around the pulp with a radiopaque white outline of two peri-implantitis. A tooth shows a radiopaque crown and part of root canal.

B) Radiograph of teeth shows black outline around the pulp of a tooth with a radiopaque white outline of two peri-implantitis.

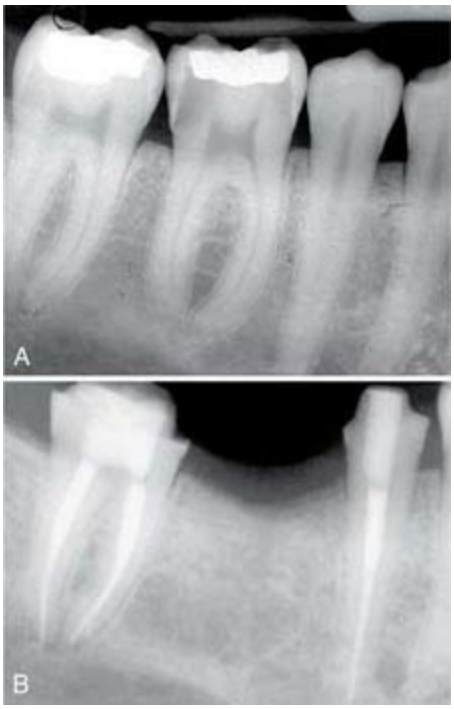

FIG. 10.12 **A,** Preoperative film showing deep caries approaching the pulp. The patient's holistic dentist advised extraction and replacement rather than endodontic therapy to retain the tooth. **B,** Fixed partial denture fabrication procedures resulted in irreversible pulpitis on both abutments requiring endodontic therapy.

A) Radiograph of teeth shows a white radiopaque patch in the crown of first molar and slightly larger radiopaque patch in the crown of second molar. Outlines of black are visible around the neck towards the root tips.

B) Radiograph shows gap between two teeth that show radiopaque crown and root canal.

Various situations may render a tooth nonrestorable (Fig. 10.13); however, the line of demarcation between restorable and nonrestorable is a movable one, depending on who is evaluating the tooth. There are several widely agreed upon situations that render a tooth nonrestorable. These include

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extensive caries or coronal fracture approaching or entering the furcation or the biologic width. This situation may render pre-prosthetic periodontal procedures ineffective (leaving a furcation involvement or poor crown to root ratio, for example) or worse, remove bone that would otherwise be useful for implant procedures. Terminal periodontal disease (extensive pocketing or mobility) or root fracture[45] generally results in loss of the tooth despite all efforts at treatment. If the patient has a life-threatening endodontic infection with extensive trismus, most oral surgeons are going to extract the tooth rather than allow less aggressive management. Some previously root-filled teeth may have endured procedural complications, such as a nonretrievable separated instrument or irreparable ledge formation. In combination with the proximity to vital anatomic structures, such as the inferior alveolar canal, endodontic retreatment, either surgical or nonsurgical, may not be feasible and extraction may be the only option. These situations are, fortunately, quite rare, and in most instances, teeth presenting with posttreatment disease can be retained with endodontic procedures.

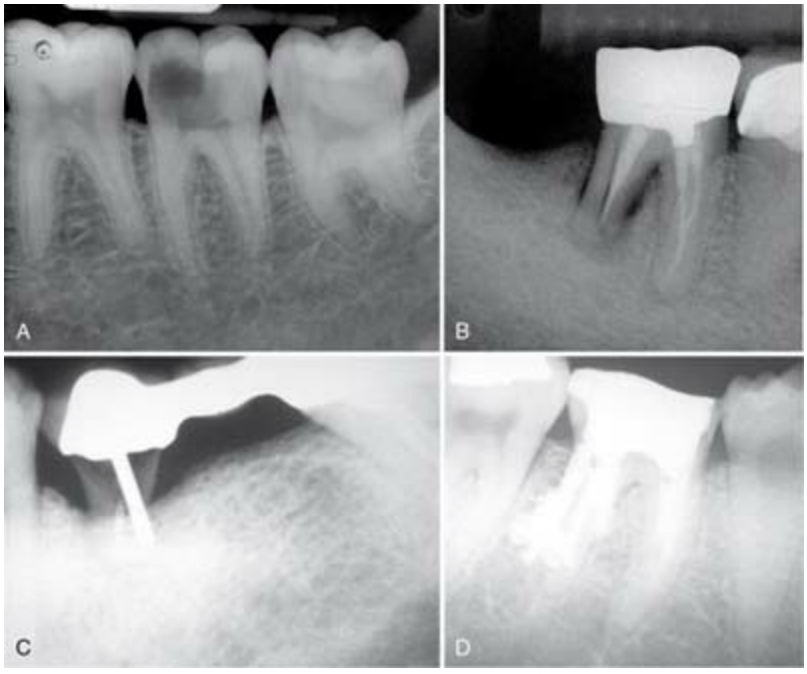

FIG. 10.13 **A,** Deep caries approaching the furcation and the biologic width. Necessary crown-lengthening surgery would open the furcation to bacterial invasion and persistent periodontal disease. **B,** Distal root vertical fracture resulting in a split root. **C,** Severe caries and post perforation. Inadequate root structure remaining to restore. **D,** Multiple

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distal root perforations so weaken the root as to make it nonrestorable. Note: Cases *A, B,* and *D* could have resective endodontic surgery such as hemisection, but long-term prognosis is poorer than for extraction and replacement.[35,142]

A) Radiograph of first molar shows radiopaque crown with radiolucency near the neck and circular cavity in the left side of crown. Second molar shows radiopaque crown with radiolucency near the neck and black smudge on the left side of crown.

B) Radiograph of a molar shows white radiopaque patch in the crown and a radiolucent root canal split in two lateral halves.

C) Radiograph of teeth with a radiopaque white outline of the perforation tool.

D) Radiograph of teeth shows a radiopaque crown and root canals with left root canal visibly segmented into chunks.

Once the decision has been made to retain the tooth, there are several choices for treatment. These can be grouped together into either nonsurgical or surgical endodontic treatments. The surgical options can be further broken down into periradicular curettage, apical root resection (with or without root filling), root amputation or hemisection, and intentional replantation (extraction/replantation).[100,190] Occasionally, a situation arises that will require both nonsurgical and surgical types of treatment to effect healing. The American Association of Endodontists has published guidelines that may help the clinician with clinical decision-making.[9] However, the choice of which option to undertake will be determined by the clinician's experience, knowledge, patient considerations, and the preoperative diagnosis. If the etiology of the posttreatment disease can be made known, the choices become more obvious. In a previous section, four basic etiologies were presented. If the suspected etiology is in the first group, which is persistent or reintroduced microorganisms, then several choices are available. However, if the cause of the posttreatment disease is either persistent extraradicular infection, foreign body reaction, or the presence of a true cyst, then nonsurgical root canal therapy has little likelihood of allowing healing to occur, and surgical methods should be employed.[259] The problem for the clinician is that, in most instances, it cannot be determined which of these etiologies exist, and so the treatment becomes more empirical.

The choice of nonsurgical retreatment versus apical surgery becomes the

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focus of the decision in most instances. Outcomes assessment studies provide some help in making this decision. This thought process has recently undergone significant change, however, due to more recent outcome studies. Past literature reported on outcomes related to older methods of performing periradicular surgery. They reported healing rates of nonsurgical retreatment that range between 74% and 98%,[75] but with older methods of apical surgery alone, only 59% heal completely.[72] When these methods of apical surgery were preceded by orthograde retreatment, however, the incidence of complete healing rose to 80%.[72] Therefore, in general, nonsurgical retreatment was the preferred choice, since it seemed to provide the most benefit with the lowest risk. It had the greatest likelihood of eliminating the most common cause of posttreatment disease, which is intraradicular infection. Nonsurgical retreatment is usually less invasive than surgery and has a less traumatic postoperative course. There is less likelihood of incurring damage to adjacent vital structures such as nerves, adjacent teeth, and sinus cavities. However, nonsurgical retreatment may be more costly than surgical treatment, especially if large restorations must be sacrificed during disassembly procedures prior to the retreatment. In addition, the amount of time needed for retreatment is usually longer than surgical intervention. There are times when the clinician may not be able to achieve the complete elimination of microorganisms from the canal space, and complete obturation may not be possible. Apical surgery was considered a potentially less successful option and was therefore chosen when nonsurgical retreatment was not possible or when the risk-to-benefit ratio of nonsurgical retreatment was outweighed by that of surgery.[73,159] Recently, several meta-analyses of longer-term surgical outcomes of newer microsurgical techniques have shown greatly improved rates of healing compared to older methods.[133,236,249,281] These studies indicated that endodontic microsurgery had success rates between 89% and 94%, much higher than the traditional surgical techniques that the previous treatment planning thought process depended on. This shift in the decision paradigm has now moved endodontic surgery to the forefront, and the decision of when to do surgery versus nonsurgical retreatment has become more well defined. Apical surgery is no longer the second choice in how to manage posttreatment disease, and this has caused greatly renewed interest among clinicians in acquiring the armamentarium, skills, and experience to

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perform surgery at a higher level.

There are many other factors to consider when deciding whether to retreat surgically or nonsurgically. The patient must be fully aware of the proposed treatment and the alternatives, and he or she must be motivated to follow through with all treatment including the final restoration. For nonsurgical management, the patient must have adequate time to undergo the required procedures. If not, then apical surgery alone may be indicated. The clinician must be armed with the best equipment and knowledge available, and critical self-evaluation should allow the experienced clinician to know what is and is not treatable. The tooth must be restorable and retreatable. Attempting nonsurgical retreatment on teeth where there is little likelihood of improving the previous treatment provides little benefit to the patient. Thus in disease situations where there is an apparently adequate root filling and no evidence of coronal leakage, surgery may be indicated.[203] If the previous treatment falls below any acceptable standard and there is no evidence of apical periodontitis, then there is no indication for any treatment unless a new coronal restoration is planned. In that case, conservative retreatment is indicated and the reported success rates are very high.[72,75] If there has been a previous procedural complication, such as a ledge that cannot be bypassed or a separated instrument that cannot be removed, then surgery may become a better option. Most times, however, it is still prudent to attempt the retreatment since ledges or separated instruments that appear impenetrable on diagnostic films can frequently be bypassed. Even if they cannot, nonsurgical retreatment can enhance the success of subsequent apical surgery as noted earlier. The clinician must be careful not to worsen the situation by overly vigorous attempts to treat the previous complication since root perforation, worsening of a ledge, or another separated instrument may be the result. Previous failed apical surgery should be retreated nonsurgically and then followed up since many surgical failures are due to poorly cleaned and filled canal systems (Fig. 10.14).[214] In many instances, performing the surgery a second time can be avoided altogether. If there is evidence of root fracture (narrow-based probing defect) and/or a J-shaped radiolucency encompassing the root apex and progressing in a coronal direction (Fig. 10.15),[266] then nonsurgical retreatment would be unlikely to improve that situation. Apical exploratory surgery may be necessary that could result in root resection or

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even extraction of the tooth.

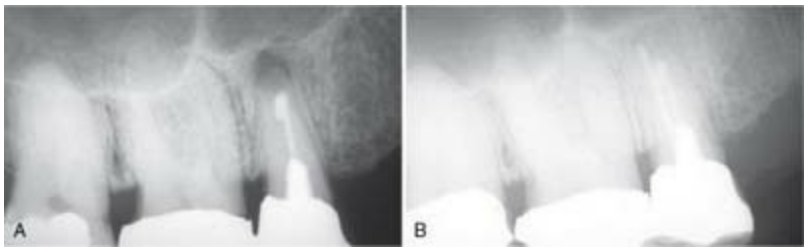

FIG. 10.14 **A,** Posttreatment disease following apical surgery. Off-center positioning of the root filling indicates the presence of a second, untreated canal. **B,** One-year following nonsurgical retreatment showing complete healing.

A) Radiograph of teeth shows radiopaque crown. Outline of the tip of plugger to fill the root is visible.

B) Radiograph of teeth shows radiopaque crown and a radiolucent root canal after healing. Outline of the tip of plugger to fill the root is visible.

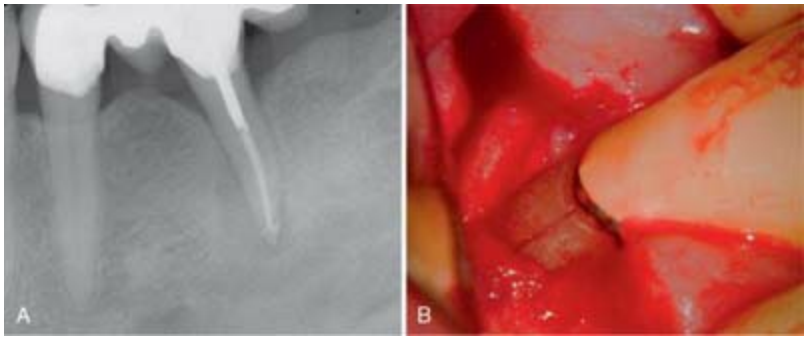

FIG. 10.15 **A,** J-shaped radiolucency possibly indicating root fracture. **B,** Exploratory surgery confirms presence of vertical root fracture.

A) Radiograph of teeth shows radiopaque crown. The root canal is segmented horizontally with upper segment showing radiopacity and lower segment showing radiolucency.

B) Close-up view of the periodontal ligament shows a thin slit between the gum and tooth with swelling and dark patch under it.

Each case should be approached as a unique set of considerations that need to be reviewed and interpreted prior to selecting a treatment method. Once the selected option is undertaken, however, the prudent clinician is always

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watchful, since additional pieces of information can be discovered during treatment that may modify previous decisions.

Nonsurgical endodontic retreatment

The primary difference between nonsurgical management of primary endodontic disease and posttreatment disease is the need to regain access to the apical area of the root canal space in the previously treated tooth. After that, all the principles of endodontic therapy apply to the completion of the retreatment case. Coronal access needs to be completed, all previous root filling materials need to be removed, canal obstructions must be managed, and impediments to achieving full working length must be overcome. Only then can cleaning and shaping procedures be instituted that will allow for effective obturation and case completion. The remainder of this chapter will be devoted to these topics in the order that they generally present themselves to the clinician treating the previously root-filled tooth.

Coronal access cavity preparation

Retreatment access has been called coronal disassembly[214,215] because of the frequent need to take apart or remove the previous coronal and radicular restoration. Following initial endodontic therapy, most teeth require and receive a full coverage restoration, and many times that restoration is supported by a post and core. Coronal-radicular access for retreatment is much more complicated in these cases when compared to endodontically treated teeth that have been minimally restored. The goal of the access preparation is to establish straight-line access to the root canal system while conserving as much tooth structure as possible. The ideal access preparation allows for instruments to enter the canals without being deflected by the access cavity walls. This is reasonably easy to achieve when the tooth is completely intact, and a pulp chamber is present since surface and internal anatomic landmarks can guide the search for the canals. Unfortunately, when endodontic retreatment is necessary, the tooth structure has almost always been altered and is commonly quite misrepresentative of the original anatomy of the tooth.

When presented with a tooth in need of retreatment that has a full coverage

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restoration, the decision for the clinician becomes whether to attempt to preserve the restoration or to plan its replacement. This decision is made simpler if there is a defect or caries associated with the restoration or if the treatment plan calls for a new crown. The old one is simply removed and replaced later in the treatment sequence (Fig. 10.16). When the crown is considered to be satisfactory, the decision becomes more complex. If the restoration is maintained, the cost for replacement can be avoided, isolation is easier, the occlusion is preserved, and the esthetics will be minimally changed. Even if the crown requires replacement, the clinician may elect to retain it during the endodontic retreatment to allow for better isolation with the rubber dam. Unfortunately, retreatment may be more difficult with the crown in place as this could lead to an increased chance for an iatrogenic mishap due to restricted visibility. In addition, removal of canal obstructions, such as posts, will be more difficult, and there is an increased chance the clinician may miss something important such as hidden recurrent caries, a fracture, or an additional canal. To preserve the restoration, two approaches can be taken: access through the crown or crown removal and replacement when retreatment is completed. The simplest choice is to prepare an access cavity through the existing crown, although there is a significant risk of damaging the restoration resulting in the need to replace it.[179] This risk must be communicated to the patient prior to instituting therapy. If the clinician decides to access through the existing restoration, there are several choices of access burs to use, depending on what material the preparation will be cut through. If the access will be primarily cut through metal (amalgam alloy or cast metal) or composite resin, carbide fissure burs such as the #1556 are usually chosen. With many restorations, it is advisable to consider using a combination of burs to achieve access. For example, when a porcelain fused to metal (PFM) crown is encountered, a round diamond is used to cut through the porcelain layer. Once the metal substructure is encountered, an end-cutting bur, such as the Transmetal bur (Dentsply Maillefer, York, PA) or the Great White bur (SS White, Staten Island, NY), can be used to cut through to and remove the core material efficiently. An important consideration for the clinician is the potential for porcelain fracture, which may occur during the preparation or possibly at a later date after completion of the treatment. This damage is especially common with porcelain jacket crowns. Restorations

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fabricated completely of porcelain are becoming more and more popular, thus creating added concern due to the increased likelihood of crack formation during access. Porcelain is a glass, and drilling through this material will create many micro-fractures, which in turn may weaken the structure of the restoration, making it more prone to future failure.[105] Copious coolant water spray and the use of diamond burs are recommended during access through porcelain to minimize occurrence of this event.[263] In a novel approach, Sabourin et al.[216] recently showed that, compared to the use of drills, air abrasion produced almost no defects in the porcelain structure of all ceramic crowns when used for endodontic access. It was, however, significantly more time consuming to access through the crown this way. Crowns made of zirconia are also becoming more popular due to their greater strength and acceptable esthetics. This material is very hard and resistant to cutting. Conventional diamond burs may not be able to cut this material well, and when attempting to do so, the production of small flashes of light, or sparks, may be seen at the interface of the spinning diamond and the zirconia. This is sometimes how it is discovered that it is indeed zirconia being cut, since many times these crowns resemble porcelain jacket crowns. Some types of tooth-colored crowns have a zirconia substructure that is overlaid with a ceramic veneer. In preparing the access, the external ceramic layer is easily cut, but upon reaching the zirconia substructure, progress slows, and light flashes can again be detected. In such cases, the veneered ceramic layer may have a higher incidence of fracture damage compared to a monolithic zirconia crown.[98] Newer diamond burs designed to cut through zirconia are available, which are much more efficient at removing the material and they do not produce the aforementioned light flashes. Copious irrigation should be used to minimize heat generation while accessing through zirconia crowns.[167]

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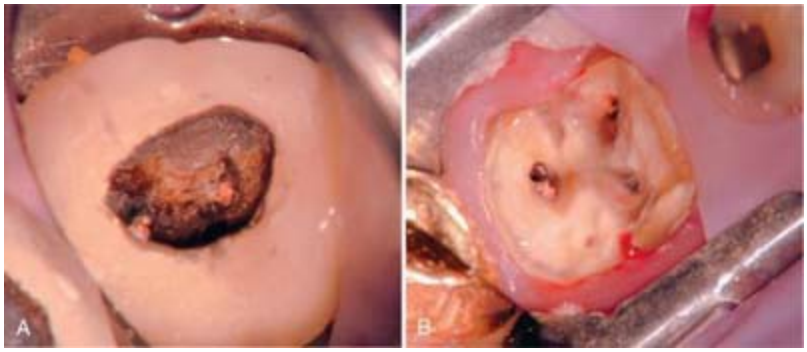

FIG. 10.16 **A,** Limited visibility and access with crown present. **B,** Enhanced visibility and access with crown off. Note: isolation achieved by using a Silker-Glickman clamp and sealing putty.

A) Close-up view of a molar with open crown exposing the decayed dentin.

B) Close-up view of a molar with three small ruptures in the crown exposing the decayed dentin.

If the decision is made to remove the crown for reuse, the visibility is increased, allowing for much easier removal of canal obstructions and a decrease in the potential for operator error; however, rubber dam clamp placement and tooth isolation may become a bigger problem. Also, despite all the varying techniques and armamentaria available for the removal of an existing restoration, the procedure remains unpredictable and many times can also result in damage to the restoration or the inability to remove it at all.

The clinician must decide how to remove the crown. If the crown is of no value, even as a temporary, then the clinician can take the easiest road and simply cut it off. However, if the crown is to be preserved, then a more conservative approach must be used. Two considerations, which may influence the decision about removal of a crown or bridge, are what material the restoration is made of and what is it cemented with. Conservative removal efforts are difficult with traditional, all-metal restorations cemented with nonbonded cements. This situation is even more of a concern lately due to the increasing popularity of tooth-colored restorations, mainly different types of porcelain, PFM, and zirconia restorations, which are being bonded to the tooth. These restorations are less likely to withstand the stresses of removal than those comprised completely of metal, and restorations that are bonded are much more difficult to remove due to the adhesive strengths of bonding agents. Each new generation of bonding agent is stronger than the previous,

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making removal increasingly more difficult as cosmetic dentistry advances.

There have been many devices developed specifically for the conservative removal of crowns. Some of the more commonly used devices are forceps, which have been designed specifically for crown removal such as the K.Y. Pliers (GC America, Alsip, IL) (Fig. 10.17), which uses small replaceable rubber tips and emery powder to enable a firm grasp of the crown without damaging it. Other instruments of this type include the Wynman Crown Gripper (Miltex, York, PA), the Trial Crown Remover (Hu-Friedy, Chicago, IL), and the Trident Crown Placer/Remover (CK Dental, Orange, CA). Unfortunately, a crown that has been cemented with long-term cement or has been bonded to the tooth will usually not be removed with one of these instruments. There are also forceps designed specifically to engage the margins of the crown while using an adjacent tooth as a fulcrum. Squeezing the handles together will cause the crown to be elevated off the tooth. The Roydent Bridge Remover (Roydent, Rochester Hills, MI) works in this fashion and can be effective in crown removal, but care must be taken to avoid damage to fine, fragile margins, especially on porcelain crowns. Another type of instrument can be engaged under the margin, and a subsequent impact delivered at this site will dislodge the restoration. The Easy Pneumatic Crown and Bridge Remover (Dent Corp, White Plains, NY) and the Coronaflex (KaVo, Lake Zurich, IL) create this impact from compressed air; whereas, the Morrell Remover (Henry Schein, Port Washington, NY) applies the force manually using a sliding weighted handle. The ATD Automatic Crown & Bridge Remover (J. Morita, Irvine, CA) uses vibrations to break the crown to preparation bond, and the Crown-A-Matic (Peerless International, South Easton, MA) delivers a shock impulse to loosen the crown. As mentioned before, crown margin damage may result as can inadvertent extraction of the tooth if the periodontium is compromised (see Fig. 10.17, *E*).[207] A different approach to conservative crown removal involves drilling a small hole through the crown in order to allow a device to thread a screw through the hole. This approach creates a lifting force that separates the crown and the tooth. The instruments that work in this manner are the Metalift (Classic Practice Resources, Baton Rouge, LA), the Kline Crown Remover (Brassler, Savannah, GA), and the Higa Bridge Remover (Higa Manufacturing, West Vancouver, BC, Canada). While very effective

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on metal crowns, these instruments may cause damage to porcelain occlusal surfaces on PFM restorations, and their use in both anterior teeth and in all-porcelain restorations is generally precluded.

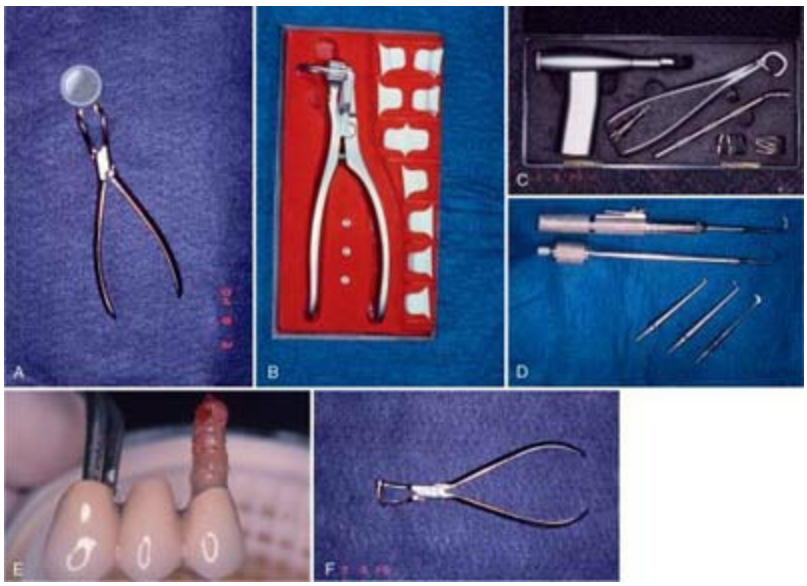

FIG. 10.17 **A,** KY Pliers (GC America, Alsip, IL) and supplied emery powder. **B,** Roydent Bridge Remover (Roydent, Rochester Hills, MI). **C,** CoronaFlex Kit (KaVo, Lake Zurich, IL). **D,** *Top,* Crown-A-Matic (Peerless International, South Easton, MA). *Bottom,* Morrell Crown Remover (Henry Schein, Port Washington, NY) with interchangeable tips. **E,** Tooth inadvertently extracted using a crown/bridge remover. Endodontic therapy was performed in hand and the tooth was replanted; a procedure known as unintentional replantation. **F,** Kline Crown Remover (Brasseler, Savannah, GA).

Set of five images shows tools for non surgical endodontic retreatment.

Another interesting technique designed to remove a crown without causing damage is performed using the Richwil Crown & Bridge Remover (Almore, Portland, OR). This material is a water-soluble resin, which is softened using warm water (Fig. 10.18). The small block of material is placed on the crown to be removed, and the patient bites into this material until the resin cools and hardens, at which point the patient opens his or her mouth, generating enough force to pull the crown off. Care must be taken by the clinician to avoid using this technique when the opposing tooth is extensively restored, since the opposing restoration may inadvertently be removed during the procedure.

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None of these techniques works in every case, and they may produce damage to the restoration being removed or possibly others. These are, however, methods that are available and may work while permitting reuse of the restoration.

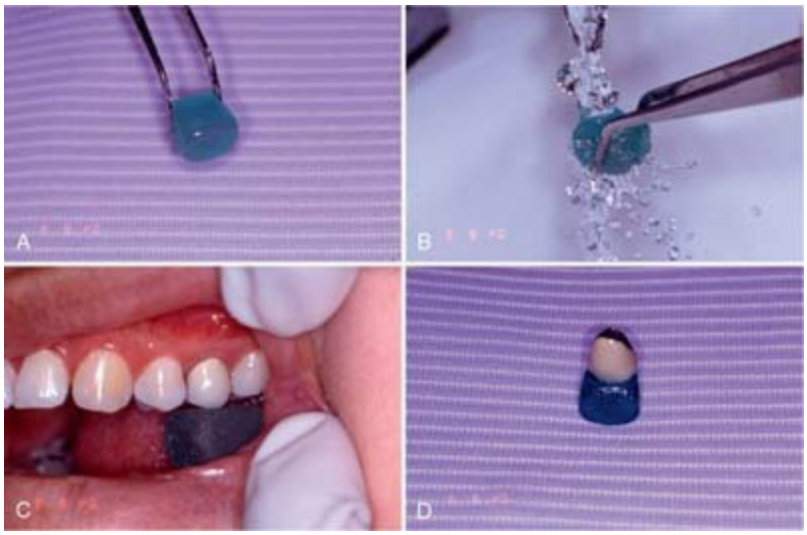

FIG. 10.18 **A,** Richwil Crown and Bridge Remover (Almore, Portland, OR). **B,** Using hot water to soften the material. **C,** The remover is placed on the restoration to be removed and the patient bites into the material. **D,** Image showing the removed crown adhering to the material.

A) Close-up of a teeth shaped water-soluble resin for removing crown and bridge held in a twizzers.

B) Close-up of the remover held in twizzers washed over a sink.

C) Close-up of a mouth with two lips held by two gloved fingers exposing the remover placed under the molars.

D) Close-up of the crown removed by the remover.

Post removal

Once the access is prepared, it is very common to encounter a post because posts are frequently used in the restoration of endodontically treated teeth. There are many different types of posts the clinician may encounter during retreatment (Fig. 10.19). These can be classified into two categories:

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prefabricated posts and custom cast posts. Historically, cast posts were more commonly used than prefabricated posts; however, over the past three decades, cast posts have become much less popular.[229] The main reason for this decrease is the convenience of placing the prefabricated post immediately after post preparation as opposed to waiting for a laboratory to fabricate the casting. There is also less likelihood of the interappointment contamination that frequently occurs with temporary post/core/crowns that are needed for cast/custom post and core fabrication. Prefabricated posts come in a variety of shapes, designs, and materials. The shapes can be subclassified into two groups: parallel sided or tapered. The design of posts also can be subclassified into active (threaded), passive, vented, fluted, and acid etched groups. There are also many materials that have been used to fabricate posts, such as stainless steel, gold, titanium, ceramic, zirconia, and fiber-reinforced composite posts. Cast posts, which are fabricated in a laboratory, will always be made up of precious or nonprecious metal alloys. These posts will also come in a variety of shapes and configurations since they are custom manufactured for each root in which they are placed. Most of these will have some degree of taper, and many will be cast in one piece with the core included.

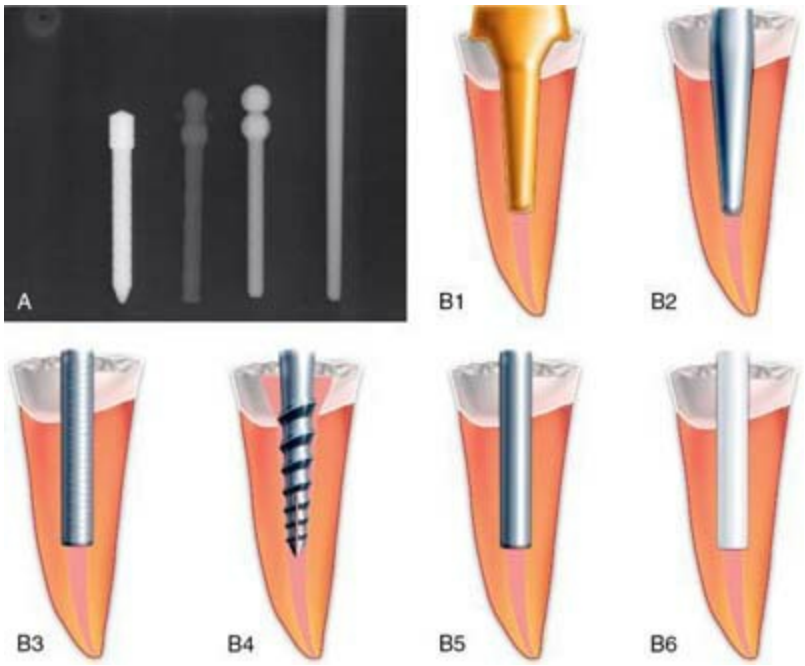

FIG. 10.19 **A,** Relative radiopacities of post materials: left to right—

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stainless steel, fiber post, titanium post, gutta-percha. **B,** Diagrammatic representation of post types: *1,* custom cast, *2,* tapered, *3,* parallel, *4,* active, *5,* passive/metal, *6,* passive/nonmetal.

A) Image shows four types of posts. A vertical rod with pointed lower tip and a cylindrical head, a vertical rod with flat lower tip and circularly curved double head, a vertical rod with curved lower tip and double circular head and a long vertical rod with narrow curved lower tip and a plain head.

B1) Diagram of a tooth with crown and root canal shows a post with broader top and narrow tip inserted into the canal.

B2) Diagram of a tooth with crown and root canal shows a post with a narrower tip inserted into the canal.

B3) Diagram of a tooth with crown and root canal shows a laterally textured post with wide tip inserted into the canal.

B4) Diagram of a tooth with crown and root canal shows a spirally textured post with pointed tip inserted into the canal.

B5) Diagram of a tooth with crown and root canal shows a plain metal post with wide tip inserted into the canal.

B6) Diagram of a tooth with crown and root canal shows a plain non-metal post with wide tip inserted into the canal.

Source: (Courtesy Dentsply Endodontics.)

In addition to the shape, design, and material of posts, there are two more very important factors that will have some influence on the clinician's ability to remove them. These factors are the adhesive material used to cement the post and the location in the arch of the tooth that requires post removal.

The same concerns regarding cements that were discussed in the section on crown removal apply to post removal. The main consideration is whether the post was cemented with traditional cement or bonded with a composite resin and dentin-bonding agent. Several post systems on the market today, such as the ProPost (Dentsply, York, PA), use acid-etched metal posts that are bonded into the canal with cements, such as Panavia (Kuraray America, New York, NY) or C&B Metabond (Parkell, Farmingdale, NY). Removal of these posts is extremely difficult and occasionally impossible, regardless of which technique is used.[94] A recent study[83] has shown that heat generation with ultrasonic vibration may help to decrease the retention of resin-cemented posts, but concern for heat-generated periodontal ligament damage may

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preclude this technique.[229]

Regarding location, the more posterior in the arch, the more difficult the post is to remove. This predicament is a result of accessibility. The more accessible the tooth is, the easier the post is to remove, since the clinician will have more techniques and instruments available to use.[3] Also, the more anterior the tooth is, the less the opposing occlusion will interfere with post removal.

Post removal techniques

After initial access and the post to be removed has been located, the clinician is faced with the decision of how to remove it. There have been many techniques developed for the sole purpose of post removal. Regardless of which technique is chosen, there is one simple yet extremely important rule to follow: it is not only what is removed, but what is left behind that is important. This rule applies to the removal of all intracanal obstructions. The reason for this rule is to make sure that the remaining tooth, after removal of the obstruction, can be restored predictably with a good long-term prognosis. For example, there is little use in successfully removing a post and leaving behind a root that is eggshell thin and prone to fracture (Fig. 10.20).

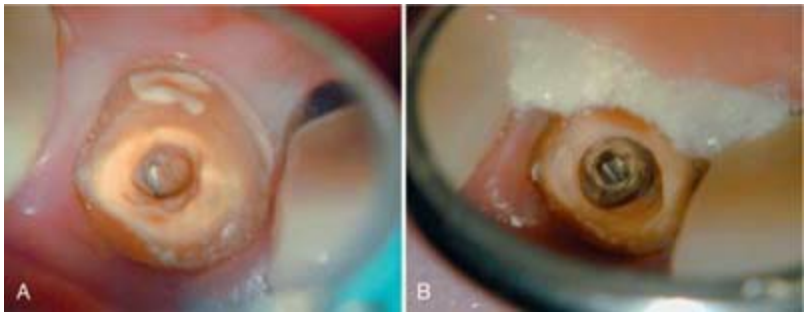

FIG. 10.20 **A,** Broken post (incisal view before excavation). **B,** Root has been so thinned and weakened by excavation procedures that restorability is questionable.

A) Close-up of the superior view of a molar with a broken and thinned post.

B) Close-up of the superior view of a thin rooted molar exposing the post.

The first step in post removal is to expose it properly by removal of all adjacent restorative materials. With preformed posts, the bulk of the core

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material around the post and within the chamber can be removed with a high-speed handpiece using cylindrical or tapered carbide or diamond burs. When most of the restorative material is removed, a less aggressive instrument, such as a tapered bur in a slow-speed handpiece or a tapered, mid-sized ultrasonic tip, should be used to remove the last of the embedding core material. This process is greatly facilitated by use of magnification and illumination. Once there is minimal restorative material remaining, a smaller-sized ultrasonic instrument should be used to minimize the risk of removing unnecessary tooth structure or thinning of the post. The more post that is left, the more options for removal, and the more tooth structure that is left, the more options for restoration. At this point, a high-speed bur is too risky to use. When the core is cast in one piece with the post, a high-speed instrument can perform this process to generate a shape that can facilitate removal.

Once the post is well isolated and freed from all restorative materials, the clinician can begin the retrieval process. There are many instruments and kits on the market that can be used to remove posts; however, prior to using one, the retention of the post should be reduced. The clinician can usually continue to use the same medium-sized ultrasonic tip which helped get to this point. Using this instrument at the interface between the post and the tooth (the cement line) and constantly moving it around the circumference of the post will disrupt the cement structure along the post/canal wall interface and decrease post retention facilitating removal[24,36,132]; although the effects of ultrasonic vibration may be minimal in reducing retention of well-fitted, long, large diameter titanium posts.[25] Titanium has a lower modulus of elasticity than stainless steel, so it may dampen the ultrasonic vibrations, which may decrease the effectiveness of the ultrasonic; however, a recent study[109] failed to duplicate this effect. Nonetheless, care should be taken not to push the ultrasonic tip against the post with too much force as this will dampen the ultrasonic wave and reduce the effectiveness of this technique. Taking away a small amount of the dentin around the coronal aspect of the post is not critical at this time as this will aid in the reduction of post retention without unduly weakening the root. If the root is thin, however, and the amount of space between the cement line and the root surface is restricted, the size of the tip that can be used may be limited. Unfortunately, the smaller tips are not only less effective for post removal they are also more prone to breakage. At this

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point, the ultrasonic handpiece should be used with copious air-water spray as a coolant. Due to the heat that can be generated from this procedure, the tip should be removed from the access every 10 to 15 seconds to allow the use of an air/water syringe to clean not only the area of debris but also to reduce the temperature produced that could potentially cause damage to the periradicular tissues.[229,307] If a rubber dam is in place, the area around the post may be flooded with a solvent, such as chloroform, prior to activating the ultrasonic instrument as this will help dissolve the cement around the post. Using a solvent in conjunction with removal of cemented obstructions may prove beneficial, since the ultrasonic energy produced will set up shock waves in the solvent and make it penetrate deeper into the canal space exerting a faster solvent action on the cement.[88]

Using an ultrasonic instrument in this fashion is not simply helpful in reducing post retention; this may also prove to be all that is needed to remove the post. Many times, after judicious use of the ultrasonic instrument, the post will loosen and spin out of the preparation completing post removal (Fig. 10.21). In addition, if post removal cannot be accomplished in this manner, the resulting post exposure will be very beneficial in contributing to the predictable use of other techniques as many of the instruments to be discussed involve using a trephine bur to shape the coronal end of the post. Ultrasonic exposure will facilitate this process. Another instrument to consider for exposing and loosening a post is the Roto-Pro bur (Ellman International, Hewlett, NY) (Fig. 10.22). There are three shapes available, all of which are six-sided, noncutting tapered burs that are used in a high-speed handpiece around the circumference of the post. The vibrations created when the noncutting flutes contact the post decrease the retention of the post, facilitating its removal.

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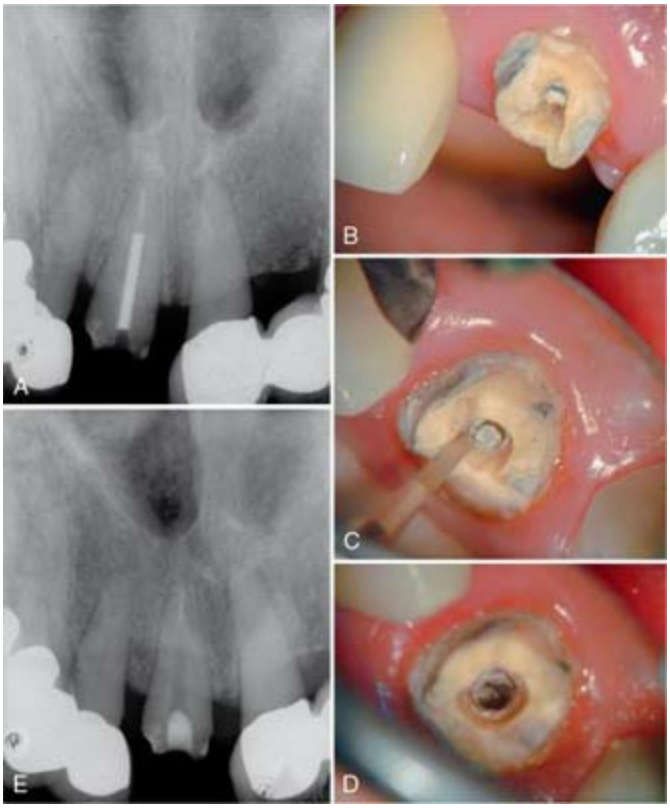

FIG. 10.21 **A,** Radiograph of fractured post. **B,** Fractured post, labial view. **C,** Ultrasonic troughing. **D,** Post removed by ultrasonic alone. **E,** Check film confirming complete post removal.

A) Radiograph of a tooth with no crown shows outline of a radiopaque post inserted in the canal.

B) Close-up of the thin rooted tooth exposing the broken post.

C) Close-up of the thin rooted tooth exposing the broken post being removed by a troughing tip.

D) Close-up of the thin rooted tooth after removal of the broken post exposing the void created in the molar.

E) Radiograph of a tooth with no crown shows radiopaque part of the root canal.

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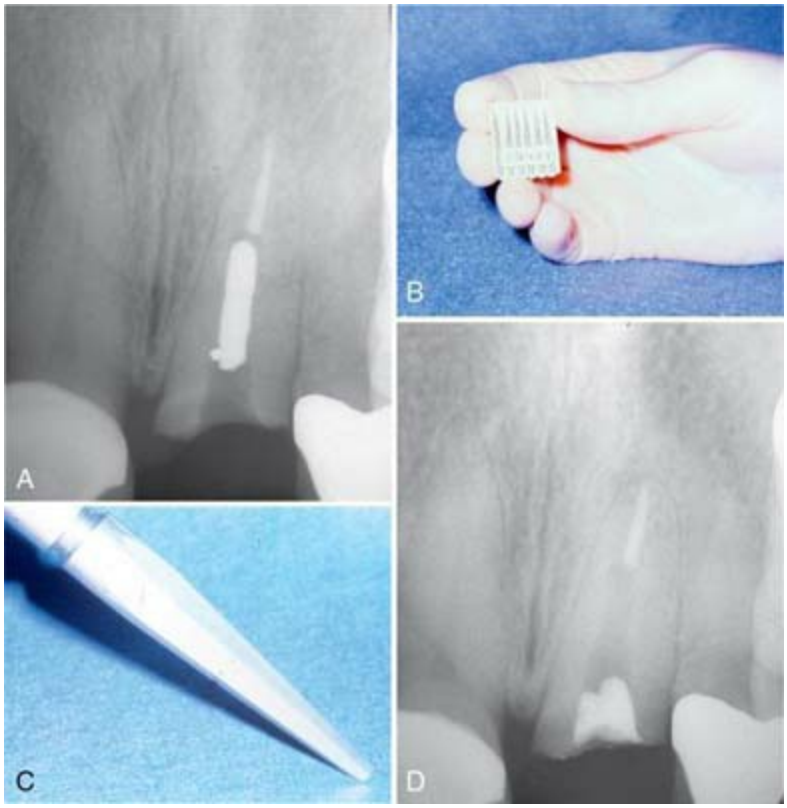

FIG. 10.22 **A,** Radiograph of fractured post. **B,** Roto-Pro Kit. **C,** Roto-Pro Bur. **D,** Post removed by vibration of the instrument alone.

A) Radiograph of a tooth with no crown shows outline of a radiopaque horizontally segmented post in the canal.

B) Close-up of a kit containing rotosonic scaler tips held between the fingers.

C) Close-up of a rotosonic scaler bur with a laterally pointed tip.

D) Close-up of the thin rooted tooth after removal of the broken post exposing the void created in the molar.

E) Radiograph of a tooth with no crown shows radiopaque part of the root canal.

If retention reduction does not remove the post, some form of vice is needed to pull the post from its preparation. Many post removal kits are available on the market today with varying degrees of effectiveness. One such device is the Gonon Post Removing System (Thomas Departement Dentaire, FFDM-Pneumat, Bourge, France) that is a very effective instrument for removing parallel or tapered, nonactive preformed posts.[160,218] This kit utilizes a hollow trephine bur that is aligned with the long axis of the post and

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placed over its newly exposed end. The trephine then cuts in an apical direction shaving off the post's outer layer not only to remove tooth structure adjacent to the post but also to reduce the circumference of the post to a specific size and shape. This procedure is necessary to allow a specific, matched-size extraction mandrel to create or tap a thread onto the exposed milled portion of the post. Once the extraction mandrel with its associated washer/bumpers (Fig. 10.23) is attached to the post, the extraction forceps or vice is applied to the tooth and post. Turning the screw on the handle of the vice applies a coronal force in a similar fashion as a corkscrew removes a cork from a bottle of wine. This method is effective because all the force is applied to the bond between the tooth and the post ideally in the long axis of the root. The main problem with this technique is the size of the vice that can make access in the molar region and between crowded lower incisors difficult. Also, if the extraction force applied is not directed in the long axis of the root, root fracture may occur.[41]

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FIG. 10.23 Gonon post removal technique. **A,** Fractured post in a lower incisor. **B,** Tooth isolated with a rubber dam. **C,** Gonon Kit. **D,** Ultrasonic exposure of the post. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Extraction device tapping a thread onto the post. Note the three bumpers needed to protect the tooth from the vice. **H,** Vice applied. Turning the screw on the vice opens the jaws creating the extraction force. **I,** Post removed.

A) Close-up of a thin rooted tooth exposing the segmented post.

B) Close-up of the tooth isolated by a rubber dam clamp exposing the post.

C) Close-up of the Gonon universal post extractor kit with extracting plier, trephines, corresponding tubular taps, torque bar, tube spacers and rubber bumpers of varying diameters.

D) Close-up of the exposed post being unfixed by a tartar scraper tool.

E) Close-up of the exposed post being shaped by a domer bur.

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F) Close-up of the exposed post being milled by a trephine bur.

G) Close-up of the exposed post being pulled out by an extraction device.

H) Close-up of the exposed post being screw-turned by a vice.

G) Close-up of the removed post in the extraction device.

The Thomas Screw Post Removal Kit (Thomas Departement Dentaire, FFDM-Pneumat, Bourge, France) (Fig. 10.24) is an instrument designed specifically for the removal of active or screw posts. The trephine burs are identical to those used with the Gonon Post Removal System; although the extraction mandrels are threaded in the opposite direction. The mandrels are reverse threaded to enable them to tap onto the screw post in a counterclockwise direction so that continued torquing force while creating the thread will unscrew the post.

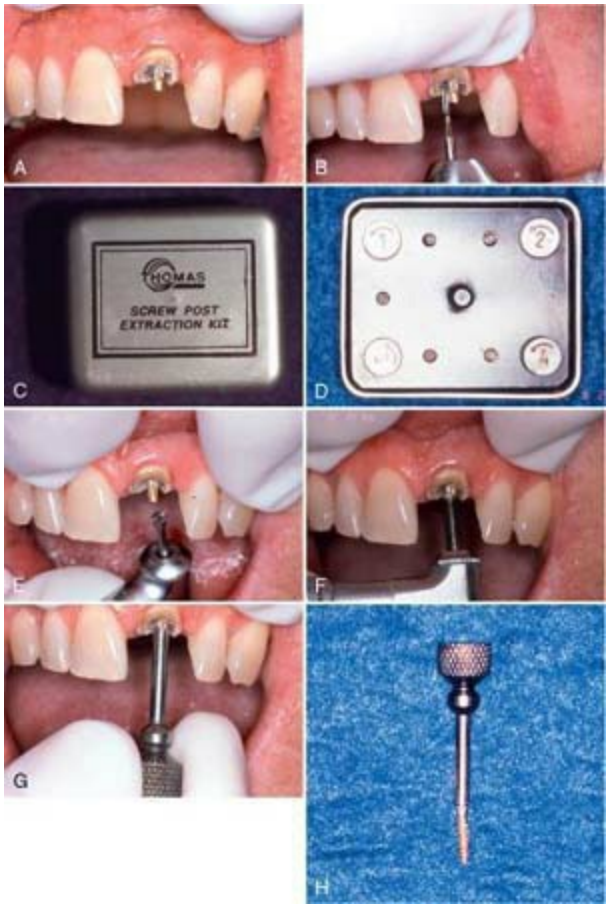

FIG. 10.24 Thomas screw post removal technique. **A,** Broken screw

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post. **B,** Head of post being contoured to a roughly cylindrical shape. **C** and **D,** Thomas Post Removal Kit. **E,** Domer bur creating a shape that the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Application of counterclockwise rotational force using the wrench. **H,** Post removed.

A) Close-up of an open mouth exposing the upper jaw with a broken screw post.

B) Close-up of an open mouth held by a gloved thumb shaping the post by a dental contouring tool.

C) Close-up of the Thomas screw post dental kit trephines and corresponding tubular taps.

D) Close-up of the components of Thomas screw post dental kit.

E) Close-up of an open mouth held by gloved thumbs shaping the post by a domer bur.

F) Close-up of an open mouth held by gloved thumbs milled by a trephine bur.

G) Close-up of the exposed post being screw-turned by a wrench.

H) Close-up of the removed post in the wrench.

The Ruddle Post Removal System (Kerr Corporation, Orange, CA)[214] (Fig. 10.25) and the Universal Post Remover (Thomas Departement Dentaire, FFDM-Pneumat, Bourge, France) were designed to combine the properties of both the Gonon and Thomas Kits. Both of these very similar kits are not only useful in the removal of parallel or tapered passive types of posts but also in removing screw posts. They can even be adapted to remove large separated instruments in the coronal straight portion of a large canal. These kits also use a trephine bur to machine the post to a specific size that will dictate which mandrel to use. These mandrels tap in the counterclockwise direction so that the same taps can be used for both passive and active posts. Once the mandrel is tapped onto the post, the extraction jaws, or vice, is applied and activated enabling removal of passive posts, or the tap is continuously rotated counterclockwise to unthread screw-type posts.

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FIG. 10.25 **A,** Perforated post requiring removal. **B** and **C,** Ruddle post removal kit. **D,** Post removed and perforation repaired.

A) Radiograph of a preoperative molar shows a radiopaque crown and radiopaque outline of the spirally textured post inserted in two root canals. The pulp shows radiolucency.

B) Close-up of the components of Ruddle screw post dental kit; an extracting plier, transmetal bur, five trephines, five corresponding tubular taps, torque bar, tube spacers and rubber bumpers of varying diameters.

C) Close-up of a Thomas screw post extracting plier, two trephine burs and a tubular tap.

D) Radiograph of a molar shows a radiopaque root canals with left root canal showing a lateral segment and right root canal shows radiopaque outline of a post inserted into it.

Another device that works in a similar fashion as the Gonon and the Ruddle Post Removal System is the JS Post Extractor (Roydent Dental Products). The biggest advantage of this kit is the size, as this is the smallest of the kits that work using a pulling action, which may help with cases where access is difficult. However, this kit does have one disadvantage; it does not have as large of a variety of trephine burs and extraction mandrels as some of the others. Therefore the size of the post may be a limiting factor.

Another post removal device is the Post Puller also known as the Eggler

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Post Remover (Automaton-Vertriebs-Gesellschaft, Germany) (Fig. 10.26).[255] This device works in a similar manner as some of the others; however, there are no trephine burs or extraction mandrels. The design of this instrument enables it to be used more efficiently with the crown removed. In addition, the design also allows this instrument to be used for cases in which the post and core are cast as one unit. This device consists of two sets of jaws that work independently of one another. With this device, both the post and the tooth are reduced to allow attachment of the post puller. Since there are no trephine burs, this reduction is done with a high-speed handpiece and bur. Next, the first set of jaws are attached to the post while the second set of jaws push away from the tooth in line with the long axis of the tooth removing the post from the canal.[255] Care must be taken to align the pulling forces of this instrument with the long axis of the root to prevent fracture,[41] and also, this technique is not recommended for the removal of screw posts. In a survey of the Australian and New Zealand Academy of Endodontists, this was the most commonly used technique for post removal.[40] However, in a survey of the American Association of Endodontists, this was one of the least used techniques.[256] Clearly, techniques that are common in one country are not always that common in another.

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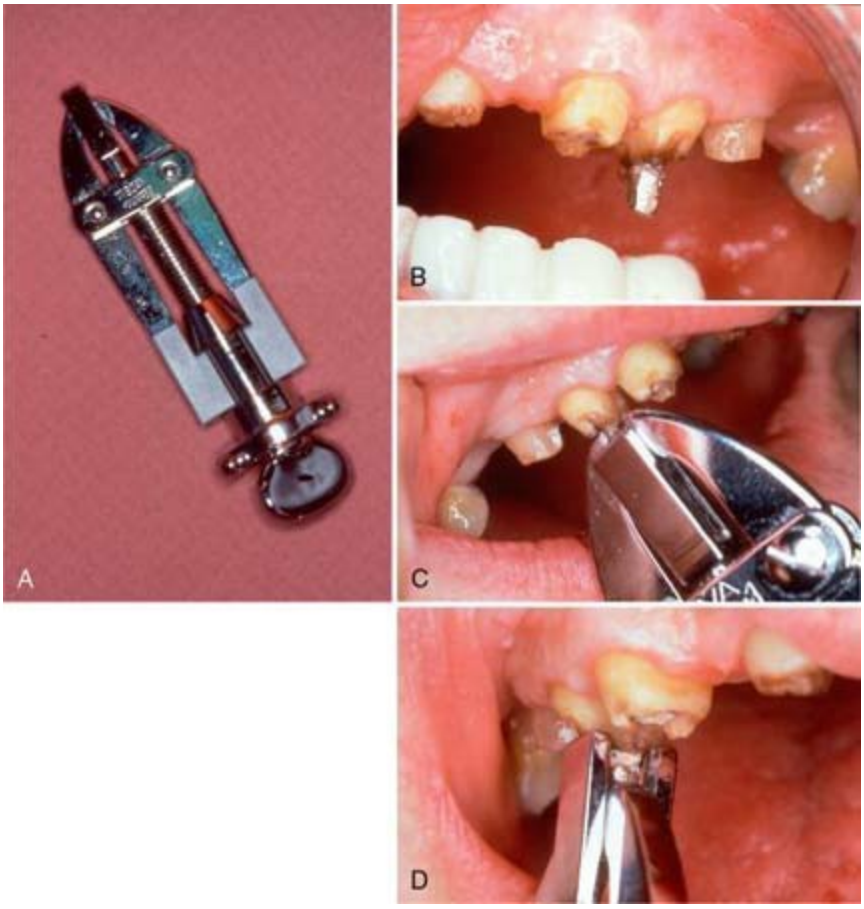

FIG. 10.26 **A,** Eggler Post Remover. **B,** Post has been contoured with a highspeed bur. **C,** Eggler Post Remover grasping the post. **D,** Elevating the post.

A) Close-up of an eggler post remover tool.

B) Close-up of an open mouth shows upper jaw with uneven teeth exposing the contoured post.

C) Close-up of an open mouth shows the remover tool extracting the post.

D) Close-up of an open mouth shows the remover tool upraising the post.

Source: (Reprinted with permission form Stamos DE, Gutmann JL: Revisiting the post puller. *J Endod* 17[9]:467, 1991.)

The recent increased popularity of cosmetic dentistry has created an impetus towards the use of tooth-colored posts that are fabricated from ceramic, zirconia, or various types of fiber-reinforced composite. Unfortunately, as with all posts, cosmetic posts also will need to be removed periodically. Neither the use of the Gonon Kit nor ultrasonic instruments

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allows for removal of cosmetic posts while the use of a high-speed bur to channel down through the post may result in a high rate of root perforation.[200,229] It is important for the clinician who is removing cosmetic or tooth-colored material or posts from the canal, to stain the coronal area of the post with a dye such as methylene blue (Fig. 10.27). By doing this, a determination can be made as to the nature of the substance to be removed and this then allows the clinician to remove as little remaining tooth structure as possible. The dye may penetrate the composite or a fiber post but more likely, it will simply outline them and the canal walls so that under the high magnification of the microscope, only the restorative material, cement, or post is removed conserving precious tooth structure. This contrasts with the task of removing a metal post where oftentimes more dentin removal is required.

FIG. 10.27 Methylene blue dye applied to cosmetic materials in the canal orifice. The dye may outline or penetrate the materials/fiber posts but will allow more conservative removal of the obstruction preserving dentin. If no dye penetration occurs at all, the post may be made of ceramic or zirconia requiring a different removal strategy. **A,** Composite outlined. **B,** Composite penetrated. **C,** Fiber post outlined. **D,** Fiber post penetrated.

Set of four images shows a horizontal section of tooth colored cosmetic material with a circular rupture in the center held in a clamp displaying an outline or penetration of methylene blue dye.

The use of the Largo Bur (Dentsply, York, PA)[85] and the Peeso drill[200] to remove these posts has been advocated and most of the post manufacturers' have removal burs in their kit.[53] These manufacturers removal kits have been

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shown to be more efficient at removing their own fiber posts than the use of diamond burrs and ultrasonics.[153] In addition, a new bur, the GyroTip (MTI Precision Products, Lakewood, NJ), has been designed for the specific purpose of removing fiber-reinforced composite posts (Fig. 10.28). These drills consist of a heat-generating tip designed to soften the matrix that binds the fibers within the fiber-reinforced post. The fibers within the post are parallel, which assists the axial travel of the drill through the center of the post. The fluted zone of the drill allows the fibers to be safely removed, creating access to the root canal filling. Above the fluted zone, a layer of plasma-bonded silica carbide reduces the heat generation that would otherwise occur if a smooth carbide surface were rotating in contact with enamel and/or dentin. This abrasive zone also provides for a straight-line access preparation and facilitates the placement of a new post. Ceramic and zirconia posts are usually impossible to retrieve. They are more fragile than metal posts, and though ceramic posts may be removed by grinding them away with a bur (a procedure with a high risk of root perforation), zirconia has a hardness approaching that of diamond and cannot be removed by this method.[229]

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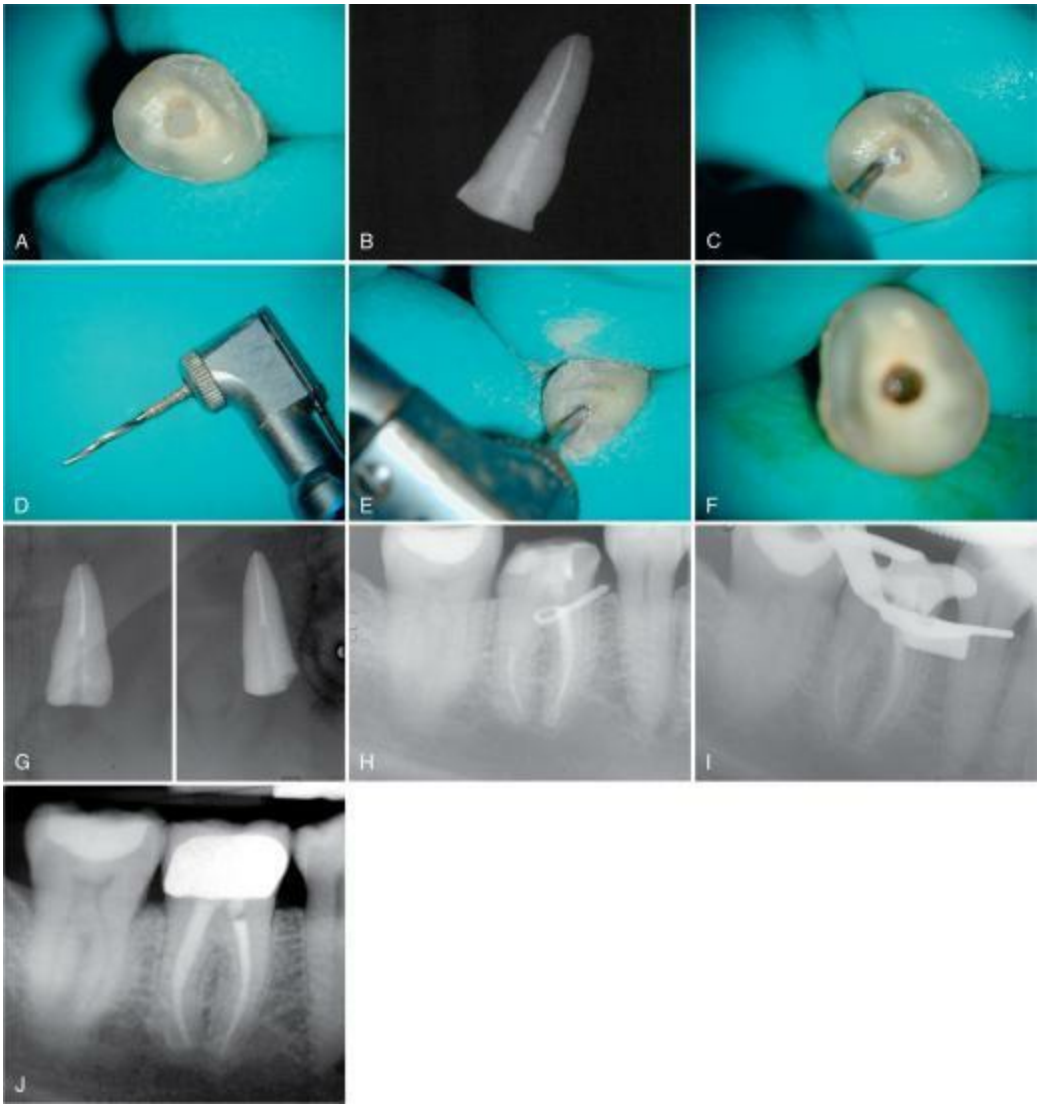

FIG. 10.28 GyroTip technique. **A,** Broken fiber post in an extracted tooth. **B,** Radiograph of test tooth with post in place. **C,** Creating a pilot hole. **D,** GyroTip instrument. **E,** GyroTip cutting through the fiber post. Note alignment with long axis of post. **F** and **G,** Post removed. **H,** Clinical case showing fiber post perforation into furcation area. **I,** Post removed with the GyroTip. **J,** One-year follow-up of MTA repair.

A) Close-up of horizontal section of tooth held between two gloved fingers exposing a broken fiber post.

B) Radiograph of a tooth with no crown shows radiopaque outline of a post inserted in the root canal and a radiolucent pulp.

C) Close-up of horizontal section of tooth held between two gloved fingers shows a gyrotip bur creating a pilot hole.

D) Close of a gyrotip tool with bur.

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E) Close-up of horizontal section of tooth held between two gloved fingers cutting through the center with gyrotip bur.

F) Close-up of horizontal section of tooth held between two gloved fingers exposing the void created after the post removal.

G) Two set of radiographs of a tooth with no crown shows radiolucency in the upper segment and radiopacity in the lower segment of the root canal.

H) Radiograph of a tooth with no crown shows radiopaque right root canal and radiolucency in the upper segment and radiopacity in the lower segment of the left root canal. Radiopaque outline of a perforating tool is visible over left root canal.

I) Radiograph of a tooth with no crown shows partially radiopaque right root canal and radiolucency in the left root canal. Radiopaque outline of a gyrotip bur is visible over left root canal.

J) Radiograph of a tooth shows a radiopaque crown with two radiolucent root canals. Radiopaque outline of a post is visible in right root canal.

Regardless of the post type or retrieval method used, once the post has been removed, the final step in exposing the underlying root filling material is to ensure that none of the post cement remains in the apical extent of the post space. This step can be easily accomplished by visualizing the cement using magnification and illumination and then using a straight ultrasonic tip to expose the underlying canal filling.

Potential complications of post removal

As with many dental procedures, post removal has risks. These risks include fracture of the tooth,[38] leaving the tooth nonrestorable due to excessive dentin removal, root perforation, post breakage, and inability to remove the post.[256] An additional concern is ultrasonically generated heat damage to the periodontium.[229]

Even though there may still be some who feel that posts strengthen teeth, it is widely accepted that they do not.[229] Actually, it has been shown that post preparation alone weakens teeth.[280] Therefore it seems obvious that any additional work which may require removal of further tooth structure will further weaken the tooth, increasing the likelihood of fracture. An in vitro study showed that cracks can form in radicular dentin during post removal using both the Gonon Kit and using ultrasonics, but there was no significant

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difference between these two groups and teeth with posts that were not removed.[8] The authors speculated that the potential for vertical root fracture might be increased; however, the clinical significance of this remains unknown. A more recent study, however, concluded that the incidence of root fracture during post removal was extremely low and that with good case selection, post removal is, in fact, a very predictable procedure.[3] If, however, post removal would also leave the remaining tooth structure in a state that may not be predictably restored with a good prognosis and if this situation can be predicted ahead of time, then surgery may be the preferred treatment option.

Perforation is an additional possible complication that can happen during post removal, especially if the post is removed by simply attempting to drill it out with high-speed burs.[200] If perforation occurs, the clinician should repair it immediately as the prognosis will worsen as the time between perforation and repair lengthens.[29,234] Once a perforation occurs, the clinician must reconsider the prognosis and determine whether the tooth should be salvaged. Terminating the procedure and pursuing a different treatment option could be considered at this point. Extraction and replacement with an implant and/or a fixed prosthesis was a treatment option prior to initiating the retreatment, and some may consider this treatment the best option once a perforation has occurred. However, with the recent development of mineral trioxide aggregate (Pro-Root MTA, Dentsply, York, PA) and other bioceramics, perforations can be repaired with a favorable prognosis.[208] The techniques and materials for perforation repair will be discussed in detail in a later section of this chapter.

Another complication is separation of the post, causing removal of the coronal segment while leaving a small portion of the post with even less accessibility. This separation will decrease the likelihood of removal and occurs more frequently when attempting to retrieve titanium posts.[229]

The use of ultrasonic energy for prolonged periods of time can generate excessive amounts of heat. The heat generated can cause damage to the surrounding periodontium.[88,229] This damage may be as serious as both tooth and permanent bone loss (Fig. 10.29). For this reason, stopping periodically to cool off the area with a water spray is necessary. This will be discussed in detail in a later section.

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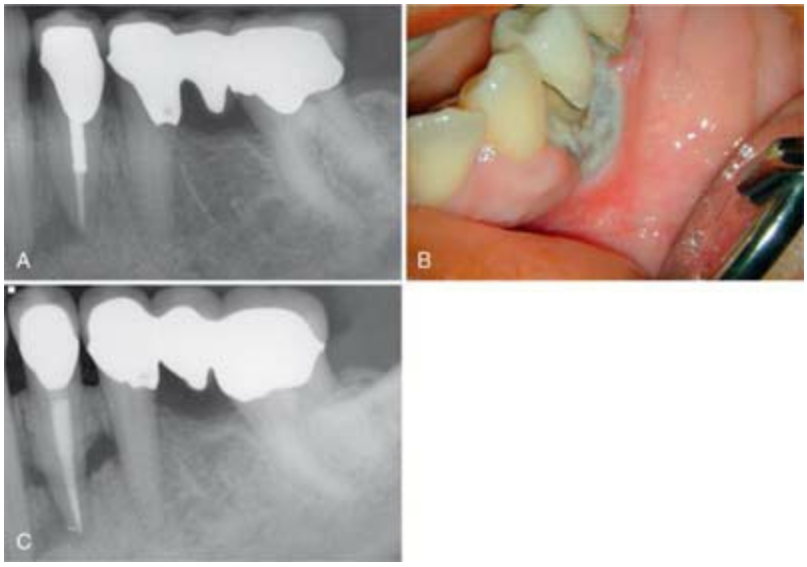

FIG. 10.29 Tissue damage from heat generated by ultrasonic application to a post during removal. The ultrasonic tip was applied to the post for no more than 5 minutes at high power with the assistant applying a constant water spray. **A,** Preoperative radiograph. **B** and **C,** These images were taken 1 month after the retreatment. Note sloughing bone visible on *C*. The tooth was lost 1 month later.

A) Radiograph of a tooth shows a radiopaque crown with a partially radiopaque root canal and radiolucency around the neck.

B) Close-up of an open mouth shows the damaged tooth segmented from the base with a white patch around the gum. Water is sprayed into the mouth using a water spray nozzle.

C) Radiograph of a tooth shows a radiopaque crown with a radiopaque outline of the separated post in the root canal.

Source: (Reprinted with permission form Schwartz RS, Robbins JW: Post placement and restoration of endodontically treated teeth: a literature review. *J Endod* 30[5]:289–301, 2004.)

If the clinician is unable to remove the post, then he or she will be faced with a decision of what to do. This decision is based on whether the post is being removed for restorative purposes or due to the persistence of disease. If the reason is for restorative purposes and the clinician can adequately restore the tooth with the existing post or post segment, then he or she should do so. If the tooth cannot be properly restored without removal of the post and placement of a new post, then extraction and replacement with an implant and/or fixed prosthesis will be needed. If the reason for post removal is due to

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the persistence of disease, the tooth should be treated surgically and restored as well as possible.

Regaining access to the apical area

Once the coronal-radicular access is made and all posts and obstructing restorations have been removed, then the clinician must regain access to the apical area by removing the previous root-filling materials (Fig. 10.30). This part of nonsurgical retreatment is complicated by the large variety of types of root fillings used. Today, most root fillings are performed using gutta-percha in various forms; however, many other materials have been and are still being used. Silver points were very popular until the 1970s and various types of pastes are, unfortunately, still in use. The authors have seen cases of definitive root filling with phenol-soaked paper points and sometimes no root filling at all. New materials, such as various bioceramic materials and sealers, are coming on the market all the time. Though all root-filling materials have their advocates and their critics, the only certainty is that all will have some incidence of persistent disease and may need retreatment.

FIG. 10.30 Accumulation of materials removed from retreated teeth in a 3-month period.

Close-up of the removed root filing materials, many of which include the laterally textured posts, spirally textured posts, plain metallic posts etcetera.

During the diagnostic phase, it is very important to ascertain the nature of

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the root filling to minimize surprises when attempting retreatment. Sometimes this is readily apparent, but, in other instances, this determination may require contacting the previous clinician to discover what type of root filling was used. Occasionally, this information cannot be determined until canal entry, so extreme caution should be used when performing access so as not to possibly remove parts of the root filling that may be useful in its removal, such as the core material in solid core obturators.

Gutta-percha removal

One of the great advantages of using gutta-percha for root filling is its relative ease of removal. When the canal contains gutta-percha and sealer or a chloropercha filling, it is relatively easy to remove this material using a combination of heat, solvents, and mechanical instrumentation.[78,214] It should be noted, however, that there is no technique, instrument, or solvent that can, by itself, remove all of the previous root filling materials from a canal space.[212] Clinicians must use multiple techniques tailored to the individual tooth and patient being treated to achieve maximal root filling removal. Upon access, it is usually relatively easy to find the treated canal orifices with the visible pink gutta-percha material inside. Initial probing with an endodontic explorer into the material can help rule out the possibility that there is a solid core carrier. If there is a plastic carrier, then heat should not be used to remove the coronal gutta-percha (more on this later). If there is no carrier, heat is applied using an endodontic heat carrier that has been heated to a cherry red glow in a torch. Unfortunately, the carrier begins to cool upon removal from the flame, and so endodontists are now using a heat source, such as the Touch ‘n Heat (Sybron Endodontics, Orange, CA) (Fig. 10.31, *A*), to provide constant, consistent heat application to soften the gutta-percha in the coronal portion of the canal.[156] Care must be exercised, however, not to overheat the root, which can cause damage to the periodontal ligament.[146,221,222] Thus, the heat should be applied in a short burst to allow the instrument to penetrate the gutta-percha mass, followed by cooling, which will cause the material to adhere to the heat carrier facilitating its removal (see Fig. 10.31, *B*). After removing as much gutta-percha as possible with the heated instrument, then remove any remaining coronal material with small Gates-Glidden drills, taking care not to overenlarge the cervical portion of the

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canal. However, since the previously treated tooth may have had an underprepared cervical third of the canals, these drills can also be used to flare the coronal aspect in an anticurvature direction to facilitate enhanced straight line access to the apical one third of the canal and to create a reservoir for potential solvent use.[165] Again, probe the canal, this time using a #10 or #15 K-file. It is sometimes possible to remove or bypass the existing cones of gutta-percha if the canal has been poorly obturated, thus eliminating the need for solvents.[254] If that is not possible, then a gutta-percha solvent must be used to remove the remaining material in the apical portion of the canal.

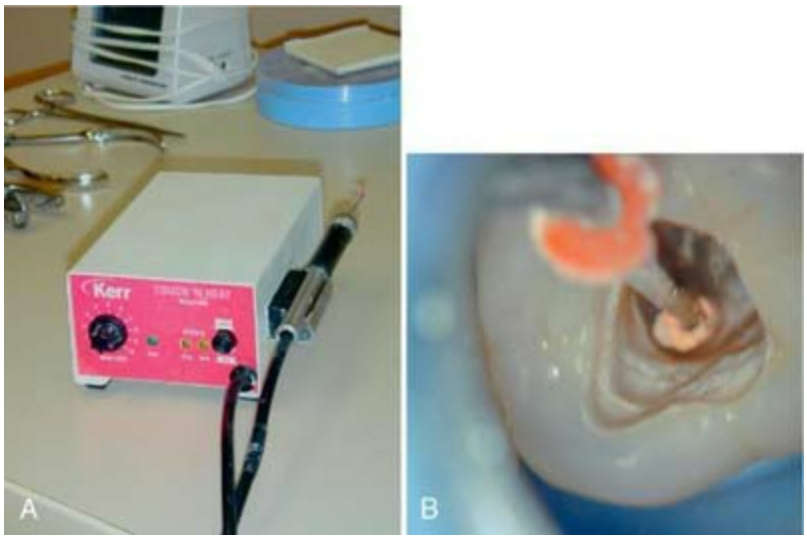

FIG. 10.31 **A,** Touch 'n Heat instrument. **B,** Gutta-percha adhering to the Touch 'n Heat tip as it cools.

A) Close-up of touch and heat device with a connected heat carrier pen with tip.

B) Close-up of gutta percha filling being inserted in an exposed crown of a tooth using a heating carrier tip.

Several solvents have been recommended to dissolve and remove gutta-percha for retreatment (Fig. 10.32) including chloroform,[170] methylchloroform,[290] eucalyptol,[306] halothane,[118,141] rectified turpentine,[134] and xylene.[104] All the solvents have some level of toxicity,[19,44] so their use should be avoided if possible; however, a solvent is usually needed to remove

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well condensed gutta-percha. The most popular solvent is chloroform since it dissolves the gutta-percha rapidly and has a long history of clinical use. In 1976, the US Food and Drug Administration banned the use of chloroform in drugs and cosmetics due to a report of suspected carcinogenicity.[282] There was no associated ban on its use in dentistry[170]; however, the report did result in the search for alternatives some of which are listed earlier. When used carefully, chloroform is regarded as a safe and effective endodontic solvent.[44,170] All of the others generally have been reported to be less effective or have some other drawback that limits their use. Xylene and eucalyptol dissolve gutta-percha slowly and only approach the effectiveness of chloroform when heated.[301] Rectified turpentine has a higher level of toxicity than chloroform[19] and produces a very pungent odor in the operatory. Halothane has been shown to be as effective a solvent as chloroform in several studies,[118,141] but a more recent study indicated that the time for removal of the root filling was longer than using chloroform.[294] The increased cost and volatility of halothane and the potential for idiosyncratic hepatic necrosis make it less desirable to use as a gutta-percha solvent.[44] While methylchloroform is less toxic than chloroform, it is also less effective as a solvent for gutta-percha.[290] Both halothane and chloroform have been shown to affect the chemical composition of dentin[60,135] and may affect bonding strengths of adhesive cements to the altered dentin.[61,272] The clinical significance of these effects remains unknown, however. The evidence for the carcinogenicity of chloroform in humans is suspect,[170] but with careful use, its toxicity may be eliminated as a risk factor to both the patient[44] and the personnel in the operatory.[7] As such, its continued use as a gutta-percha solvent is recommended.

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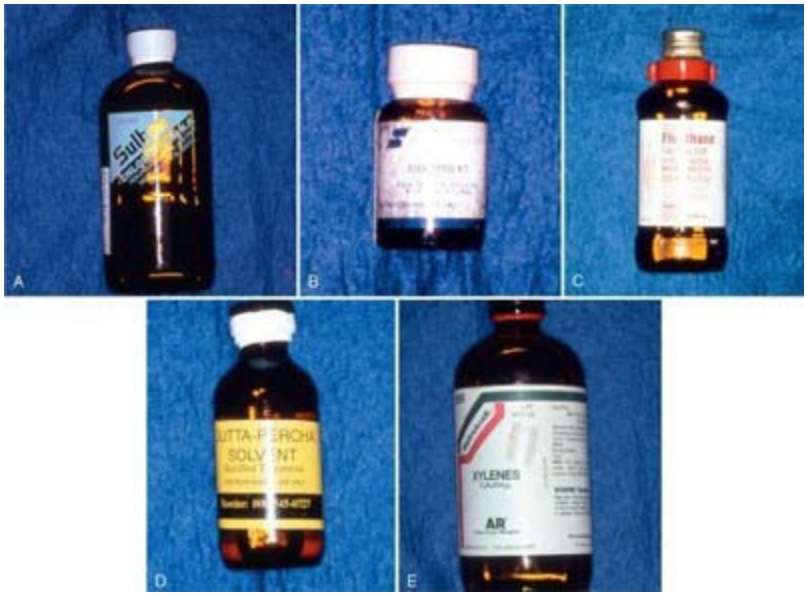

FIG. 10.32 **A,** Chloroform. **B,** Eucalyptol. **C,** Halothane. **D,** Rectified Turpentine. **E,** Xylenes.

Five sets of images, A to E show the solvents used to dissolve and remove gutta percha filling.

Using an irrigating syringe, the selected solvent is introduced into the coronal portions of the canals, which will then act as a reservoir for the solvent. Then, small hand files (sizes #15 and #20) are used to penetrate the remaining root filling and increase the surface area of the gutta-percha to enhance its dissolution. This procedure can be facilitated by using precurved, rigid files such as the C+ File (Maillefer, Baillagues, Switzerland) (Fig. 10.33), which can penetrate the gutta-percha mass more efficiently than the more flexible types of K-files. The C+ file is a stainless steel, end-cutting hand file that is twisted from a square blank. The secret to its stiffness is that the taper varies along the shaft giving it the rigidity and strength to cut through well-condensed gutta-percha efficiently. The gutta-percha must be removed carefully, however, to avoid overextending the resultant mixture of gutta-percha and solvent beyond the confines of the canal to minimize the risk of severe postoperative pain.[156] Unfortunately, electronic apex locators, which in nonretreatment situations are very accurate,[137] seem to misread the working length frequently when gutta-percha is initially being removed. This clinical observation may be due to the file being covered with chloropercha that may affect its conductivity. It has been shown that apex locators may be

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less accurate in retreatment situations[289]; however, in this study, the error was that readings indicated a working length that was too short. In a more recent study, an apex locator built into a rotary handpiece indicated working lengths that were too long in simulated retreatment situations.[283] Another recent study indicated that overestimation of length can occur in retreatment situations and could lead to overinstrumentation and overextension of root filling materials during retreatment.[164] It is recommended that a radiograph be made to gain a preliminary measurement when the estimated length is approached in order to avoid overextending root filling materials into the periodontium. Alternatively, recent studies have shown that CBCT imaging can be used to measure estimated working length more accurately than periapical radiographs.[304] If a CBCT was made for preoperative analysis of the tooth to be retreated, which is within the AAE/AAOMR guidelines, then the volume can also be used to estimate the preoperative working length.[151] Well into the retreatment, after the root fillings have been thoroughly removed, the apex locator will regain its accuracy if a clean file is used. Once the working length is reached, progressively larger diameter hand files are rotated in a passive, nonbinding clockwise reaming fashion to remove the bulk of the remaining gutta-percha until the files come out of the canal clean (i.e., with no pink material on them). Frequent replenishment of the solvent should be used, and when the last loose-fitting instrument is removed clean, the canal is flooded with the solvent, which then acts as an irrigant. The solvent is then removed with paper points. The wicking action of the absorbent points[214] will remove much of the remaining film of gutta-percha and sealer that remains adhering to the canal walls and in the irregularities of the canal system.[296] Cleanliness of canals after gutta-percha removal is not improved by using a microscope[16]; however, using kinked small files, the clinician should probe the canal wall looking for irregularities that may harbor the last remnants of gutta-percha. These irregularities can usually be felt rather than seen and should be cleaned using this method.[156]

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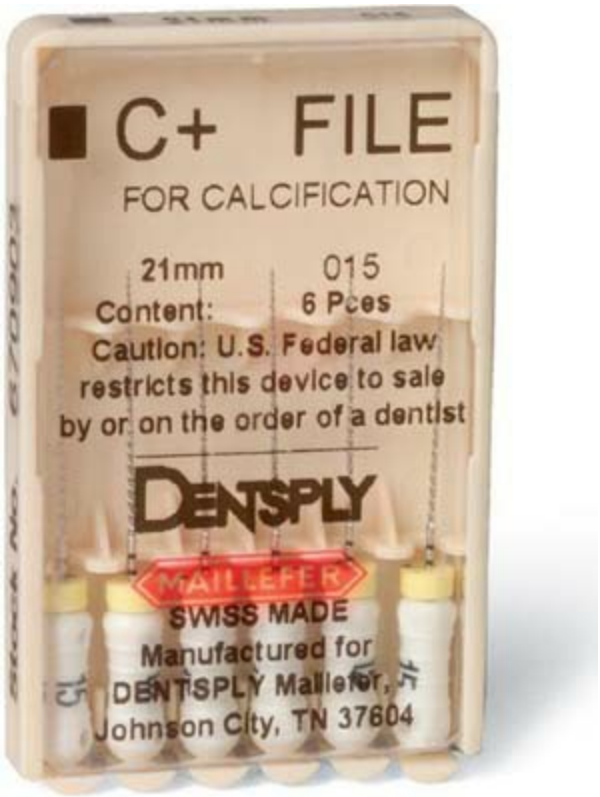

FIG. 10.33 C+ files. These rigid instruments remove gutta-percha more efficiently than more flexible types of K-files.

A pack of six stiff and strong C plus files.

It should be noted that there exists a glass ionomer-based endodontic sealer (Ketac-Endo, ESPE, Seefeld, Germany) that is used in conjunction with gutta-percha.[202] This sealer is virtually insoluble in both chloroform and halothane,[292] and must be retreated by removing the gutta-percha and then by using ultrasonics to debride the canal walls. Canal cleanliness can approach that of other gutta-percha retreatment cases, but it is difficult and time consuming to achieve this result.[76,176] Recently, bioceramic sealers such as EndoSequence BC Sealer (Brasseler, Savannah, GA) have been introduced. These materials become hard when set and are also resistant to dissolution and removal from the canal system.[54]

Overextended gutta-percha removal can be attempted by inserting a new Hedstrom file into the extruded apical fragment of root filling using a gentle clockwise rotation to a depth of 0.5 to 1.0 mm beyond the apical constriction. The file is then slowly and firmly withdrawn with no rotation, removing the

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overextended material (Fig. 10.34).[173] This technique works frequently, but care must be taken not to force the instrument or else further extrusion of the gutta-percha or separation of the file may result. The overextended apical fragment should not be softened with solvent as this application can decrease the likelihood of the Hedstrom file getting a solid purchase hindering retrieval.[254]

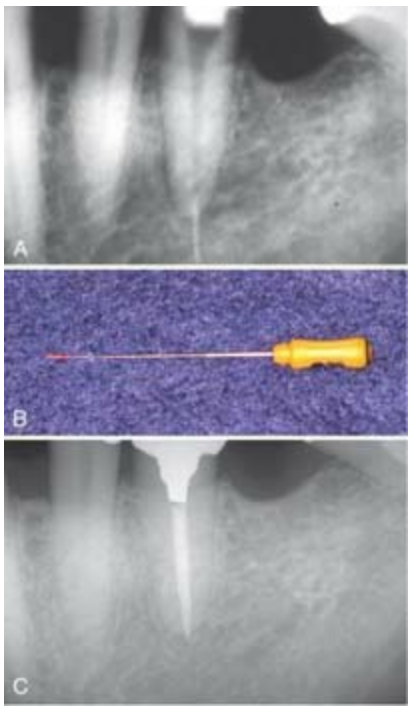

FIG. 10.34 Removal of overextended gutta-percha. **A,** Preoperative radiograph showing overextended filling material. **B,** A small Hedstrom file pierces the overextended material and retrieves it. **C,** Eighteen-month reevaluation. The tooth is asymptomatic.

A) Radiograph shows a radiolucent root canal of a tooth with an radiopaque outline of the C plus file.

B) Close-up of a used C plus file with removed gutta percha filling on the tip.

C) Radiograph of a tooth shows a radiopaque part of the crown with a radiopacity near the neck and upper canal and radiolucency in the lower canal.

Using rotary systems to remove gutta-percha in the canals has been advocated due to enhanced efficiency and effectiveness in removing gutta-percha from treated root canals.[214] This has generally been borne out in the

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literature. There are several types of mechanical rotary systems available for gutta-percha removal, including rotary file systems such as the ProFile (Dentsply, York, PA) (Fig. 10.35); a mechanical push-pull, quarter-turn file system, the Canal Finder (Endo Technique Co., Tustin, CA); and dedicated gutta-percha removal instruments, such as the GPX (Brasseler, Savannah, GA), the ProTaper Universal retreatment files (Dentsply, York, PA) (Fig. 10.36), and the Mtwo R (Sweden and Martina, Padua, Italy). These engine-driven instruments mechanically chop up the gutta-percha and sealer while thermoplasticizing the root filling mass via frictional heat to aid in removal. A recent survey of Australian dentists showed that 54% of the respondents who perform endodontic retreatment used rotary instrumentation to remove gutta-percha either always (15%) or sometimes (39%) with an increased likelihood of rotary gutta-percha removal if the clinician had more experience with the use of these instruments.[195] In vitro studies have generally shown these systems to be efficient in that they generally need less time to remove the bulk of the gutta-percha filling material than hand removal,[17,68,87,116,117,217,271] although in two studies, they were slower to remove the root filling than hand filing.[20,121] Assessments of canal cleanliness and extruded apical debris generally indicated that there were no overall differences between hand and mechanical gutta-percha removal.[20,68,116,117,122,187,217,246,271,310] In one study using Quantec SC instruments (Tycom, Irvine, CA), however, it was found that hand files with solvent cleaned canals more effectively.[27] This finding has been repeated recently using the ProTaper retreatment files,[103] but in another study in the same journal issue, ProTaper retreatment files were found to leave canals cleaner than hand files.[87] Clearly, this is an area where further research is warranted. It is recommended that after rotary gutta-percha removal, subsequent hand instrumentation is needed to remove the residual obturating materials completely from the canal. In several studies of mechanical gutta-percha removal, fracture of the mechanical instruments or the tooth root occurred.[17,27,32,117,121,271] However, this result was reported to occur less frequently when the instrument rotary speed was increased from 350 to 1500 rpm[32] and one study showed no separation or other canal defects when using dedicated retreatment files, the ProTaper Universal and Mtwo R instruments.[248] The dedicated retreatment files have end cutting tips to

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enhance penetration and removal of the root filling mass thus increasing their efficiency (see Fig. 10.36, *B*).[268] This, in combination with flute design and techniques advocated, may be the reason for the potential reduced risk of separation. In a more recent retrospective study, it was found that the incidence of instrument separation was significantly greater during retreatment than in de novo endodontic therapy.[6] While the mechanical gutta-percha removal systems may provide an enhanced efficiency, the increased risk of instrument separation, further complicating retreatment, may outweigh this benefit. Dedicated retreatment files may reduce this risk.

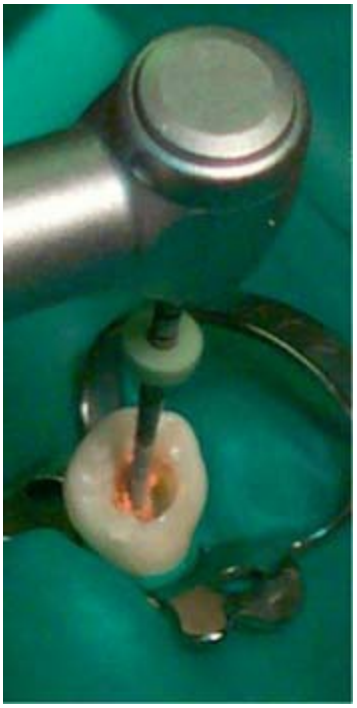

FIG. 10.35 Nickel-titanium rotary profile thermoplasticizing and removing gutta-percha. Optimum rotary speed is 1500 rpm.

Close-up of a nickel titanium rotatory file removing gutta percha filling from a perforated tooth held in a clamp.

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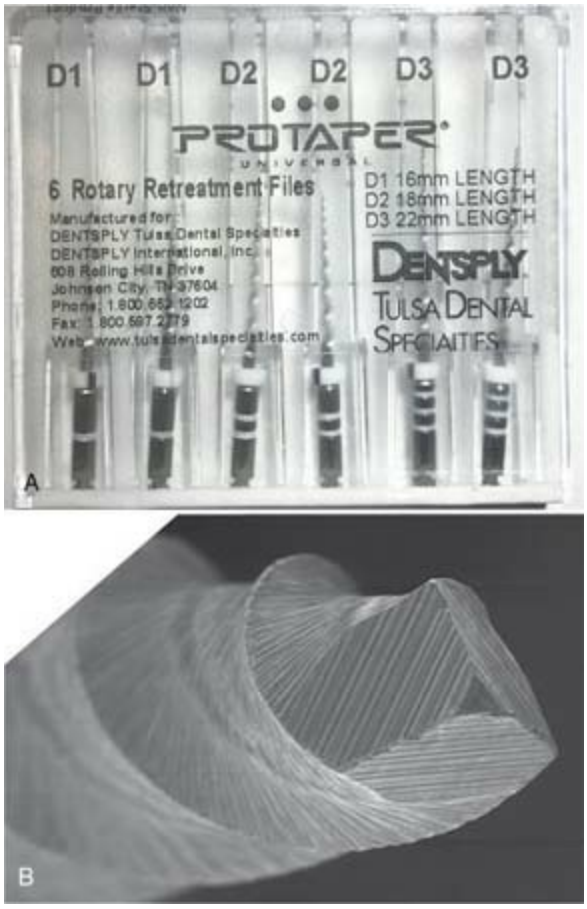

FIG. 10.36 Rotary gutta-percha removal instruments. **A,** ProTaper Universal Package. **B,** ProTaper Universal retreatment file has a cutting tip for enhanced penetration of the root filling materials.

A) Close-up of a pack of six nickel titanium rotatory file tips with diameters of 16 millimeters, 18 millimeters, and 22 millimeters.

C) Micrograph shows a textured and spiral tip of nickel titanium rotatory file.

Engine-driven instruments can also help with the removal of residual root filling materials after the bulk of the gutta-percha has been removed. A new instrument, the Self-Adjusting File (SAF) (ReDent, Ra'anana, Israel), has been recommended for removal of the root-filling residue that remains after root-filling removal. When the Self-Adjusting Files were used after rotary retreatment files, reduction of canal residues were 66%, 68%, and 81% in the coronal, apical, and middle thirds of the canal, respectively, when compared to using ProTaper Universal retreatment files alone.[4] Another study compared the use of ProTaper Retreatment files followed by F1 and F2 ProTaper instruments to the use of a #25.06 Profile followed by the Self-

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Adjusting File, for their effectiveness in removing gutta-percha-based root fillings.[247] With the use of high-resolution micro-CT and an arbitrarily selected threshold of less than 0.5% residue remaining as effectively cleaned, the ProTaper group had no cases meet this threshold while 57% of the cases in the ProFile/SAF group met the threshold. Unfortunately, in both of the previously mentioned studies, none of the retreatment methods rendered all of the treated canals completely free of all root-filling residue. The SAF file is considered a canal conforming file, and there have been new additions to this group recently, such as the TruShape file (Dentsply, York, PA) and the XP File system (Brasseler, Savannah, GA). These more recent conforming files have multiple curvatures along the length of the cutting segment of a single instrument core in order to increase the amount of contact between the instrument and the walls of pulp canals with irregular contours (such as oval-shaped canals) (Fig. 10.37). Studies to date show that these instruments can reduce the amount of remaining root filling materials after endodontic retreatment to a larger extent than traditional cylindrical core tapered engine-driven files, but they still do not render the canals completely free of all residual filling materials.[15,191]

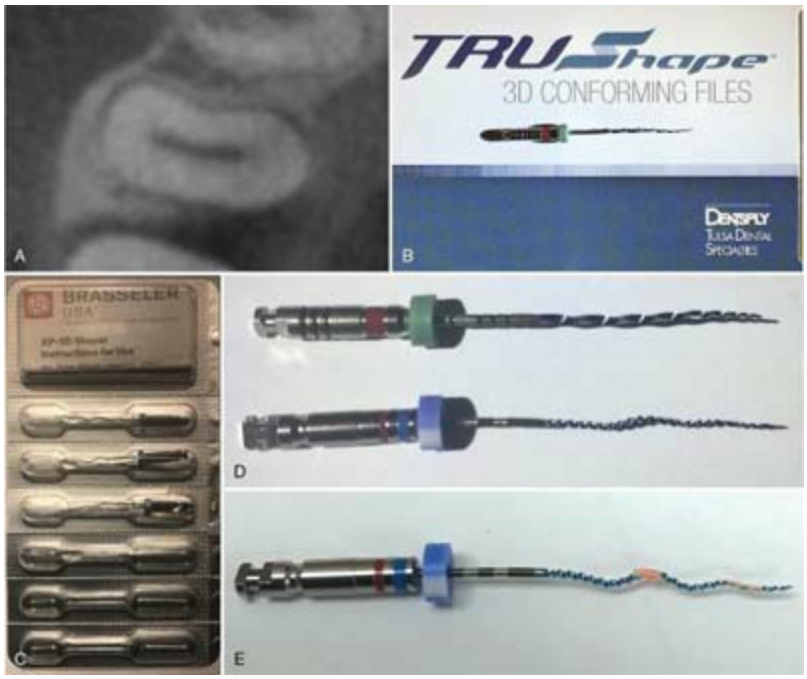

FIG. 10.37 Canal conforming files for enhanced cleaning of irregular and ovoid canals. **A,** CT slice on axial plane showing the ovoid nature

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of canal systems. **B,** TruShape package. **C,** XP System package. **D,** TruShape instrument *(top)* and XP Shaper Instrument *(bottom)*. **E,** XP Shaper instrument removed more gutta percha after the canal had already been retreated, cleaned, and shaped with traditional tapered instruments.

A) Radiograph of a horizontal section shows an ovoid shaped molar and an line-like canal.

B) Close-up of true shape, three dimensional conforming files used to clean and shape the complex canal anatomy.

C) Close-up of a pack of true shape files with six shapers.

D) Close-up of a close spirally textured instrument on the top and tight spirally textured shaper at the bottom.

E) Close-up of a tight spirally textured shaper with removed gutta percha on the tip surface.

Use of the Nd:YAG laser to remove gutta-percha from root-filled teeth has been investigated in vitro.[285] The time taken to remove the gutta-percha was within the range of other studies of mechanical gutta-percha removal, and the addition of solvents did not improve the performance of the laser. As in most other studies, gutta-percha, in varying amounts, was left in the canals after laser removal. Root surface temperatures did increase, however, and without further investigation proving safety and efficacy, laser gutta-percha removal cannot be recommended at this time.

Resilon (Resilon Research, LLC) (Fig. 10.38) is a thermoplastic polyester polymer that was bonded into the canal space using an unfilled resin bonding system (Epiphany, Pentron Clinical Technologies, Wallingford, CT). It was also marketed as RealSeal (Sybron Endodontics, Orange County, CA). It was advocated as a root canal obturating material to replace traditional gutta-percha and sealer due to its apparent, enhanced sealing ability[238] and potential to strengthen root resistance to fracture as a result of internal bonding.[270] Resin-bonded obturation systems have been advocated in the past[149]; however, the inability to retreat canals filled with this obturating material has prevented its widespread use. The Resilon polymer itself is reported by the manufacturer to be soluble in chloroform and may be removed by heat application, a behavior that is like gutta-percha. Recent studies have shown that the polycaprolactone polymer of the Resilon is

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removed easily and leaves canal walls cleaner than removal of gutta-percha and AH+ sealer,[50,52,65,227] although this finding has been disputed. Hassanloo et al. found that there was less residue on the walls when removing gutta-percha/AH+ if the materials were allowed to set for a longer period.[108] This finding has been corroborated[227,269] and indicates that there may be a temporal effect, which introduces a methodological bias in these studies. There may also be a problem with removal of the unfilled Epiphany resin sealer, especially since the sealer tags have been shown to penetrate deep into dentinal tubules[238] and presumably also into anatomic ramifications of the canal that need cleaning during retreatment. After the Resilon core has been removed using heat and chloroform, the authors would recommend the use of a resin solvent such as Endosolv-R (Septodont, Paris, FR) (Fig. 10.39) to attempt elimination of the unfilled resin sealer prior to instrumentation.

FIG. 10.38 Epiphany Obturation System using Resilon material.

A Resilon-Epiphany system with three components including a resilon, epiphany sealer and the primer is kept next to other instruments on a table top.

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FIG. 10.39 Endosolv-E *(left)* and Endosolv-R *(right)*.

Set of two resin solvents used to remove the unfilled epiphany resin sealer before retreatment and paste fills.

Unfortunately, recent studies have shown that the Resilon material is not stable and breaks down within the tooth over time, presumably due to enzymatic degradation.[111] The authors have retreated cases obturated with Resilon and have found discolored, soft, easily removed breakdown materials in the canal. While this may make retreatment easier, it also appears to negatively affect the long-term success of teeth obturated with this material and it was withdrawn from the market in 2014.[18] Nevertheless, clinicians should be aware of it since, like any other obturation technique, there will be a need to retreat these cases occasionally.

Managing solid core obturators

Solid core canal obturation systems, such as Thermafil, Dens-Fil, and the GT Obturator (Dentsply Endodontics, Tulsa, OK), have become very popular since their introduction several years ago. After cleaning and shaping procedures are completed, the clinician, using this technique, heats a solid core obturator (alpha-phase gutta-percha surrounding a core that is attached to a handle) in an oven and places the carrier in the canal. The solid core carries the gutta-percha down in the canal and condenses it while the material is cooling. This system provides a rapid and simple technique for warm gutta-

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percha endodontic obturation; however, as with any obturating material, retreatment will be necessary occasionally.

Retreatment of solid core materials is considered to be more complex and difficult than is the case with removal of gutta-percha alone due to the presence of the solid carrier within the mass of gutta-percha. The nature of the carrier will determine the method used and complexity of the retrieval. There are three types of carriers found in these systems: metal (stainless steel or titanium), plastic, and modified gutta-percha. The plastic carriers are smooth sided, as are some brands of metal carriers; however, most metal carriers are fluted and resemble endodontic hand files with a layer of gutta-percha on the outside. The fluted metal carriers present an exceptional challenge to the retreating clinician since many times they are improperly inserted and either wedged or screwed into the canal to make up for inadequate canal shaping or the lack of skilled use of the size verifying techniques available. This makes them especially difficult to remove. Once the carrier has been placed, it is cut off in the pulp chamber using a bur and the tooth is restored. The level at which the metal carrier is severed is important in its retrieval. If it is cut down to the level of the canal orifice, retrieval is difficult,[156] so the prudent clinician plans for retrievability by severing the handle from the carrier, leaving 2 to 3 mm of carrier exposed in the access above the pulp chamber floor to allow easier removal if retreatment is ever needed. Unfortunately, this is not always the case. Some clinicians place a nick in the mid-canal level of the carrier to allow the clinician to rotate the carrier handle and sever the obturator deep in the canal. This technique is used to allow creation of a post space; however, the rotational force used to create the “twist off” apical plug can engage the flutes of a metal carrier, increasing the complexity of removal if retreatment is needed.[309]

It is advantageous to determine prior to initiating treatment if there is a solid core obturation in the root-filled tooth. The preoperative radiograph may show this, since the stainless steel carriers will exhibit a fluting effect on the radiograph (Fig. 10.40); however, the titanium carriers rarely are distinguishable from gutta-percha, and the plastic ones never are. Unfortunately, in most instances, the clinician finds that they are dealing with a carrier-based obturator after initial access to the pulp chamber. This is why,

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as was stated in an earlier section, careful access and probing of the root filling material is necessary when entering a canal. If there is a carrier, it will be detected as either a metallic structure embedded in the gutta-percha mass or a black or gray spot indicating a plastic or modified gutta percha carrier. Occasionally, the carrier may be found embedded in the coronal core material and so careful excavation with small burs and straight, tapered ultrasonic tips may be necessary to preserve the carrier intact to help with removal.[156]

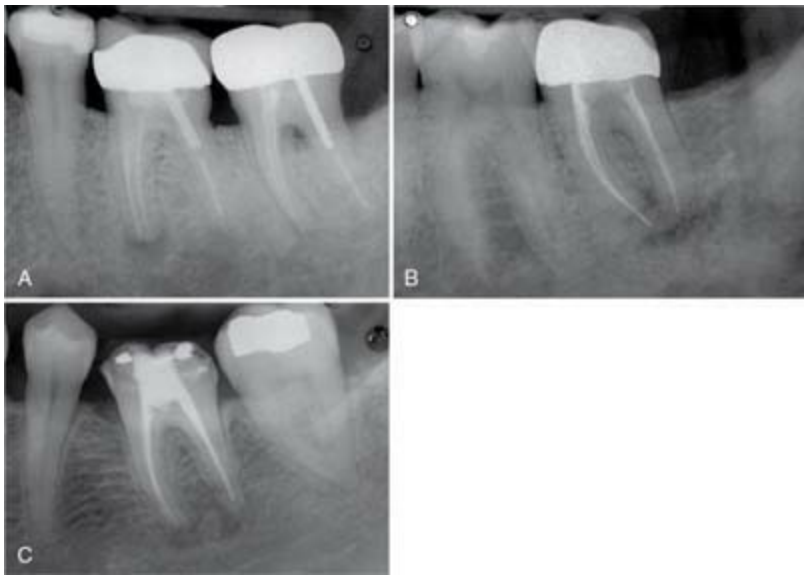

FIG. 10.40 Comparison of radiographic appearances of three different obturating materials. **A,** Gutta-percha. **B,** Stainless steel Thermafil carrier (note subtle fluting effect in the fill). **C,** Plastic Thermafil carrier.

A) Radiograph of teeth with first and second molar showing radiopaque crowns with outline of gutta percha in their partial right root canals.

B) Radiograph of teeth with a molar showing radiopaque crown with outline of metallic thermafil carrier in the root canals

C) Radiograph of teeth with first molar showing an outline of radiopaque plastic thermafil carrier and second molar showing a while radiopaque patch on crown.

Removal of a metal carrier is accomplished with initial use of heat application to the carrier that can soften the gutta-percha surrounding it, facilitating its removal with Peet silver point forceps (Silvermans, New York, NY) or modified Steiglitz forceps (Union Broach, York, PA) (Fig.

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10.41).[156,214,293,295] Often, there is not enough of the carrier remaining in the access to grasp with forceps, and so removal will require solvent application and removal of the surrounding coronal gutta-percha using small hand instruments, usually followed by ultrasonic excavation around the carrier and removing it like a separated instrument[156,214] (Fig. 10.42) as described in a later section of this chapter. Care should be exercised to avoid excessive heat generation during this procedure. This is also the case if the metal carrier has been sectioned for post space preparation. The metal carrier has been shown to be much more difficult to remove than plastic ones,[71,309] frequently resulting in nonretrieval. Fortunately, their use in endodontic therapy has been declining.

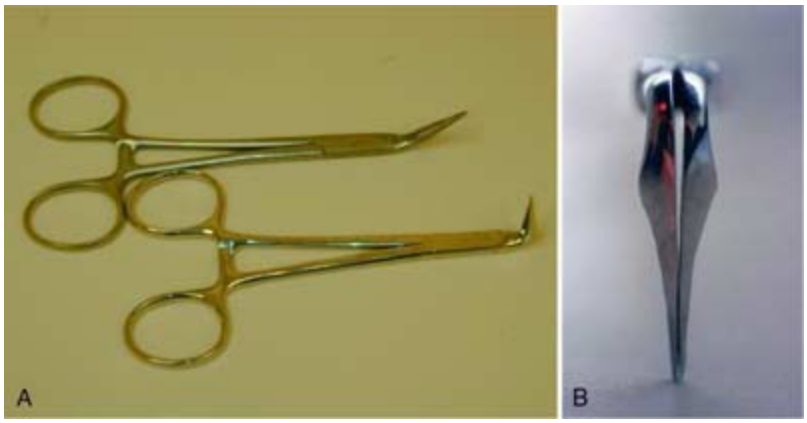

FIG. 10.41 **A,** Steiglitz forceps in 45- and 90-degree head angles. **B,** Tips of the Steiglitz forceps ground to a thinner contour to create the "modified" instrument. This allows deeper penetration into the tooth to enhance removal of obstructions.

Two set of images show tools used for the total removal of the remaining carrier obstructions by the instruments labeled in A and B.

Source: (*B,* Courtesy Dr. Daniel Erickson.)

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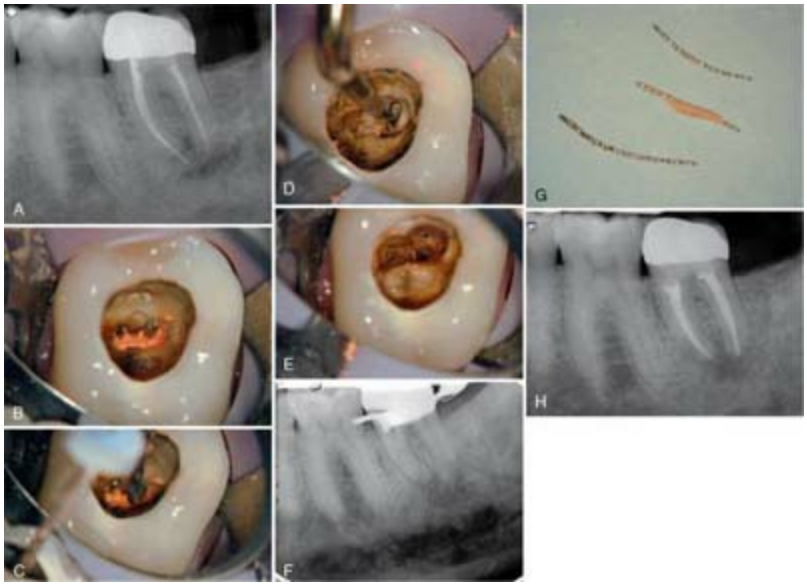

FIG. 10.42 Metal carrier retreatment. **A,** Preoperative radiograph. **B,** Metal carriers exposed by careful excavation of gutta percha. **C,** Use of the Touch 'n Heat instrument to heat the carriers and soften the gutta-percha. This allowed removal of one of the carriers using modified Steiglitz forceps. The other could not be removed using heat or solvents. **D,** Ultrasonic troughing around the carrier to facilitate grasping it with forceps. **E** and **F,** Carriers removed and confirmed with a radiograph. **G,** Metal carriers showing gutta-percha still adhering to them. **H,** Final obturation of the tooth.

A) Radiograph of teeth with first molar showing radiopaque crown with radiopaque canals. Right root canal is visibly cut off from the crown.

B) Close-up view of a molar with open crown shows the decayed dentin with residue of gutta percha exposing parts of metal carriers.

C) Close-up view of a heated autoclavable tip inserted in the molar with open crown exposing the gutta percha and parts of metal carriers.

D) Close-up view of a molar with open crown shows removal of other obstruction by troughing and further removal by forceps.

E) Close-up view of a molar with open crown shows complete removal of metal carriers.

F) Radiograph of teeth with first molar showing radiopaque crown with translucent canals.

G) Close-up of three textured metal carriers show one of them with the removed gutta percha on the tip surface.

H) Radiograph of teeth with first molar showing radiopaque crown with root canals and radiolucency around the neck.

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Removal of plastic carriers is similar to removal of gutta-percha root fillings, except that, generally, heat should be avoided to minimize the likelihood of damaging the carrier.[156] The older Thermafil plastic carriers were made of two different materials depending on their size. In the smaller sizes (up to size #40) the material used was Vectra, which is insoluble in available solvents; whereas the larger sizes used polysulfone, which is soluble in chloroform.[196] Solvents, on the other hand, seem not to affect the newer GT plastic carriers, so their use can be recommended (Fig. 10.43).[26] The access is flooded with a solvent, such as chloroform, and the gutta-percha surrounding the carrier is removed with hand files in a larger to smaller sequence (e.g., #25, #20, #15), with each file progressively penetrating deeper around the carrier. The solvent should be replenished frequently, and when a #08 file can penetrate to the apical extent of the carrier and there is little remaining gutta-percha, a larger Hedstrom file is inserted into the canal alongside the plastic carrier and gently turned clockwise to engage the flutes. When the file is withdrawn, it invariably brings the carrier with it, and the rest of the gutta-percha and sealer removal proceeds as described earlier. Care must be taken to avoid overstressing the Hedstrom file. It should not be “screwed” into the canal or separation of the file or the carrier may result.[26] Occasionally, grasping pliers will be needed to remove the carrier if it is accessible[123,214] after the gutta-percha has been removed. Another potential problem with retrieval is present if the carrier has been overextended beyond the apical foramen during the previous root canal treatment. This overextension may make it prone to separation and unable to be retrieved, potentially resulting in the need for apical surgery.[120]

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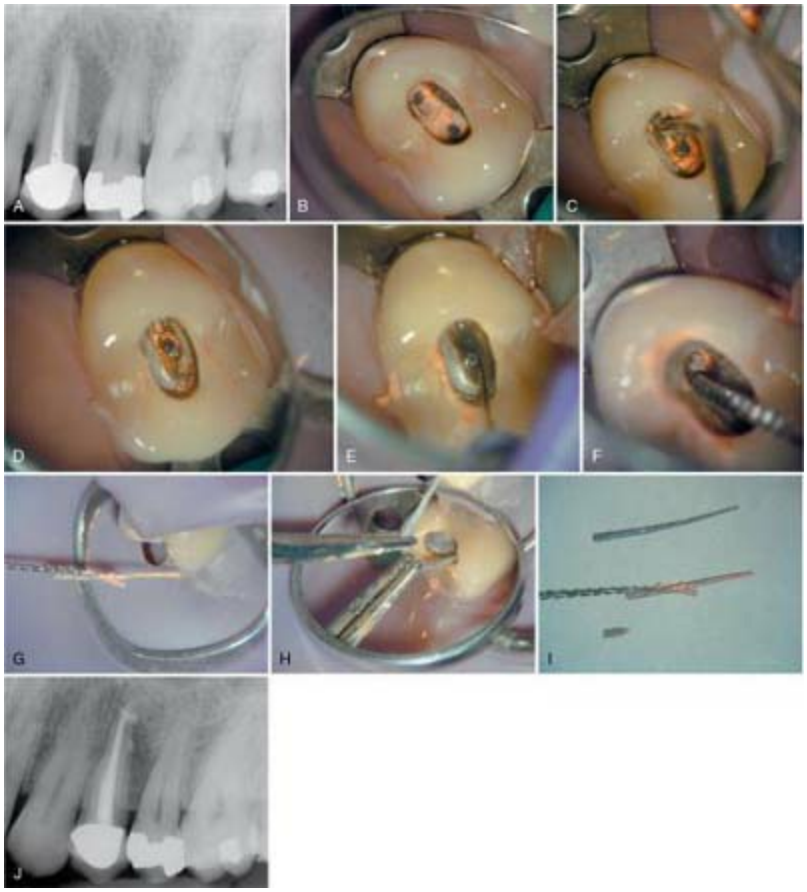

FIG. 10.43 Plastic carrier retreatment. **A,** Preoperative radiograph. At this stage, the nature of the root filling is unknown. **B,** Plastic carriers visible in the access as two black spots in the gutta percha mass. **C,** Gutta percha in the chamber is carefully removed from the carriers. **D,** Carrier is exposed. **E,** Chloroform solvent is placed into the chamber and a small file is worked alongside the carriers to remove the gutta percha. **F** and **G,** A Hedstrom file is gently screwed into the canal alongside the carrier and withdraws it on removal. **H,** A hemostat removes the other carrier. **I,** Plastic carriers removed. **J,** Final obturation with Resilon and Epiphany sealer.

A) Radiograph of teeth with first tooth showing radiopaque crown with radiopaque partial canal. Second tooth shows a radiopaque patch in the crown while third and fourth teeth show radiopaque patches in the parts of crowns.

B) Close-up view of a molar with open crown shows residue of gutta percha exposing two black dots depicting plastic carriers.

C) Close-up view of a heat plugger inserted in the molar with open crown exposing the gutta percha and parts of plastic carriers.

D) Close-up view of a molar with open crown shows completely exposed plastic carrier obstruction.

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E) Close-up view of a molar with open crown shows a syringe tip placing chloroform inside the molar cavity.

F) Close-up view of a molar with open crown shows a hedstrom file extracting the carrier.

G) Close-up view of a molar with rubber dam clamp shows hedstrom file with the extracted carrier.

H) Close-up view of a molar with rubber dam clamp shows hemostat removing the other carrier.

I) Close-up of three textured plastic carriers show two of them with the removed gutta percha on the tip surface.

J) Radiograph of teeth with first tooth showing radiopaque crown with radiopaque canal. Second tooth shows a radiopaque patch in the crown while third tooth shows radiopaque patches in a part of crown.

Recently, a technique for plastic carrier removal has been described using a System B HeatSource (Sybron Endodontics, Orange, CA) to soften the gutta-percha surrounding the carrier without melting the carrier itself.[299] The temperature is set at 225°C, and the heat plugger is placed buccal and lingual to the carrier after which #50-55 Flex-R hand files are placed around the carrier and braided to engage the carrier and remove it. This technique has been shown to require significantly less time to remove the carrier compared to using solvent[299]; however, concerns regarding heat generation in the periradicular tissues have been raised[154] with the authors concluding that caution should be exercised when using this technique. When other techniques have been unsuccessful and the plastic carrier has been sectioned apical to the orifice resulting in limited access, the clinician may attempt to retrieve the carrier by placing a heated System B tip directly into it. As apical pressure is maintained, the heat is turned off. This allows the plastic carrier to adhere to the tip while cooling and may result in its removal upon withdrawal of the heat tip (Fig. 10.44).

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FIG. 10.44 Plastic Thermafil carrier adhering to a System B heat plugger.

A heat plugger with a conical tip and a right angled body is kept on a platform.

Rotary instruments have been advocated for use in removal of plastic carriers and gutta-percha, and a recent study showed that removal of plastic carriers was successful in all but one of the teeth obturated with them.[17] Unfortunately, root fracture occurred in the lone specimen that did not have a successful retrieval, and two instances of rotary instrument fracture also occurred in the study. As with gutta-percha removal, the clinician must carefully weigh the risks of rotary carrier removal against the perceived benefits.[213]

Modified gutta-percha is also being used as a core material for solid core obturation. This obturation system is called GuttaCore (Tulsa Dental Products, Tulsa, OK) and, at first glance, it appears very similar to plastic carrier-based systems. With GuttaCore, however, the carrier is fabricated from cross-linked gutta-percha rather than plastic. The cross-linking connects the polymer chains, which changes the material and gives the carrier different properties than the plastic carriers. The gutta-percha surrounding the carrier is alpha-phase gutta-percha, which is identical to that which encompasses the plastic carrier-based systems.

To understand how to effectively remove this type of filling material, one must understand how it is used. Obturation with GuttaCore is performed in a similar manner as obturation with Plastic Thermafil. The obturator is heated in a proprietary oven and then gently placed into the canal. While it is flexible, the modified gutta-percha carrier is much more brittle than the plastic carrier, and too much insertion force will cause the shank to collapse and the handle can break off prior to full insertion. Thus it is very difficult to

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wedge these obturators into underprepared canals and usually, in that instance, a short fill will result. This forces the operator to either accept a short fill or, ideally to enlarge the canal preparation prior to inserting the obturator, but in either case, retrieval of the root filling during retreatment is made easier by not having the carrier wedged into the canal.

While performing initial obturation, when it is time to section the carrier, the clinician will experience another difference from plastic carriers. While the use of a heat generating Prepi bur (Tulsa Dental Products, Tulsa, OK) or a System B Heat Source (Sybron Dental Specialties, Orange, CA) have proven to produce predictably reliable results in terms of sectioning plastic carriers at the desired level, heat has virtually no effect on the GuttaCore carrier and the authors found using heat to section the carrier to be unsuccessful. The modified gutta-percha core will not melt even when probed with a Touch ‘n Heat (Sybron Dental Specialties) tip at full power.

Because of the brittleness of the modified gutta-percha carrier, GuttaCore will separate by simply bending the carrier away from the access cavity wall. The material has very little ability to resist even the smallest of lateral forces without separation and so simply pushing down laterally on the carrier handle will separate the carrier handle from the canal fill. This has implications for retreatment since this separation method will generally result in the carrier being separated at or near the canal orifice level leaving little or nothing to grasp with forceps when attempting removal of the carrier.

Removal of the GuttaCore obturators is directly affected by the properties of the modified gutta-percha carrier, and so it is somewhat different from the approach taken to remove plastic carriers. Because of this, it is imperative that the clinician who has discovered a solid core, nonmetallic obturator while disassembling a tooth for nonsurgical retreatment can discern what type of core they are trying to remove. There are two ways to do this. First, the color of the modified gutta-percha carrier is pink or gray while the color of the plastic carriers is black (Fig. 10.45). Second, because the GuttaCore carrier is heat resistant, the clinician can lightly touch the coronal extent of the unknown carrier to see if it melts (plastic carrier) or if it does not (modified gutta-percha carrier). Once the nature of the carrier is determined, then the clinician can proceed with removal. If it is plastic, then the methods of removal described previously can be used, but if it is a GuttaCore carrier, then a different approach will be needed.

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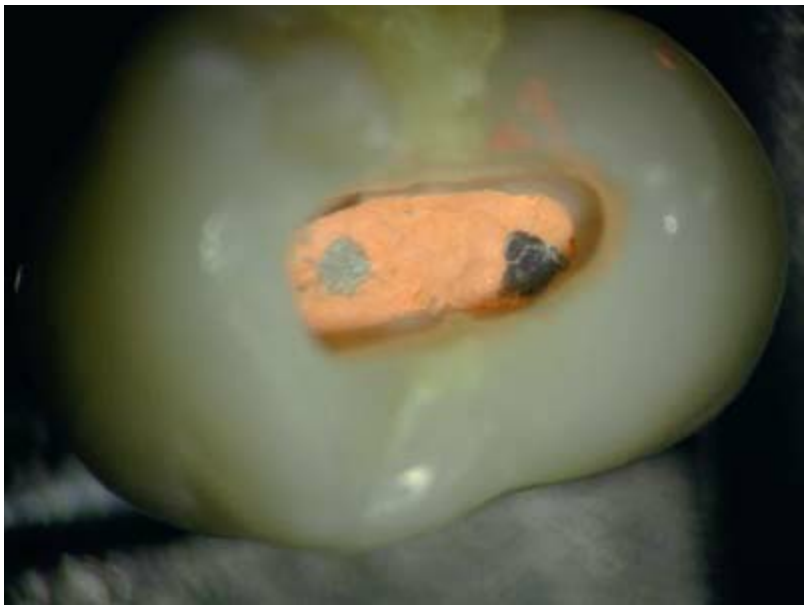

FIG. 10.45 Two different solid core carriers in an endodontic access preparation upon initial exposure. GuttaCore is on the left and a plastic carrier Thermafil Plus is on the right. Note the lighter grey color of the new modified gutta-percha core compared to the plastic carrier.

Close-up view of a molar with open crown shows residual gutta percha exposing a white dot depicting guta core in the left and a black dot depicting plastic carrier in the right.

The use of heat and solvents, which are two of the most common methods for endodontic retreatment, have no effect on the GuttaCore carrier. To date, the authors are unaware of any known solvents or chairside heat sources that can soften the cross-linked gutta-percha carrier. Also, the use of hemostats or other grasping pliers have shown inconsistent results. When straight-line access can be achieved and a few millimeters of the carrier is able to be grasped removal may be accomplished; however, due to the brittleness of the GuttaCore carrier many times it will end up separating at the apical extent of the grasping forceps. In spite of those concerns, the authors have found that Guttacore can be removed quite easily with predictable results using a variety of hand, rotary, and retreatment instruments. One study found that when using ProTaper retreatment files, GuttaCore was more efficiently removed from moderately curved canals than either thermoplasticized gutta percha or plastic carrier obturation.[22]

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The authors of this chapter have investigated various methods of removal of GuttaCore and confirmed the previous observation from the literature. The modified gutta-percha carrier is usually easily chopped up and removed by rotary ProTaper and reciprocal WaveOne (Tulsa Dental Products, Tulsa, OK) instruments and any remaining fragment of the carrier in the apical extent of the canal can be removed or penetrated using hand instruments. A problem arises, however, if the modified gutta-percha carrier has been overextended beyond the apical foramen. The authors' experience shows that upon removal of the segment of the carrier within the canal, the overextended tip of the carrier will separate, remaining in the periapical tissues. This potentially contaminated foreign material becomes difficult and many times impossible to remove. If this occurs, and post-retreatment apical periodontitis persists or recurs after initial resolution, then apical surgery or extraction will be necessary.

Another approach is to treat the carrier like some hard paste fills which the clinician is unable to find a solvent for. Using this approach, the clinician will find that using ultrasonic instruments in a similar fashion as they are used to remove pastes to be very helpful. Once a proper access preparation has been created, GuttaCore can be safely and easily removed with the use of ultrasonic instruments in the straight coronal portion of the canal. Upon reaching the canal curvature, the authors recommend using hand files or rotary files with safe noncutting tips in order to avoid perforation or excessive thinning of canal walls.

Once the carrier has been removed, the remaining gutta-percha and sealer must be removed from the canal, and like removal of root-filling materials described previously, there is no technique that completely removes all materials from the canal system. Canal cleaning may be even more difficult when removing carrier-based obturations since the more highly processed gutta-percha used with these carriers may be more difficult to remove than other forms of the material. Wilcox and Juhlin described a sticky film of gutta-percha and sealer adhering to the canal walls upon removal of metal carrier obturators and found more of it than if the canals had been obturated with lateral condensation.[295] Their findings have not been corroborated, however, and other studies have shown no difference in residual debris remaining after carrier-based removal.[71,123,309] It is important to remove as

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much of the residual gutta-percha and sealer from the pulpal anatomic spaces as possible, so flooding the canal with solvent and “wicking” it out with paper points is also recommended for carrier-based retreatment.[214]

Paste retreatment

Various pastes have been used as root canal obturating materials, especially outside of North America. Because of the wide variety of paste compounds used in endodontics, it is impossible to categorize them all. The individual clinician who is using it formulates most pastes, so the ultimate composition of a paste found in a tooth with persistent disease is generally indiscernible. Many of the pastes used, such as N2 or RC2B, contain formaldehyde and heavy metal oxides and so are toxic and potentially present a danger to the patient’s health, both local and systemic if overextended beyond the confines of the root canal system.[30,194] None has the potential to seal the canal effectively,[102] and many render a tooth impossible to retreat,[228] so their use is strongly discouraged. On radiographic examination, they can usually be discerned due to their lack of radiopacity, the presence of voids, and they usually show evidence of inadequate canal shaping and poor length control (Fig. 10.46). When a paste fill is suspected or found in a tooth, a telephone call to the previous treating dentist should be made, if possible, to find out the exact formulation of the paste, since this information may help in its removal.

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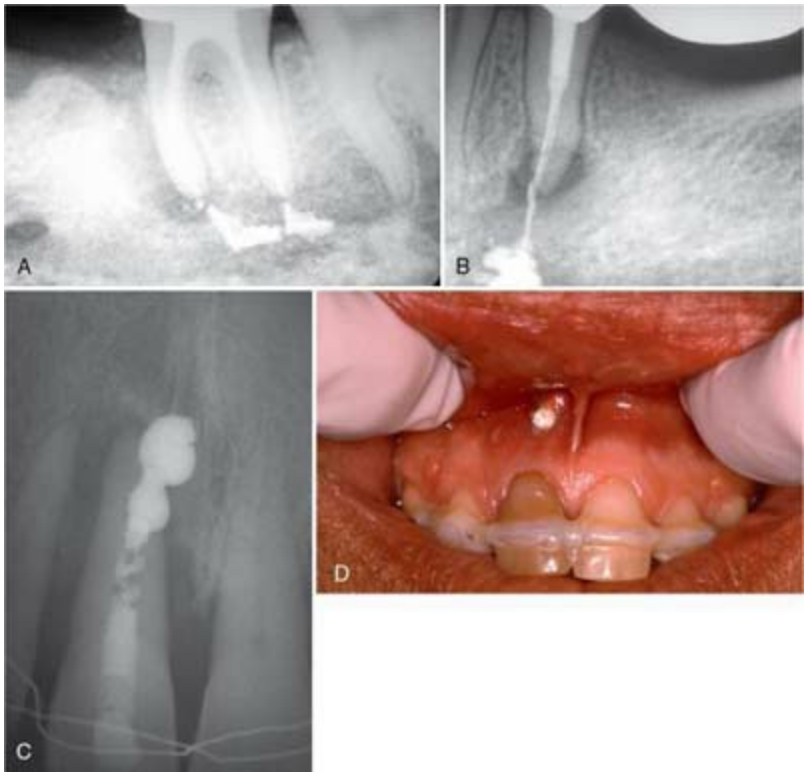

FIG. 10.46 Example of poor length control with paste root fillings. **A,** Overextended paste filling into the inferior alveolar canal. **B,** Overextended paste filling into the mental foramen. **C,** Overextended paste filling extending through a perforation in an upper central incisor. **D,** Clinical appearance of the case in *C*. Note the material extending out through a sinus tract.

A) Radiograph shows a molar with radiopaque root canals and radiolucent pulp. Two irregular L-shaped white patches of paste filling below the canals are visible.

B) Radiograph shows a tooth with radiopaque root canal and radiolucent pulp. An irregular projection of white patch of paste filling below the canal is visible.

C) Radiograph shows a tooth with parts of radiopaque radiolucent canal extending into a white smudge across the tooth surface.

D) Close-up an open mouth held with gloved thumbs shows an illuminated dot over the upper gum. An exposed central left incisor is stained and a material tract extends across the frontal teeth.

For purposes of retreatment, paste fills can be categorized as soft or hard, and all should be considered potentially toxic. Great care should be exercised when removing the paste to avoid overextension, potentially severe

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postoperative pain,[97] and possible paresthesia/dysesthesia from the paste's potential neurotoxicity.[33,235] Soft pastes are generally easy to remove using crown-down instrumentation with copious sodium hypochlorite irrigation to minimize extrusion.[156] Greater difficulty arises when the paste is set hard.[228] Since the nature of the paste remains unknown, removing it becomes an empirical process. Following access preparation and coronal orifice exposure, the paste is probed with an endodontic explorer and files. If hard and impenetrable, then the coronal paste can be removed with burs[78] or a straight, tapered ultrasonic tip in the easily accessible straight portions of the canal using magnification and illumination.[214] Once the canal curvature is reached, further use of this method will result in damage to the canal walls and possible perforation. Precurved, small hand files are inserted to probe the apical area. Many times, the density of the paste filling material decreases in the apical extent of the fill so that penetration to the apex may be possible.[214] If not, a solvent must be used to attempt to soften the remaining paste. The choice of solvent is usually made by trial and error starting with chloroform. If that does not soften the material in a reasonable amount of time and does not allow penetration with small files, then the chloroform is wicked out of the canal and another solvent is chosen. There are two frequently used solvents for paste fills: Endosolv-E and Endosolv-R (Septodont, Paris, FR) (see Fig. 10.39). The Endosolv-E is selected if the paste contains zinc oxide and eugenol, and the Endosolv-R is chosen for resin-based pastes. The obvious problem is that the nature of the paste is usually unknown at the time of removal, so contacting the previous clinician, if possible, can help with this choice. Otherwise, the choice is just a guess. The chosen solvent should be placed in the access, and attempts should be made to penetrate the paste with hand or ultrasonic files; however, care must be taken to avoid creating a ledge or other defect in the canal that may preclude successful retreatment. The progress is frequently slow,[81] and the clinician may elect to leave some solvent in the canals between appointments to soften the paste.[214] Care should be taken in the choice of temporary restoration since the solvent left in the canal may also soften the temporary, potentially leading to breakdown of the interappointment seal.[175]

Ultrasonically activated files have been advocated for use in penetrating hard-set pastes in the curved apical segments of canals (Fig. 10.47).[130,140]

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The ultrasonic energy breaks up the paste, and the irrigation floats the fragments in a coronal direction until the apical terminus is reached.[78] This technique is reported to be very time consuming, and care must be exercised to avoid instrument separation, perforations, or alteration of canal morphology. On occasion, despite all best efforts, the paste cannot be removed from the tooth, so apical surgery or extraction should be considered in these cases.[97]

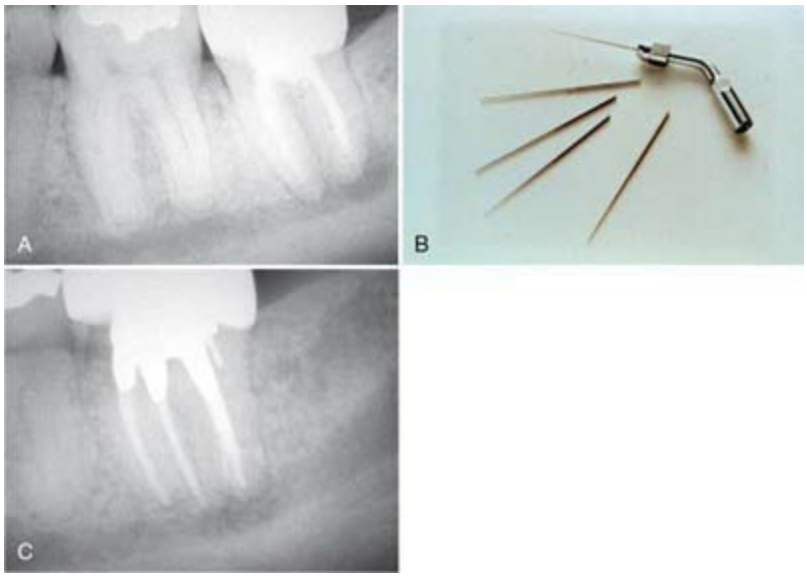

FIG. 10.47 **A,** Preoperative radiograph of a hard paste root filling exhibiting a short fill, inadequate seal, and periapical radiolucency. Note the proximity of the inferior alveolar canal. **B,** Ultrasonic files like this were used to break up the hard paste in the apical third of the canal allowing removal. **C,** Seventeen-month postoperative follow-up. The patient is asymptomatic and has no paresthesia.

A) Radiograph of first molar shows radiopaque crown and root canal. Second molar shows a radiopaque white patch in a part of crown depicting outline of the hard paste filling.

B) An image shows an ultrasonic curved file with one needle device attached to it and other four on the surface.

C) Radiograph of the molar shows radiopaque crown, neck and upper canals with radiolucent lower canals.

Biocalex 6.9 (currently known as Endocal 10) (Fig. 10.48) is a hard setting calcium oxide paste that has been popular in Europe for over 30 years, but is

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now being used in North America since its recent FDA approval.[93] The paste seems to seal well, but there is an unacceptably high incidence of root fracture due to the large amount of expansion on setting.[93] Retreatment will be complicated by the hard setting nature of this material; however, since it is a calcium oxide paste, EDTA may soften it, facilitating its removal. Since the EDTA also softens dentin, care must be taken not to gouge or ledge the canal walls during retreatment, and root fractures must be suspected as a potential complicating factor in the outcome.

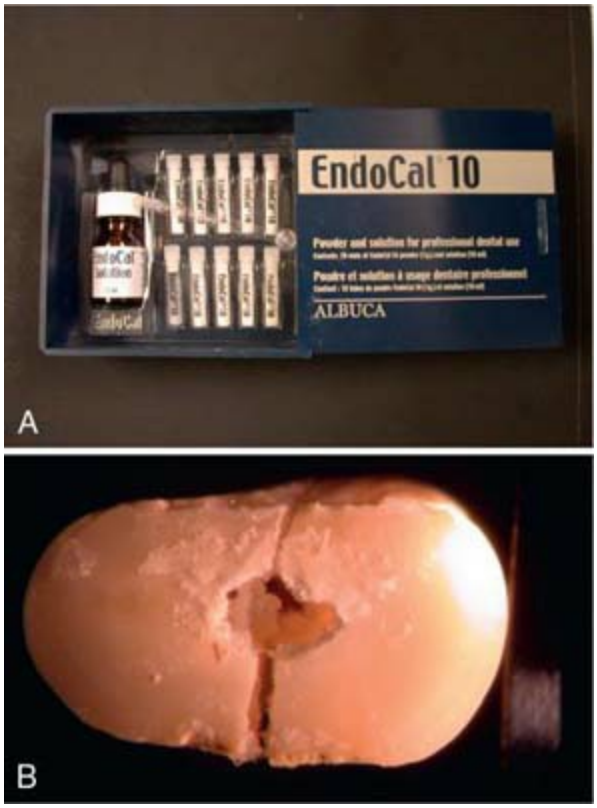

FIG. 10.48 **A,** Endocal 10 (formerly known as Biocalyx). **B,** Split root in a case filled with Endocal 10.

A) Close-up of setting calcium oxide kit, endocal 10, with one solution bottle and ten powder cartridges.

B) Close-up of a horizontal section of molar exposing a crack filled by calcium oxide paste.

Source: (Courtesy Dr. Rob Goldberg.)

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Silver point removal

Historically, the use of silver points for endodontic therapy has been extremely popular and quite successful because of their ease of handling and placement, ductility, radiopacity, and that silver appears to have some antibacterial activity.[230] However, over the past few decades, the usage of silver points has dramatically diminished, so presently they are considered a deviation from the standard of care.[307] The main reason for this change is because they corrode over time (Fig. 10.49), and the apical seal may be lost.[31] Also, silver points do not produce an acceptable 3D seal of the canal system; rather, they simply produce a plug in the apical constriction while not sealing the accessory canals that are frequently present (Fig. 10.50).[158,226] The corrosion of silver points occurs when they come in contact with tissue fluids and certain chemicals used in endodontics, including sodium hypochlorite and some sealers.[99] This corrosion produces chemicals such as silver sulfide, silver sulfate, silver carbonate, and silver amine hydrate,[233] which have been shown to be cytotoxic in tissue culture.[230] Corrosion occurs mainly at the apical and coronal portions of the points indicating that leakage is responsible.[233] Gutta-percha root-filling techniques do not suffer from these disadvantages and have replaced the use of silver points in endodontics. Due to this decrease in usage over the past quarter of a century, the quantity of cases the clinician will come across that will require silver point removal has also decreased. Nevertheless, there are still occasions when their removal will be necessary.

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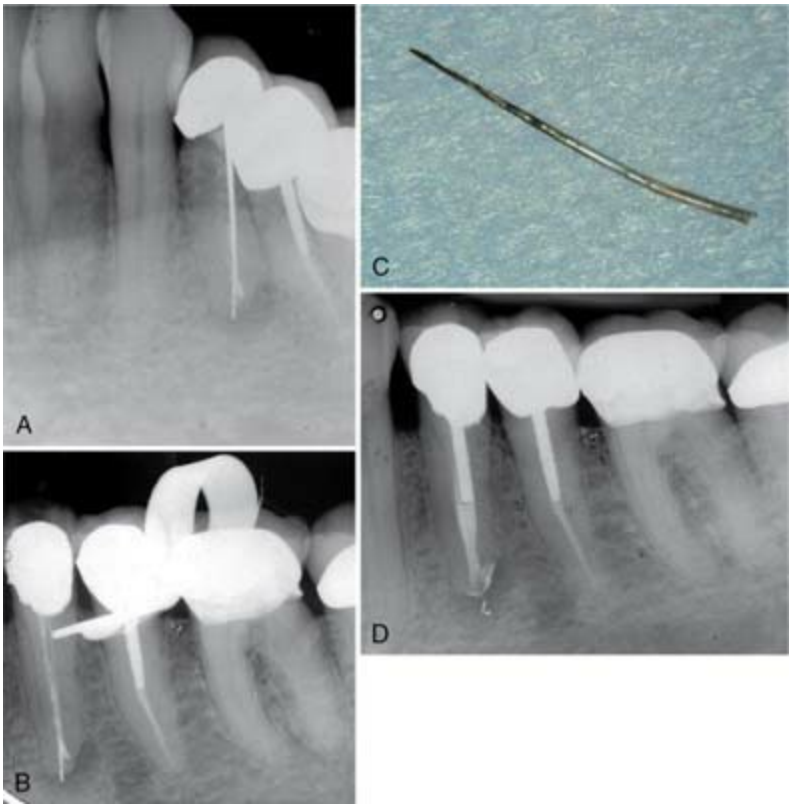

FIG. 10.49 **A,** Persistent disease in a silver point filled tooth. **B,** Silver point removed. Note the radiopaque material in the apical portion of the canal system. This represents corrosion products remaining in the canal and a possible separated apical segment of the cone. **C,** Removed silver point showing black corrosion products adhering to the apical one half. **D,** Crown-down instrumentation prevents extrusion of most of the corrosion products into the periradicular tissues.

A) Radiograph of teeth shows radiopaque crowns and upper canals. Radiopaque outlines of the silver tip inserted in a root canal is visible.

B) Radiograph of teeth shows a tooth with radiopaque crown and upper canal. Radiopaque outlines of file used for the removal are visible.

C) Close-up of a silver point with long pointed tip and black residual corrosive products on the tip surface.

D) Radiograph shows teeth with radiopaque crown and upper canal and radiolucent lower canals.

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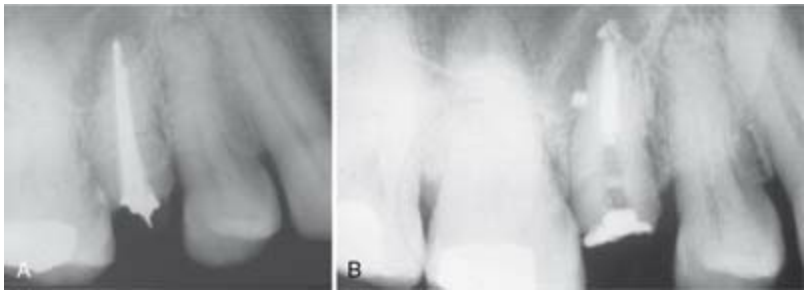

FIG. 10.50 **A,** Preoperative view of a silver point case with persistent disease. Note that the periapical radiolucency extends coronally on the distal aspect of the root end indicating the presence of an unfilled lateral canal. **B,** Postobturation radiograph showing the cleaned and filled distal canal branch.

A) Radiograph of teeth without a crown shows a radiopaque outline of the silver tip inserted in a root canal.

B) Radiograph of teeth without a crown shows a radiopaque outline of the canal branch inserted in the lower canal with a white radiopaque patch near the neck.

Many of the same techniques described for removing separated instruments in the following section apply to the removal of silver points. Silver points have a minimal taper and are smoothed-sided and corrosion may loosen the cone within the preparation. Therefore the clinician should encounter a much easier time removing them than would be the case with separated instruments, which may be mechanically engaged into canals. Silver point canal preparation techniques produced a milled, round preparation in the apical 2 to 3 mm of the canal and, coronal to that, the clinician will frequently find space between the round silver point and the flared canal walls that can usually be negotiated with hand files facilitating point removal.[214]

The first step in the removal of silver points is to establish proper access. Frequently, the coronal portion of the cone is embedded in the core material. This material must be carefully removed with burs and ultrasonics, taking care not to remove any of the silver point within the access cavity preparation. The more of the silver point the clinician has to work with, the more predictable will be its removal. Once proper access is established, the clinician should flood the access preparation with a solvent, such as chloroform, in order to soften or dissolve the cement, enabling easier

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removal. An endodontic explorer or small file may be used to carry the solvent down along the silver point to dissolve as much of the cement as possible. The chamber can be rinsed and dried, and this step may be repeated since fresh solvent enhances the efficiency of cement removal. At this point, the easiest technique, which is also very predictable, is to grasp the exposed end of the silver point with a Stieglitz pliers (Henry Schein, Port Washington, NY) (see Fig. 10.41) or other appropriate forceps, and gently pull it out of the access cavity preparation. If too much extraction force is needed, however, the point may separate so slow force application is advised. The clinician will need a variety of sizes and angles of forceps available to deal with the variety of cases that will need to be treated. Occasionally, the forceps may not get a good purchase on the silver cone and will slip off. In these instances, gripping the cone with the forceps and then gripping the forceps in a hemostat or needle driver to increase the squeezing force of the forceps will allow removal of the cone (Fig. 10.51).[156] If the silver point is held tight by the frictional fit in the preparation, indirect ultrasonics may be employed to loosen it. The silver point is retained in a pair of forceps and ultrasonic energy is applied to the forceps, not the point (Fig. 10.52). This transmits energy down the cone and may loosen it.

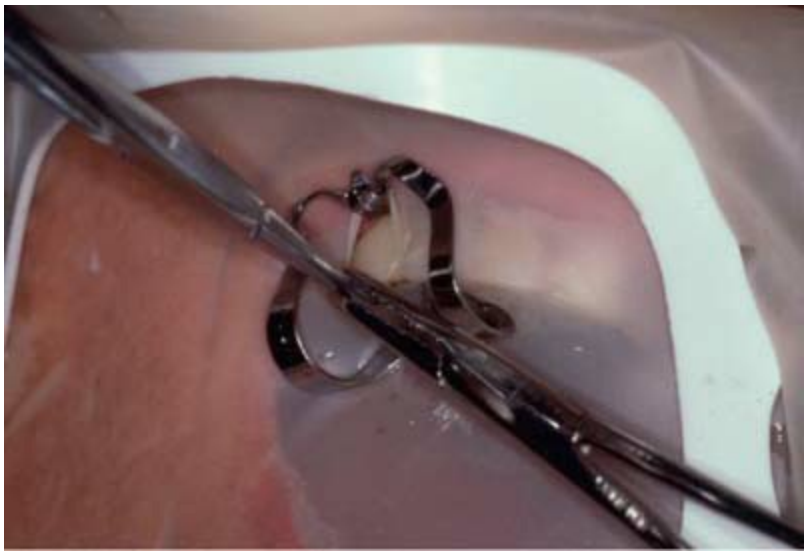

FIG. 10.51 Removal of a highly retentive silver point using a needle driver to squeeze the tips of the Steiglitz forceps. This applies increased gripping force to aid in removal.

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Close-up of an open mouth covered by rubber dam and a clamp shows intrusion of an exposed incisor by ultrasonic tip and forceps.

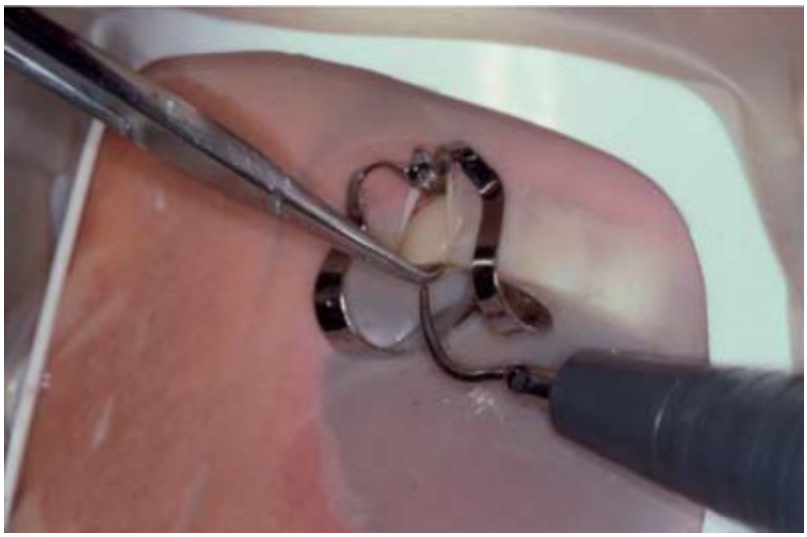

FIG. 10.52 Application of indirect ultrasonic energy to a silver point by placing the ultrasonic tip against forceps that are holding the silver point.

Close-up of an open mouth covered by rubber dam and a clamp exposing the incisor shows removal of silver point by needle device and forceps.

If the silver point cannot be dislodged by the forgoing techniques, the clinician should consider using Hedstrom files to remove the silver point. The Hedstrom file technique requires at least some coronal length of canal space around the silver point to be negotiated first.[155] The sealer is dissolved as previously mentioned, and then files are negotiated as far apically as possibly in two to three areas around the silver point. If only one space can be negotiated, this technique may still be effective. The spaces surrounding the silver point are carefully instrumented to size 15, and then small Hedstrom files are gently screwed in as far as possible apically. They should not be screwed in too tightly to prevent breakage. The flute design of Hedstrom file allows for much better engagement into the silver point compared to other file designs. The files are then twisted together and pulled out through the access (Fig. 10.53). If the first attempt fails, this technique may be repeated, possibly using larger Hedstrom files. If this technique does not completely

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remove the silver point from the canal, it may still be dislodged to the point where it can be grasped by forceps and removed.

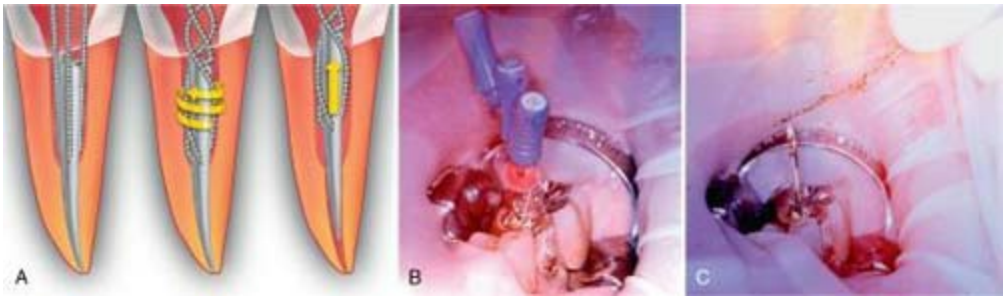

FIG. 10.53 **A,** Diagram illustrating the braiding of Hedstrom files around a silver point. By twisting the braided files, a gripping force is applied which aids in removal of the obstruction. **B,** Small files being braided around a silver point. **C,** Pulling coronally with the braided files removes the silver point.

A) A set of three diagrams of tooth with crown and root canal shows a silver point inserted into the canal. First diagram shows three hedstrom files vertically holding the silver point, the second diagram shows three hedstrom files spirally twisting the silver point marked by a spiral arrow and third diagram shows three hedstrom files extracting the silver point vertically upwards marked by an upward arrow.

B) Close-up of an open mouth covered by rubber dam and a clamp shows set of files removing a silver point. A dental water spray is visible on the mouth surface.

C) Close-up of an open mouth covered by rubber dam and a clamp shows a removed silver point using a set of files. A dental water spray is visible on the mouth surface.

If the clinician needs to expose more of the silver point to enable removal, the use of trephine burs and microtubes or ultrasonics may be necessary.[78] Trephine burs are used in the same manner as described for separated instrument removal; however, the clinician must be more careful when using ultrasonic instruments for the retrieval of silver points. When using ultrasonics for post removal, or separated instrument removal, the tip of the ultrasonic instrument can be placed at the interface between the obstruction and the canal wall. While applying the ultrasonic energy directly to a post or file may prove beneficial in vibrating them loose, silver points are much softer, and if ultrasonic instruments are applied directly to them, the portion

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in contact may be shredded, leaving a smaller segment to work with because elemental silver rapidly erodes during mechanical manipulation.[214] The ultrasonic instrument is used on tooth structure circumferentially around the silver point. This is a very delicate process requiring a microscope or other powerful source of magnification. The energy supplied by the careful use of the ultrasonic instrument can safely expose silver points as well as break up the cement around them.

In many cases, a silver point may have been sectioned deep in the canal to allow post space preparation. In these cases, where the most coronal portion of the silver point is well below the orifice, the use of Gates-Glidden burs to obtain straight-line access to the most coronal extent of the point may be necessary. The burs should be used in a brush-like manner, cutting on the outstroke while applying gentle pressure in an anticurvature direction to decrease the risk of root perforation. Following this, techniques that involve the use of an end-cutting trephine bur to remove tooth structure around the point and then use of an extraction device to remove it may be employed (Fig. 10.54). There are many kits that use these principals with slight variations from one another, including the Masserann Kit (Medidenta International, Woodside, NY) and the Extractor System (Roydent, Troy, MI) (Fig. 10.55). Additional techniques that are effective for removing silver points include the S.I.R. (Separated Instrument Retrieval) System (Vista Dental Products, Racine, WI), the use of a dental injection needle with a 0.14 mm wire, the use of stainless-steel tubing with a Hedstrom file,[214] and the Instrument Removal System (San Diego Swiss, San Diego, CA).

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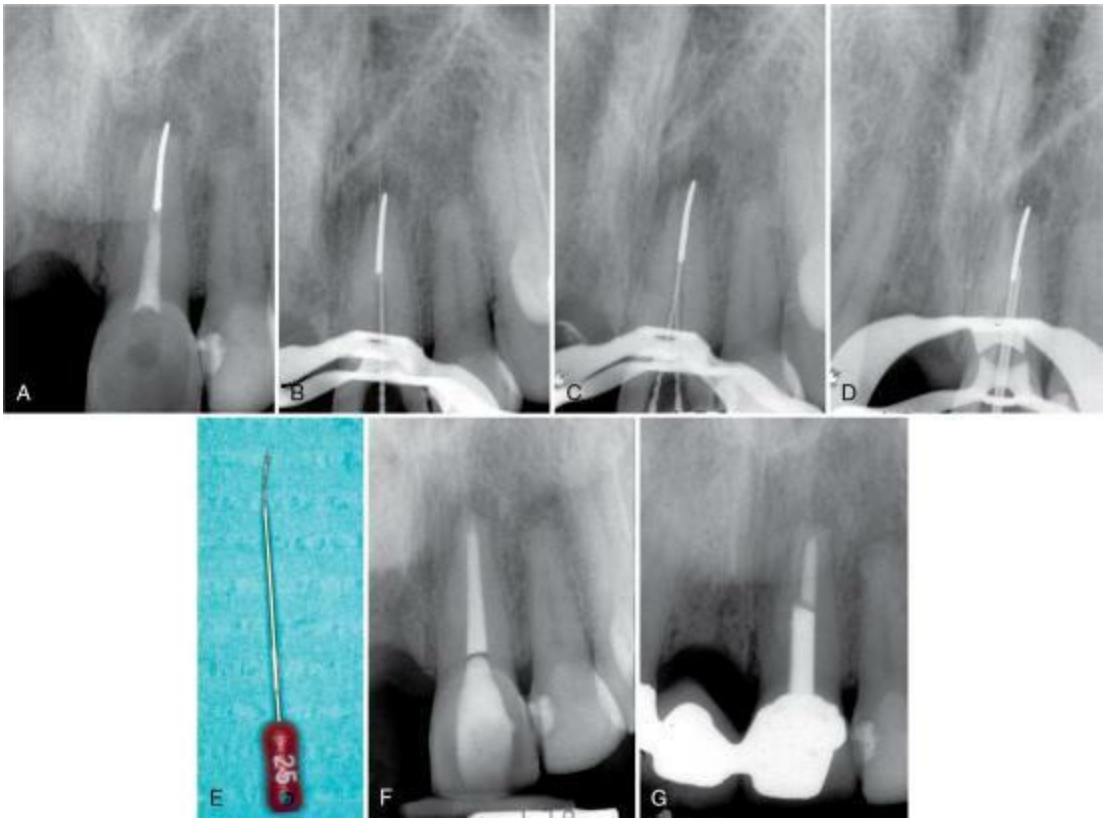

FIG. 10.54 Twist-off silver point case. **A,** Preoperative radiograph showing apical periodontitis and a split silver cone ("twist-off") obturation technique. **B,** The cone was initially bypassed but could not be loosened. **C,** The braided Hedstrom file technique was attempted but was unsuccessful. **D,** A Brasseler Endo Extractor tube is cemented to the cone with cyanoacrylate cement. This instrument is no longer on the market. **E,** The silver point is removed. **F,** Immediate postobturation radiograph. **G,** One-year follow-up showing apical healing.

A) Radiograph of a tooth shows a radiopaque outline of the lower silver tip and radiolucent outline of the upper silver tip.

B) Radiograph of a tooth shows a radiopaque outline of the lower silver tip with a radiolucent outline of the bypassing needle. Radiopaque outline of the dam clamp is visible.

C) Radiograph of a tooth shows a radiopaque outline of the lower silver tip with a radiolucent outline of the braided hedstrom files. Radiopaque outline of the dam clamp is visible.

D) Radiograph of a tooth shows a radiopaque outline of the lower silver tip with a radiolucent outline of the bras-seler endo extractor tube. Radiopaque outline of the dam clamp is visible.

E) Close-up of a silver point removed by extractor tube in number 25.

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F) Radiograph of a tooth shows a radiolucent crown with radiolucent outlines of the core filling material. A small horizontal line is visible under the neck.

G) Radiograph of a tooth shows a radiopaque crown with radiopaque and cylindrical outline in the upper canal. radioluceny is visible in the lower canal.

Source: (Reprinted with permission from Gutmann JL, Lovdahl PE: Problems in the assessment of success and failure, quality assurance, and their integration into endodontic treatment planning. In Gutmann JL, Dumsha TC, Lovdahl PE, Hovland EJ, editors: *Problem solving in endodontics*, ed 3, Mosby, 1997, pp. 180–181.)

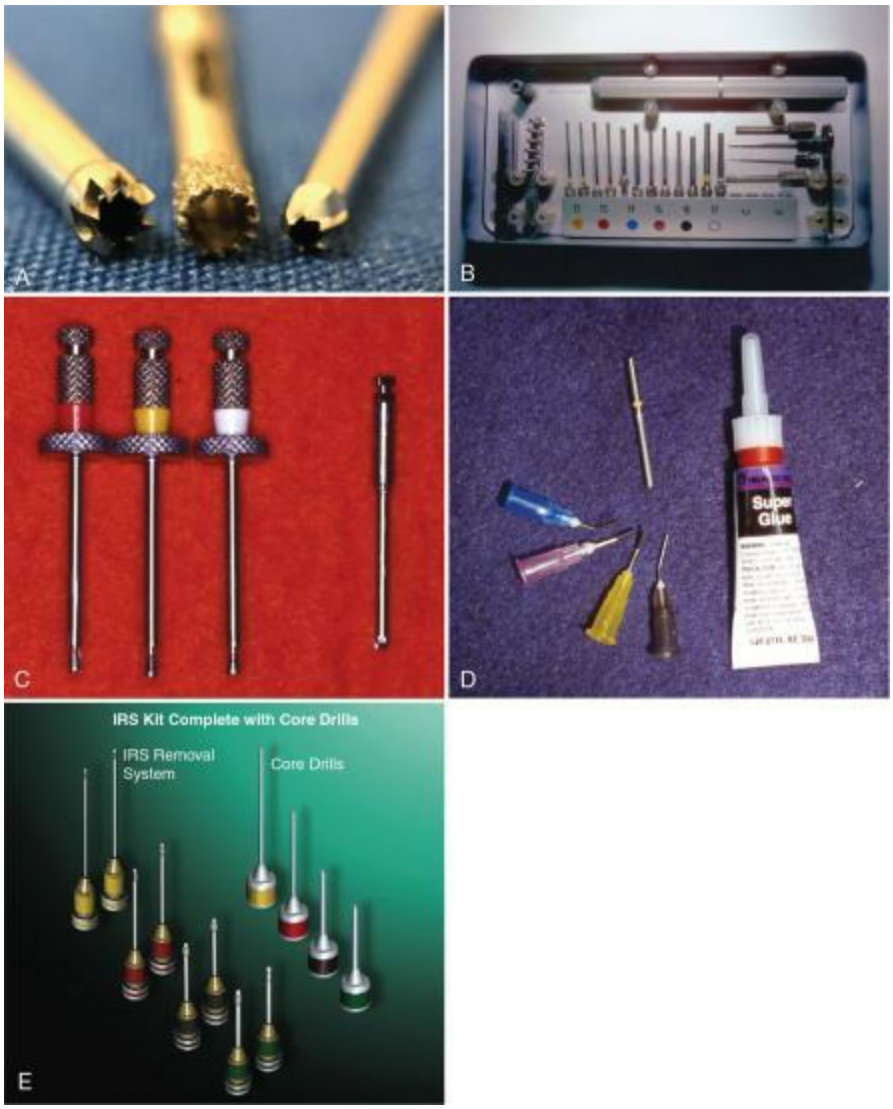

FIG. 10.55 **A,** Meisinger Trephine Burs. **B,** Masserann kit. **C,** Roydent Extractor System. **D,** Separated Instrument Retrieval System (SIR). **E,**

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Instrument Removal System *(IRS)*.

Set of five images show the tools used in the removal of silver points and separated instruments from A through E, where E shows instrument removal system including IRS removal system and core drills.

Source: (**B,** Courtesy Dr. Daniel Erickson.)

These kits are not only effective in the removal of silver points but also in the removal of separated instruments, and because of this common approach to removing both during endodontic retreatment, these techniques will be discussed in detail in the following section on separated instrument removal.

After the silver point is removed, it is very important that subsequent instrumentation procedures be performed in a crown-down manner to minimize extrusion of the silver corrosion products into the periradicular tissues to decrease the occurrence of painful acute flare-ups. This goal is complicated by the fact that ledges are frequently encountered at the level of the apical extent of the silver point due to the type of milled preparation that was frequently used in this technique. Managing ledges will be discussed in a following section of this chapter.

Occasionally, the apical portion of a silver point will separate upon the removal attempt. If it cannot be bypassed or removed, then the case should be completed and followed carefully (Fig. 10.56). Apical surgery or extraction could be necessary in the future (Fig. 10.57).

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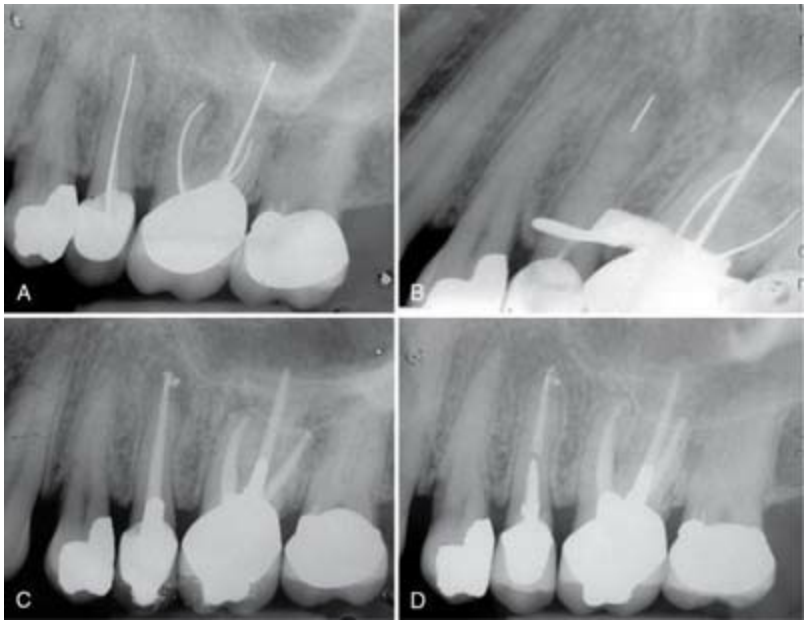

FIG. 10.56 Case illustrating healing despite inability to remove a separated silver point. **A,** Preoperative radiograph showing persistent disease in an upper premolar and molar. **B,** Working film showing the separated cone that could not be retrieved despite extensive clinical efforts. **C,** Final obturation after two appointment procedure using a calcium hydroxide interappointment medicament. **D,** Four-year follow-up showing apical healing.

A) Radiograph of teeth show a radiopaque crowns and radiopaque outline of silver tip in the canals.

B) Radiograph of a tooth shows a radiopaque line in the lower canal. Radiopaque outline of the dam clamp is visible.

C) Radiograph of a tooth shows a radiopaque crown and neck. Radiolucency is visible in the lower root canal.

D) Radiograph of a tooth shows a radiopaque crown and neck with radiopaque spirally textured silver point. Radiolucency is visible in the lower root canal.

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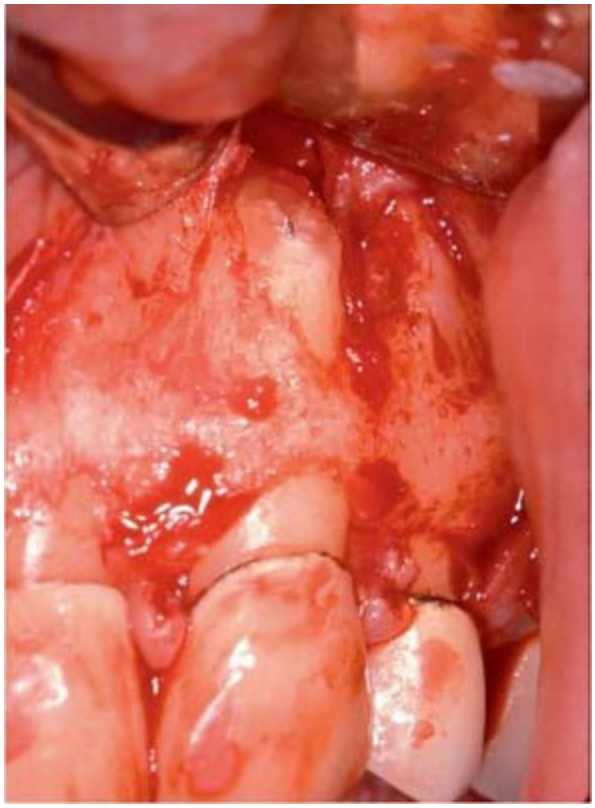

FIG. 10.57 If silver point retreatment is unsuccessful, then apical surgery may be needed. Note the silver point visible on the resected root end. If the point cannot be pulled out in a retrograde direction, then ultrasonic root-end preparation may be complicated by its presence, and root-end preparation using rotary burs may be necessary.

Close-up of the periodontal ligament surface shows wounded apical tissues with white circular patch over the affected tooth.

Removal of separated instruments

Causes of instrument separation

Occasionally during nonsurgical root canal therapy, an instrument will separate in a canal system, blocking access to the apical canal terminus. This instrument is usually some type of file or reamer but can include Gates-Glidden or Peeso Drills, lentulo spiral paste fillers, thermo-mechanical gutta-percha compactors, or the tips of hand instruments, such as explorers or gutta-percha spreaders. During retreatment, it may be obvious after completing the diagnostic phase that there is a separated instrument in the canal system or it may only become apparent after removal of the root filling

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materials (Fig. 10.58). It is therefore useful to expose a check radiograph after removal of the root filling to see if there is any metallic obstruction in the canal. Regardless of which type of instruments the clinician uses, whether stainless steel or nickel-titanium, and how they are used, by hand or engine driven, the potential for separation exists. The incidence of hand instrument separation has been reported to be 0.25%[127] and for rotary instruments, it ranges from 1.68% to 2.4%.[127,300]

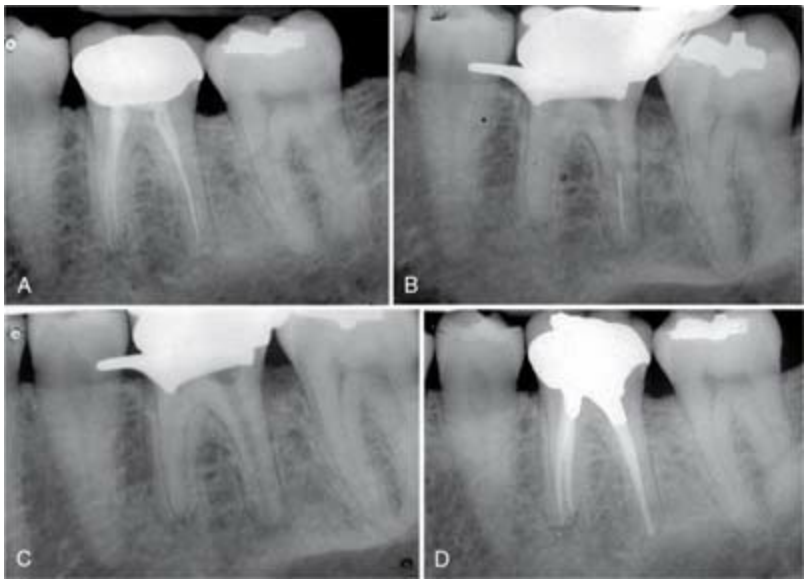

FIG. 10.58 **A,** Preoperative radiograph of a tooth with symptomatic posttreatment disease. **B,** Although not readily apparent on the preoperative film, there is a separated nickel-titanium instrument in the distal canal. **C,** Check film showing that ultrasonics has removed the separated file. **D,** Thirteen-month recall film. The patient was asymptomatic.

A) Radiograph of first molar shows a radiopaque crown and radiolucent root canals. Second molar shows a white radiopaque patch on a part of crown of second molar.

B) Radiograph of a tooth shows a radiopaque crown with a radiolucent outline of nickel-titanium instrument in the right root canal. Radiopaque outline of clamp is visible.

C) Radiograph of a tooth shows a radiopaque crown with a radiolucent pulp.

D) Radiograph of first molar shows a radiopaque patch on the crown, radiopaque neck projecting into two radiopaque upper canals with radiolucent lower canals.

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The most common causes for file separation are improper use, limitations in physical properties, inadequate access, root canal anatomy, and possibly manufacturing defects.

A very common cause for instrument separation is improper use. Included in this are overuse or not discarding an instrument and replacing it with a new one when needed. The following is a list of guidelines for when to discard and replace instruments.[89]

> "1. Flaws, such as shiny areas or unwinding, are detected on the flutes.
> 2. Excessive use has caused instrument bending or crimping (common with smaller-sized instruments). A major concern with nickel-titanium instruments is that they tend to fracture without warning; as a result, constant monitoring of usage is critical.
> 3. Excessive bending or precurving has been necessary.
> 4. Accidental bending occurs during file usage.
> 5. The file kinks instead of curving.
> 6. Corrosion is noted on the instrument.
> 7. Compacting instruments have defective tips or have been excessively heated."[89]

Another type of improper use is to apply too much apical pressure during instrumentation,[277] especially when using engine-driven nickel-titanium files. This pressure can lead to deflection of the instrument within the canal system or increased frictional binding against the canal walls that can overstress the metal, resulting in separation. Regardless of which type of files the clinician uses, they should never use them in a dry canal, as attempting to instrument a dry canal will cause excessive frictional stresses on an instrument.[277] Continual lubrication of the canal with either irrigating solutions or lubricants is required[89] as this will reduce the frictional resistance as well as increase the efficiency of the instrument. All files have flutes that have the ability to build up with dentin shavings, which will decrease the efficiency of the instrument leading to greater frictional forces and ultimately separation. Therefore files should be periodically removed and cleaned during the instrumentation process.

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Inadequate access cavity preparations can lead to many problems, one of which is excessive or unnecessary force applied to the instrument if it is not allowed to enter the canal freely without interference from the access cavity walls. If the file is in contact with the access cavity wall during instrumentation, the chance for separation is greatly increased. Inadequately enlarged access preparations also increase the number and severity of curvatures that the file must negotiate. This underprepared access can lead to the creation of an iatrogenic S curve that can overstress the instrument. This situation is especially hazardous when using rotary instrumentation since traversing an S curve greatly stresses the rotating file, leading to separation (Fig. 10.59).

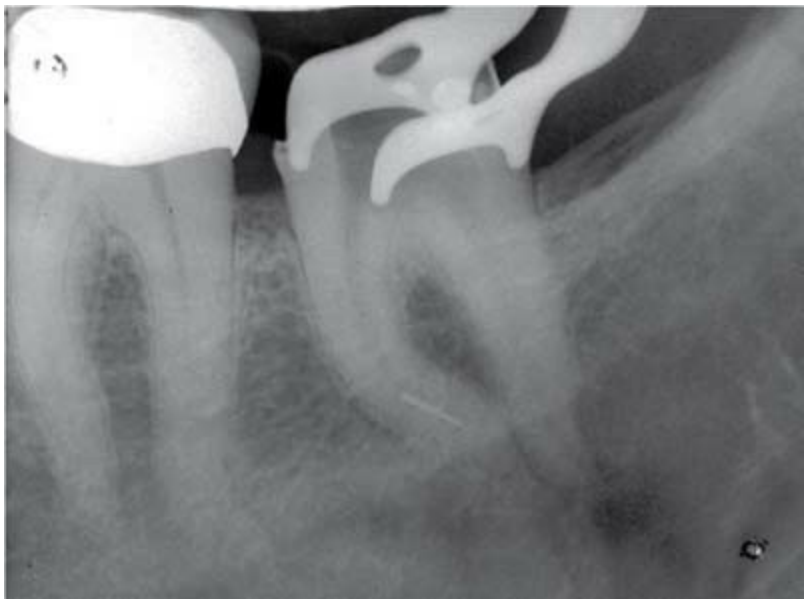

FIG. 10.59 Complicated canal anatomy can increase stress on rotary instruments leading to separation such as in this S-Shaped canal.

Radiograph of first molar shows a radiopaque crown and radiolucent neck and canals. Second molar shows radiolucent outlines of two rotatory tools.

Anatomy, such as abrupt curvatures or anatomic ledges, increases the likelihood of instrument fracture. When the file's progress is hindered, it is quite natural to try to force it further. This approach will rarely result in the file advancing along the naturally occurring path and indeed may result in file separation, perforation, or ledge creation.

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Some clinicians would like to blame instrument separation on manufacturing defects; however, this has never been shown to be of clinical relevance and is quite rare.[277]

The best treatment for the separated instrument is prevention. If proper techniques for cleaning and shaping of the root canal system are followed, file separation should be an infrequent occurrence. Nevertheless, an occasional event may take place. When instrument separation occurs, a radiograph should be taken immediately.[277] This radiograph will not only confirm the separation, it will also give the clinician information that may aid in removal, such as location, size of the file segment, root canal anatomy, and, ultimately, the possibility of removal. The patient should be advised of the accident as well as its effect on the prognosis.[47] In addition, when a file separates, as with other procedural accidents, detailed documentation is necessary for medical-legal considerations,[277] and the remaining segment of the file should not be discarded but rather placed in a coin envelope and kept in the patient record.[47,307]

Prognosis

A separated instrument does not necessarily mean surgery or loss of the tooth. Actually, the prognosis may not be reduced at all depending on what stage of instrumentation the separation occurs, the preoperative status of the pulp and periradicular tissues, and whether or not the file can be removed or bypassed.[252] The presence of a separated instrument in the canal in itself does not predispose the case to posttreatment disease. Rather, it is the presence of any necrotic, infected pulp tissue that remains in the apical canal space that determines the prognosis. The outcome is better if the canal was instrumented to the later stages of preparation when the separation occurs.[277] If the preoperative pulp was vital and noninfected (e.g., irreversible pulpitis), and there was no apical periodontitis, the presence of the separated instrument should not affect the prognosis.[49] If the file can be removed without excessive over enlargement of the canal[82] or causing an additional iatrogenic mishap, such as a perforation, the prognosis will not be affected. Bypassing the instrument and incorporating it into the obturation should also have no effect on the prognosis. However, if the instrument cannot be removed or bypassed in a tooth with a necrotic, infected pulp and apical periodontitis, the

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prognosis will be uncertain. These cases should be followed closely and, if symptoms persist, apical surgery or extraction should be considered.[277,303]

The potential to remove a separated instrument depends on many factors that should be considered during the diagnostic workup. The location of the separated instrument is of critical importance. If the separated instrument extends into the straight, coronal portion of the canal, retrieval is likely. If, however, the instrument has separated deep in the canal and the entire broken segment is apical to the canal curvature, then orthograde removal will not be possible and attempts to do so could lead to a much higher rate of iatrogenic complication.[237,250] If there is persistent disease and the file cannot be bypassed safely, either apical surgery or extraction will be necessary. Because of the need to enlarge the coronal radicular access, root curvatures, external root concavities, and root thickness all will be important factors to consider when deciding which treatment option will provide the best chance of long-term success. Teeth with thin roots and deep external root concavities have a greater likelihood of being perforated during the coronal radicular access, so surgery should be considered as an alternative to orthograde instrument retrieval. The type of material the separated instrument is made of will affect the chances of removal. Nickel-titanium files tend to shatter when ultrasonic energy is applied to them, hindering removal, whereas stainless steel instruments are more robust and more easily removed with ultrasonics.[214]

Removal techniques

Many different instruments and techniques will be discussed in this section, all of which are important to include in the armamentarium for separated instrument removal. None, however, is more important than the operating microscope (Fig. 10.60). This instrument will not only increase visibility using magnification and light, but it will also increase the efficiency and safety of almost all the techniques to be discussed. The use of a headlamp and magnifying loupes will help with the removal of many canal impediments. However, the use of the operating microscope has caused a quantum leap in visualization due to the enhanced lighting and magnification that it offers,[138] and many of the techniques to be described should not even be attempted without the use of this valuable tool.[262]

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FIG. 10.60 The surgical operating microscope is not only invaluable in helping to remove separated instruments; it is in fact a necessary tool for these procedures.

A surgical operating microscope shows the supporting structure, body of the microscope and the light source.

Once the patient has been advised of the treatment options and the decision has been made to attempt removal, the clinician's first choice in treatment will be based on the location of the instrument. If the file is clinically visible in the coronal access and can be grasped with an instrument, such as a hemostat or Stieglitz Pliers (Sullivan-Schein, Port Washington, NY) (see Fig. 10.41), then these should be used to obtain a firm hold of the file and extract it out through the access cavity preparation. Many different sizes and angles of forceps are available, and almost all are necessary in order to have the ability to remove obstructions from the many different angles and levels of accessibility presented to the clinician. These will work well if the object is loose fitting within the canal and if the clinician has good access. However, establishing a firm purchase can sometimes be difficult without removing excessive tooth structure. Once a purchase onto the file has been achieved, it is best to pull it from the canal with a slight counterclockwise action. This action will unscrew the flutes that are engaged in the dentin as the file is being removed. This is the easiest technique for removal of a separated file; however, unfortunately, many files separate at a point where these forceps cannot be used.

Frequently, a file will separate at a point deeper in the canal where

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visibility is difficult. In order to remove separated root canal instruments predictably, the clinician must create straight-line coronal radicular access. Either removing the crown or creating a large access cavity preparation establishes adequate coronal access to allow the use of the appropriate instruments. Straight-line radicular access can be created with the use of modified Gates-Glidden drills. These drills may be ground down or sectioned with a bur at their maximum cross-sectional diameter. This process will create a circumferential staging platform to facilitate ultrasonic use (Fig. 10.61).[214] A recent study showed that use of a similarly modified Lightspeed nickel-titanium rotary instruments (Lightspeed Technology, Inc., San Antonio, TX) created a staging platform that was more centered in curved canals than the Gates-Glidden drills.[128]

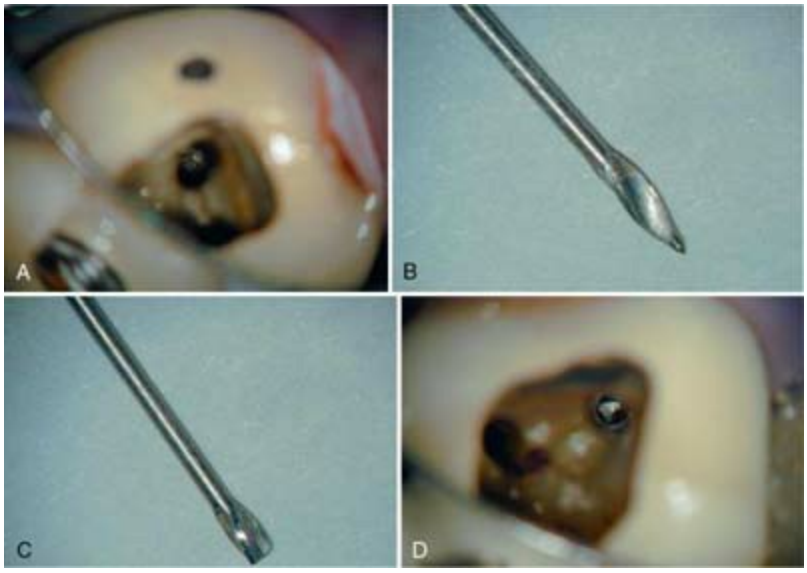

FIG. 10.61 **A,** Separated instrument in the mesiobuccal canal of a molar. **B,** Unmodified Gates-Glidden drill. **C,** Modified instrument. The tip has been ground off to the maximum diameter of the cutting head. **D,** Staging platform created in the straight coronal section of the canal. Note the enhanced visibility and the triangular cross section of this rotary instrument.

A) Close-up view of separated instrument and molar with open crown shows dentin exposing a black dot inside the cavity and another black dot over the tooth surface.

B) Close-up of a gates-glidden drill with a spirally pointed tip.

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C) Close-up of a gates-glidden drill with a spirally flat tip.

D) Close-up blur view of separated instrument and molar with open crown shows dentin exposing black dots inside the cavity.

Ultrasonic instruments have been shown to be very effective for the removal of canal obstructions.[42,180,214] The ultrasonic tip is placed on the staging platform between the exposed end of the file and the canal wall and is vibrated around the obstruction in a counterclockwise direction that applies an unscrewing force to the file as it is being vibrated. This technique will help with removing instruments that have a clockwise cutting action. If the file had a counterclockwise cutting action (such as the hand GT files), then a clockwise rotation will be needed. The energy applied will aid in loosening the file, and occasionally the file will appear to jump out of the canal. It is prudent to cover the orifices of the adjacent open canals with cotton or paper points to prevent the removed file fragment from falling into them, causing further case complications (Fig. 10.62, *A*).[214] There are many different sizes and angles of ultrasonic tips available for this purpose but, in general, the deeper in the canal the obstruction is, the longer and thinner an ultrasonic tip must be. It should be remembered that long, thin tips must be used on very low power settings to prevent tip breakage (see Fig. 10.62, *B*). Occasionally, if the separated instrument can be bypassed, the use of ultrasonic files can loosen it. Care must be taken, however, to avoid ultrasonic file separation or root perforation.[115] As mentioned previously, nickel-titanium instruments often break up into fragments when subject to the energy supplied by an ultrasonic instrument (Fig. 10.63). The clinician may be tempted to use this information to their advantage by applying the tip of the ultrasonic directly onto nickel-titanium files. Occasionally, this method may work; however, the chance of pushing the separated file further into the canal or beyond the apical foramen may increase the risk of this technique.

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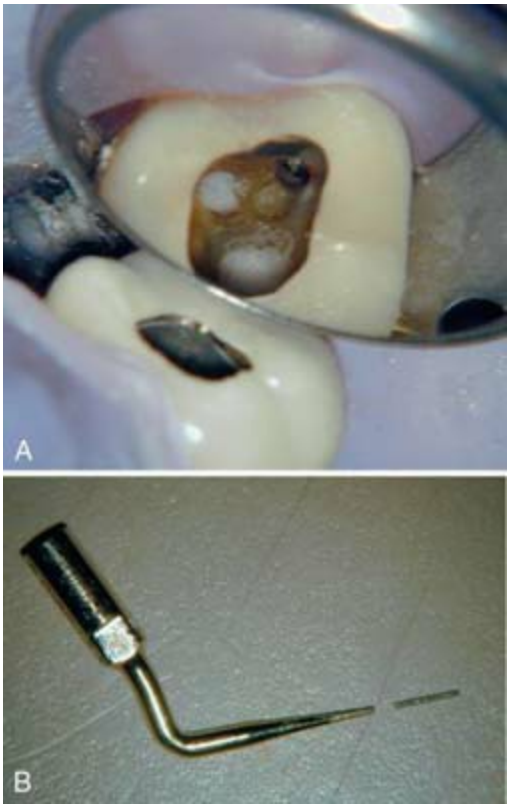

FIG. 10.62 **A,** Cotton pellets protecting the orifices of the other canals when ultrasonic obstruction removal is needed. **B,** Ultrasonic tip separated during excavation around separated instrument. If the adjacent canals were not protected, further unnecessary complication could result.

A) Close-up sagittal view of tooth with an opening and the mouth mirror reflecting a virtual image of open crown exposing black dots and cotton pellets.

B) Close-up of a curved dental file with broken ultrasonic tip.

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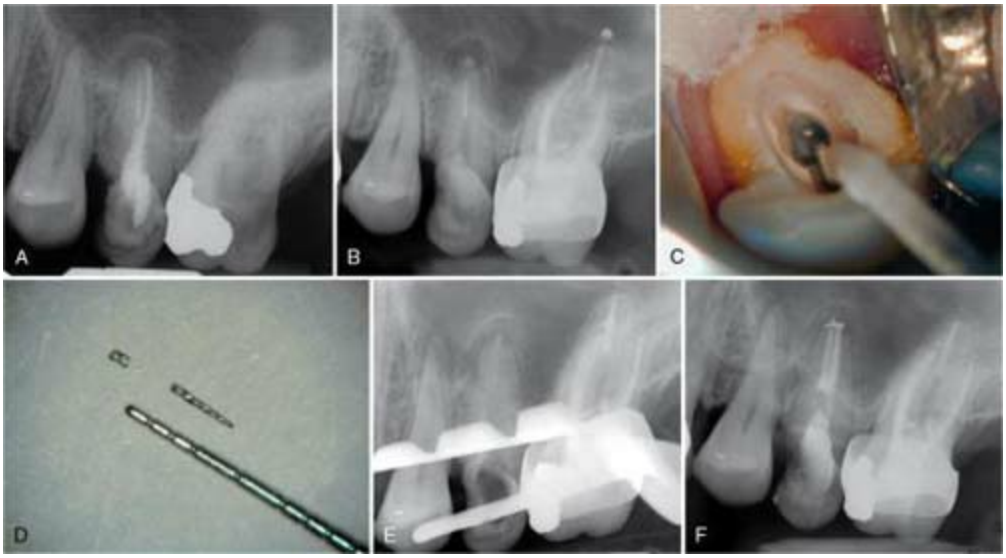

FIG. 10.63 **A,** Preoperative radiograph showing a separated file in the palatal canal, potential coronal leakage, and apical periodontitis. **B,** Check film showing the separated instrument after gutta-percha removal. **C,** Photograph showing the separated instrument in the palatal canal and a paper point in the buccal canal to protect it. **D,** Separated nickel-titanium file removed. Note that it is in two pieces; a result typical when applying ultrasonic energy to nickel-titanium. **E,** Check film showing that the file has been completely removed. **F,** Final canal obturation.

A) Radiograph of teeth shows the first tooth with radiolucent crown, radiopaque neck and radiopaque canals. Second tooth shows a white radiopaque patch on a part of crown.

B) Radiograph of teeth shows the first tooth with radiolucent crown, neck and canals with a radiopaque vertical line depicting a nickel-titanium file in the lower canal. Second tooth shows a radiopaque crown and canals with a white radiopaque patch on a part of crown.

C) Close-up view of an examining tool and a molar with open crown shows separated instrument, exposing circular black spot inside the cavity.

D) Close-up view of the laterally separated textured nickel-titanium file.

E) Radiograph of teeth shows the tooth with dark black patch on the crown, with radiolucent neck and canal. A radiopaque outline of the separated textured nickel-titanium file is visible.

F) Radiograph of teeth shows tooth with radiolucent crown, radiopaque neck and radiopaque lower canal.

If the direct application of ultrasonic energy does not loosen the separated

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instrument sufficiently to remove it, the fragment must be grabbed and retrieved. This is accomplished with a variety of techniques, most using some variant of a microtube. The staging platform is further reduced by ultrasonics until enough of the separated instrument is exposed to retrieve (about 2 to 3 mm).[214] This reduction must be done carefully to avoid root perforation. One relatively simple microtube technique is to use a short piece of stainless-steel tubing that is pushed over the exposed end of the object. A small Hedstrom file is then pushed between the tube and the end of the object using a clockwise turning motion that produces a good mechanical lock between the separated instrument, the tube, and the Hedstrom file. The three connected objects can then be removed by pulling them in a coronal direction (Fig. 10.64).[261]

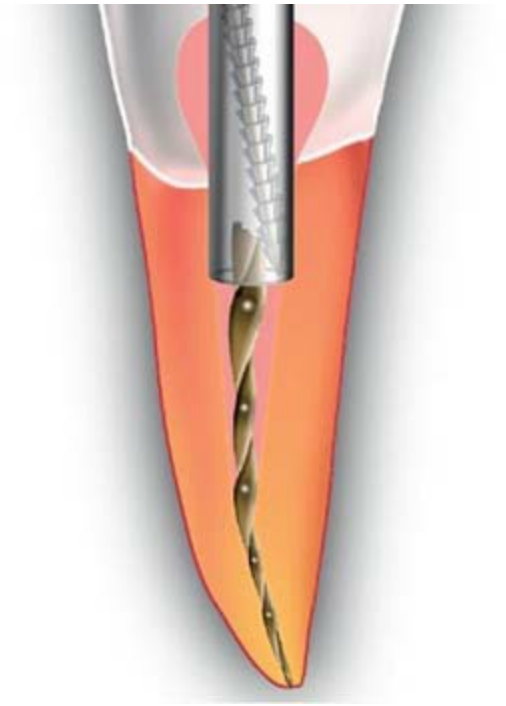

FIG. 10.64 Tube and Hedstrom file removal technique. The tube is slipped over the obstruction and a Hedstrom file is gently screwed into the space between the tube and the obstruction. Pulling the tube and Hedstrom file together can withdraw the obstruction.

A diagram of tooth with crown and root canal shows a tube with a spirally textured hedstrom file. File extracts the spirally textured pointed obstruction vertically upwards.

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Source: (Courtesy Dentsply Endodontics.)

Another technique is to use a 25-gauge dental injection needle along with a 0.14 mm diameter steel ligature wire. The needle is cut to remove the beveled end as well as the opposite end so it no longer extends beyond the hub. Both ends of the wire are then passed through the needle from the injection end until they slide out of the hub end creating a wire loop that extends from the injection end of the needle. Once the loop has passed around the object to be retrieved, a small hemostat is used to pull the wire loop up and tighten it around the obstruction and then the complete assembly is withdrawn from the canal.[211] Occasionally, a larger diameter tube and thinner 0.11 mm ligature wire will facilitate assembly of this extractor (Fig. 10.65).

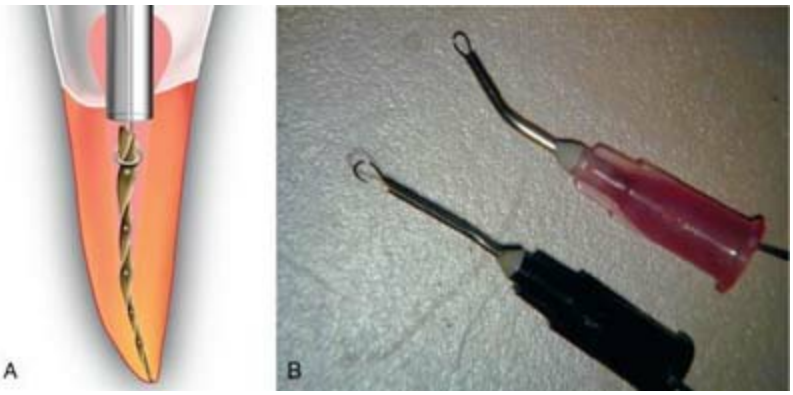

FIG. 10.65 **A,** Diagram illustrating the wire loop and tube method of obstruction removal. The wire loop is carefully placed around the obstruction, tightened, and then removed. **B,** Larger diameter tubes and smaller diameter 0.11 mm ligature wire enhances the efficiency of this technique.

A) A diagram of tooth with crown and root canal shows a tube with a loop headed wire grasping the spirally textured pointed obstruction and extracting it vertically upwards.

B) Close-up of two wire loops with different loop diameters.

Source: (**A,** Courtesy Dentsply Endodontics.)

Another effective technique, especially in cases where access or obtaining an adequate purchase on the file is difficult, is to use an end-cutting trephine bur to remove tooth structure around the file and then use an extraction

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device to remove it. There are many kits that use these principals with slight variations from one another, including the Meisinger Trephine Burs, the Masserann Kit (Medidenta International, Inc., Woodside, NY), and the Extractor System (Roydent Dental Products, Troy, MI) (see Fig. 10.55).

Tube and glue techniques use a cyanoacrylate adhesive, which bonds a hollow tube to the exposed end of the file for removal. This technique requires the use of hollow trephine burs followed by tube extractors with the adhesive inside the tip. The most important factor in using this technique is the snugness of fit between the extractor tube and the obstruction. It has been shown that, even with only 1 mm of overlap between the extractor tube and the obstruction, if there is a snug fit, the bond created with the cyanoacrylate may be strong enough to remove many obstructions. However, the recommended amount of overlap between the tube and the obstruction is 2 mm. The time needed for the adhesive to set to ensure adequate bond strength for removal is 5 minutes for a snug fit and 10 minutes for a loose fit.[86,253] One disadvantage of this technique is that the trephine burs may be much larger than the coronal end of the instrument to be removed, so the use of the smallest trephine burs that can expose the coronal end of the separated instrument is suggested. Smaller trephine burs correspond better with smaller extraction tubes and remove less dentin that can decrease the likelihood of weakening the root leading to fracture.[70] Another disadvantage is that the burs cut very aggressively when new but dull rather quickly. When new, this aggressive cutting may lead to perforation or even separation of the obstruction. Therefore great care is needed when using this technique (Fig. 10.66). Once the separated instrument has been removed, the extraction tubes may be reused, either by using acetone to remove the embedded instrument from the extractor tube or by simply cutting the extraction tube with a bur beyond the extent that the separated instrument has penetrated.

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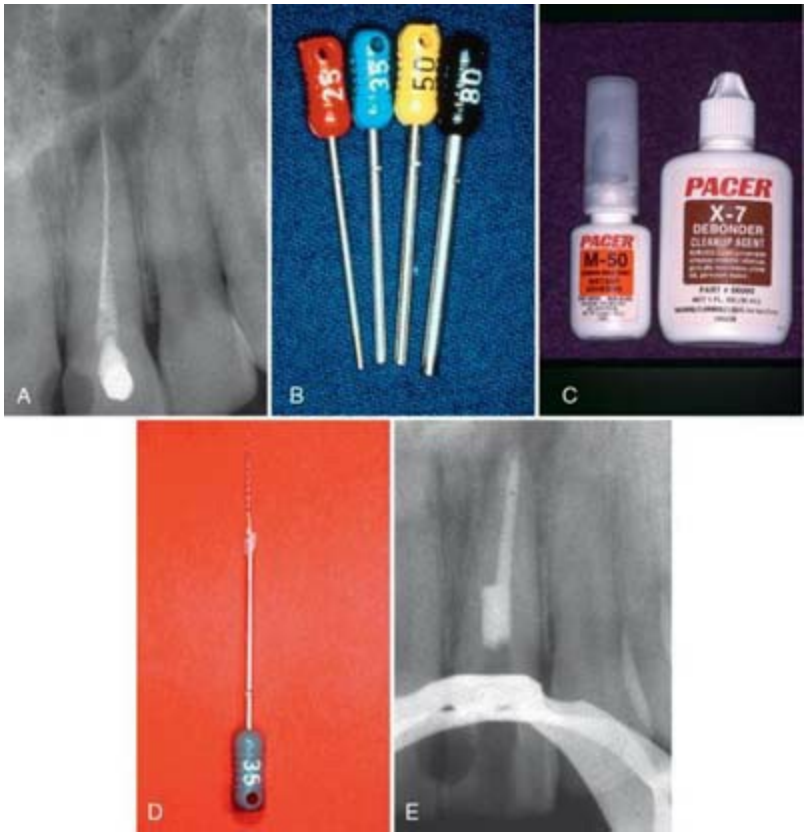

FIG. 10.66 **A,** Separated file wedged into an upper incisor. **B,** Brasseler Endo Extractor tubes (no longer available). **C,** Cyanoacrylate cement and de-bonding agent. **D,** Separated file pulled out by the bonded tube. **E,** Final obturation. Note the excessive amount of tooth structure removal by the trephine bur that was needed to bond the tube.

A) Radiograph of tooth shows the radiopaque outline of a separated file with broader base and pointed tip.

B) Close of basseler endo extractor tubes in sizes 25, 35, 50 and 80.

C) Close-up of two bottles show an instant adhesive and a debonder clean up agent.

D) Close-up of a spirally textured file extracted by a tube in size 35.

E) Radiograph of tooth shows a radiopaque neck and canal. A radiopaque outline of dam clamp is visible.

The Masserann technique has also been recommended for the removal of separated instruments.[168] This technique is similar to the Endo Extractor in that it uses trephine burs and a specific extraction device. This kit comes with

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a very convenient gauge that aids in predicting the size of the bur and the extractor to be used, and it contains many different sizes of trephine burs. In addition, the trephine burs with this kit cut in a counterclockwise direction that provides an unscrewing force on separated files. The extraction mandrels have an internal stylus that wedges the file against the internal wall of the mandrel allowing the obstruction to be removed. While effective, this technique may require removal of an excessive amount of radicular dentin[78] leading to root weakening and the risk of perforation[305]; therefore this instrument must be used with caution.

The Extractor System from Roydent comes with only one bur and three extraction devices. The bur is very conservative and removes a minimal amount of tooth structure, enabling access to the obstruction. The extractor tubes are also quite small and, therefore, will only work for the removal of small obstructions. The extractor surrounds the obstruction with six prongs that can be tightened onto the object, enabling removal. This works in the same way a drill chuck tightens onto a drill bit (Fig. 10.67). The disadvantages of this kit are the lack of variety of instruments, the possibility of separating the obstruction with the bur, and the potential problem of breakage of the prongs in the extractor if they are submitted to bending rather than applying strict tensile force during removal.

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FIG. 10.67 Close-up view of the Roydent Extractor tip. The tip is placed over the separated instrument and tightened to grasp the obstruction.

A Roydent extractor tip with a cylindrical body and a small screw wedge at the end, kept on a platform.

Another device designed specifically for the purpose of separated file removal is the Instrument Removal System (San Diego Swiss, San Diego, CA) (see Fig. 10.55, *E*). This kit consists of two different sizes of extraction devices that are tubes with a 45-degree bevel on the end and a side cutout window. Each tube has a corresponding internal stylus or screw wedge. Prior to usage of this instrument, 2 to 3 mm of the obstruction are exposed by troughing around it with an ultrasonic instrument. Once the file is exposed, the appropriate size microtube is selected and slid into place over the obstruction. Once in place, the screw wedge is turned counterclockwise to engage and displace the head of the obstruction through the side window. The assembly is then removed.[214] This instrument is very useful in the straight portion of the canal, but it is difficult to force large diameter separated files through the cutout window, hampering their removal (Fig. 10.68).

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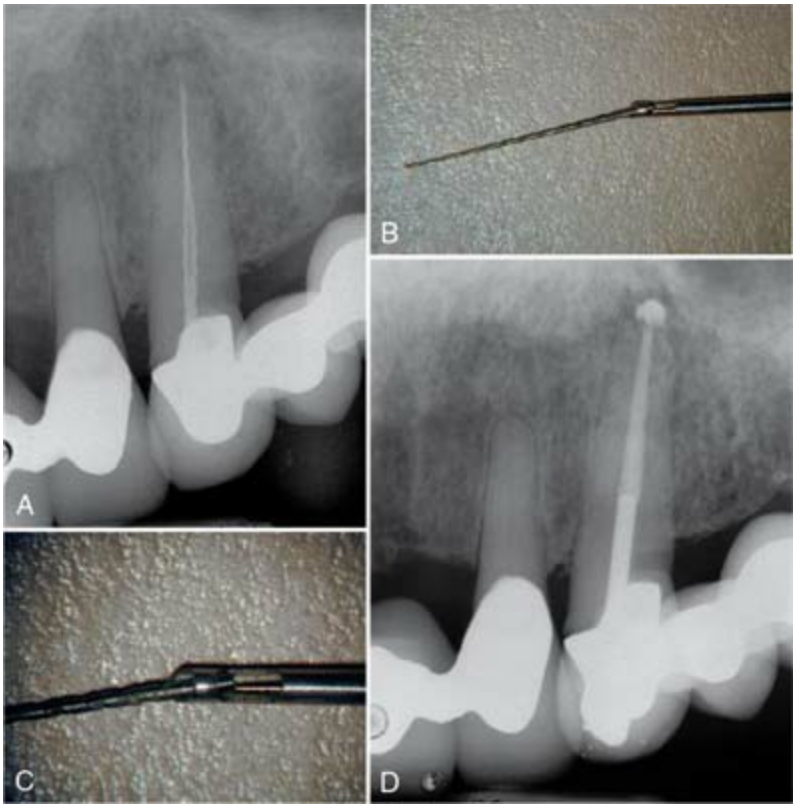

FIG. 10.68 **A,** Preoperative radiograph showing two separated instruments in one tooth. **B,** IRS instrument with a removed file. **C,** Note that large size files are difficult to push through the cutout window. **D,** Postoperative radiograph.

A) Radiograph of tooth shows a radiopaque crown with a radiopaque outline of a spirally textured file in the root canal.

B) Close-up of an IRS tube with removed spirally textured file.

C) Close-up of a part of spirally textured file grasped in a tube.

D) Radiograph of tooth shows a radiopaque crown and upper canal and radiolucent lower canal with a dot like attachment.

The S.I.R. (Separated Instrument Retrieval) System (Vista Dental Products) (see Fig. 10.55, *D*) is another microtube method of separated instrument retrieval. It utilizes extractor tubes bonded onto an obstruction, enabling removal. Once the obstruction is exposed using ultrasonics or the trephine burs from one of the other kits described, the bendable dead-soft tubes are bonded onto it. Once the adhesive is set, the obstruction is removed through the access cavity preparation. Included in this kit are the necessary bonding agent, a bottle of accelerator, five different sizes of tubes, assorted

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fulcrum props, and a hemostat. The accelerator causes the bonding agent to set almost instantaneously. The ability to bend these tubes allows for access in most areas of the mouth. A hemostat allows the clinician to establish a firm purchase onto the tube, creating the ability to lever the bonded obstruction out of the canal. A vinyl, autoclavable instrument prop provides protection for the next most anterior tooth, which is to be used as a fulcrum; however, if the clinician has access to grasp the extractor with the fingers to remove the extractor/obstruction unit, the hemostat may not be necessary.

There is now a commercially available kit containing multiple instruments that allow the clinician to use most of the aforementioned techniques. It is the Terauchi File Retrieval Kit (Dental Engineering Laboratories, Santa Barbara, CA) (Fig. 10.69). This kit contains modified Gates Glidden burs to prepare a staging platform, specialized ultrasonic tips for use deep in the canal space, and a tube and loop instrument to grasp the coronal extant of the separated instrument when it has been exposed. The kit is very effective for removing separated instruments and also contains a barbed tip gutta-percha removal instrument that efficiently removes fragments of gutta-percha that adhere to canal walls in the coronal portion of the canal.

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FIG. 10.69 The Terauchi File Retrieval Kit. This kit was designed to distill down all the techniques mentioned in the text into a simple to use system for separated file removal.

A Terauchi file retrieval kit. The components include modified Gates Glidden burs, ultrasonic tips, a tube and barbed tip instrument for gutta percha removal.

Another interesting recent development is the use of the GentleWave (Sonendo, Inc., Laguna Hills, CA) canal irrigation system to remove separated instruments. This device creates multisonic energy in the canal system while dispensing irrigation solutions and has been advocated for canal cleaning and disinfection. The powerful vortices produced in the solutions within the canal system can dislodge and remove separated instruments (Fig. 10.70).[297] Until more science is developed regarding this particular use for this very interesting technology, including possible complications and parameters for implementation, caution should be exercised in attempting to remove separated instruments by this method.

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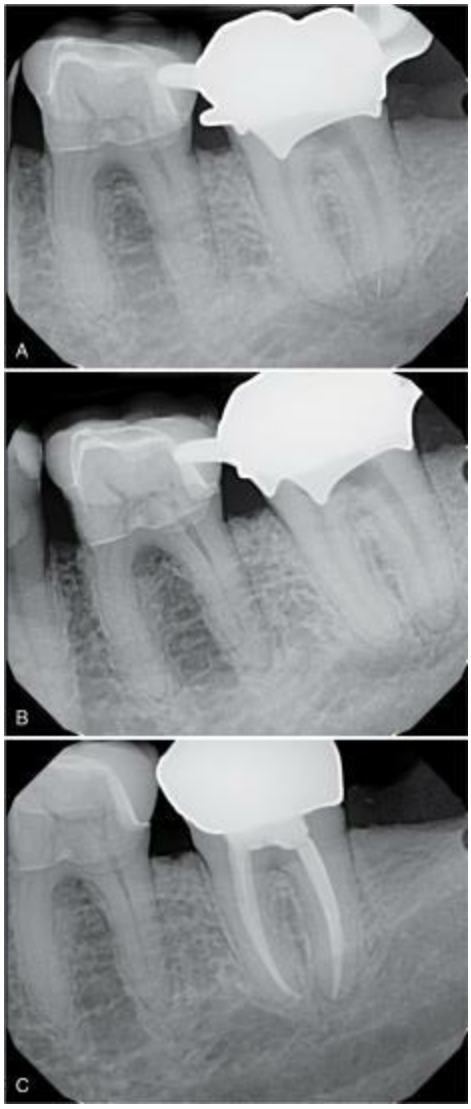

FIG. 10.70 Separated file removal using the GentleWave System. **A,** A small fragment of file is lodged at the apical extent of the canal. **B,** After one irrigation cycle of the GentleWave device, a check radiograph shows that the separated instrument has been removed. **C,** Postobturation image.

A) Radiograph of tooth shows a radiopaque crown with irregularly spread gray patch on the crown. The radiopaque patch projects into another tooth.

B) Radiograph of tooth shows a radiopaque crown with irregular gray patch on the crown. The radiopaque patch projects into another tooth.

C) Radiograph of tooth shows a radiopaque crown with regular gray patch on the crown with radiolucent neck and canals.

Source: (Courtesy Dr. Kevin Axx.)

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Heat generation during retreatment procedures

There are many procedures in endodontic therapy that can generate heat, but perhaps the area with the greatest risk of heat-related tissue damage occurs during nonsurgical retreatment. Use of heat to soften canal filling materials to aid in their removal[146,154] and use of ultrasonics to dislodge posts[220] and separated instruments[106,161] can potentially generate enough heat to raise the temperature of the external root surface by 10°C or more. Temperature elevations of the periodontal ligament in excess of 10°C can cause damage to the attachment apparatus.[13,62,222,223]

The greatest danger of heat-related damage occurs with the use of ultrasonic energy to dislodge foreign objects in the canal space in order to gain access to the apical portion. As has been described earlier, this has recently become an extremely important and useful part of the clinician's armamentarium due to the ability of ultrasonic energy application to conserve tooth structure while removing the obstruction. These instruments have allowed clinicians to perform predictable retreatment when surgery would have been previously indicated. As with most instruments used in dentistry, however, these devices must be used with caution as in vitro research and clinical case reports imply that they have the potential to damage the tooth attachment apparatus due to the heat generated during usage.[34,88,229] One in vitro study has showed that ultrasonic vibration for post removal without coolant can cause root surface temperature increases approaching 10°C in as little as 15 seconds.[55] Thermal damage to the periradicular tissues may be so serious as to result in both tooth loss and permanent bone loss (see Fig. 10.29). This is not to say that ultrasound energy should be avoided for the removal of canal obstructions since it is many times the only way to reach the apical area of the canal.

The factors that may contribute to a heat-induced injury are the length of the post, post diameter, post material, and type of luting cement. Studies need to be done in order to establish heat reduction protocols based on these variables. Some have proposed that the dentin thickness between the outer surface of the post and the root surface may affect root surface temperature rise[91,92] and one study showed this.[220] However, a more recent study has shown that the dentin thickness is statistically insignificant as a factor in root surface temperature rise.[114] One possible mitigating factor would be the

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effect of the periradicular blood supply, which could act as a heat sink dissipating the generated thermal energy and thus helping to prevent injury. This may be why the effect of seemingly similar conditions of ultrasonic application can have such differing results on different patients. Clearly, more in vivo research is needed in this area.

It has become accepted that the heat-induced damage to periradicular tissues during the usage of ultrasound energy for post removal is time dependent.[34,119] Recent studies have advised cooling of the ultrasonic tips can greatly reduce heat buildup[34,119] despite reducing the efficiency of debonding resin bonded posts.[55,83] The amount of time the clinician can use these instruments safely is very difficult to determine as specific protocols that are evidence based using in vivo research have yet to be established. Therefore, the authors feel any specific recommendations regarding rest intervals between usage of ultrasound energy, ways of monitoring heat buildup, or duration of consistent activation cannot be made based on the available research at the time of publication. However, the authors do feel strongly with regards to several recommendations for the use of ultrasound energy during the removal of canal obstructions, and they are as follows:

- Use ultrasonic tips with water ports whenever possible.
- If your ultrasound device does not have tips with water ports, have your assistant use a continuous coolant air/water spray during usage.[64]
- Take frequent breaks in order to let the tooth cool down.
- Avoid using the ultrasound on the high-power setting.[64]

Prudent of clinicians must take extreme care when applying ultrasound energy to a canal obstruction as it has been shown that even with the use of water coolant, root surfaces can increase in temperature rapidly.[55,220] Thus until evidence-based heat reduction protocols are developed, caution when using instruments that can generate heat in the periodontal ligament is required.

Management of canal impediments

Following removal of all root-filling materials, further progress to the apical

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constriction may be prevented by the presence of a block or a ledge in the apical portion of the canal. Most of these impediments are iatrogenic mishaps resulting from vigorous instrumentation short of the appropriate working length and failure to confirm apical patency regularly during instrumentation. A blocked canal contains residual pulp tissue (sometimes necrotic, often fibrosed or calcified) and packed dentinal “mud” in the apical several millimeters of the canal system.[214] This debris is frequently infected resulting in persistent disease and must be removed if possible. A ledge is the result of placing non-precurved, end-cutting instruments into curved canals and filing with too much apical pressure.[89,129] It is a type of canal transportation that results in a canal irregularity on the outside of the canal curvature that is difficult or impossible to bypass. The canal space apical to the ledge is not thoroughly cleaned and sealed, so ledges frequently result in posttreatment disease. The best treatment for blocked and ledged canals, as with all iatrogenic problems, is prevention. If the clinician is careful and attentive during the instrumentation process, the chance for an impediment to develop is minimized. When the clinician becomes careless or hurried, problems occur. The strategies for the prevention of blocks and ledges are found in another chapter of this text.

During the treatment-planning phase, blocks and ledges may be detectible on radiographs as a root filling short of the ideal working length, and the patient should be warned that they might prove impenetrable and require future apical surgery or extraction.[77] This should not deter the clinician from choosing nonsurgical retreatment, however. In a recent study, 74% of teeth showing short root fillings were successfully negotiated to adequate length with the authors stating that presence of a short fill should not be considered a technical contraindication to retreatment.[66] The clinical encounter usually occurs after removal of the previous root-filling material when apical advancement of small files is impeded. At this point, the clinician may be unaware of which type of impediment exists, but a common approach to management is helpful in the early stages of the process. The coronal portion of the canal should be enlarged to enhance tactile sensation and remove cervical and middle third obstructions in the canal space. The canal should be flooded with irrigant, and instrumentation to the level of the impediment should be accomplished using non-end cutting rotary files, such as the

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Lightspeed (Lightspeed Endodontics, San Antonio, TX), the Profile or GT instruments (Dentsply, York, PA), or the K-3 instrument (Sybron Endodontics, Orange, CA), in a crown-down manner. This procedure will enlarge and flare the canal space coronal to the impediment while minimizing the likelihood of worsening any ledge present.

At this point, the impediment should be gently probed with a precurved #8 or #10 file to determine if there are any "sticky" spots that could be the entrance to a blocked canal. A directional rubber stop should be used so that the clinician knows in which direction the tip of the instrument is pointing, which helps in visualizing the 3D layout of the canal system. Frequently, evacuating the irrigant and using a lubricant, such as RC Prep (Premier, Plymouth Meeting, PA) or Pro-Lube (Dentsply, York, PA), will enhance the ability to place the small file into the apical canal segment. If repeated, gentle apical pressure or "pecking" of the hand file against the blockage results in some resistance when withdrawing the instrument on the outstroke ("stickiness"), then the clinician should continue to peck at the "sticky" spot until further apical advancement is accomplished.[214] This is frequently a slow and tedious process. The endeavor can be made more efficient by using precurved stiff files such as the C+ file (Maillefer, Baillagues, Switzerland), but there is a risk of deviating from the original canal path, creating a ledge and, ultimately, a false canal leading to zip perforation.[89] It is prudent to make a working radiograph when some apical progress has been made to confirm the placement of the instrument into the suspected apical extent of the canal. The clinician should resist the urge to rotate the file excessively. If the tip of a small instrument is tightly bound in the blocked segment of the canal and the tip has been worked by pecking, it is prone to fracture in the apical area, further complicating the case.[214] The separated file tip is frequently irretrievable and surgery or extraction may be the result. Dropping down to the next smaller size file and using a very gentle reciprocal rotational motion ("twiddling") will aid in advancement through the blocked canal. Frequently, as apical progress is being made, the clinician will be using an electronic apex locator to gauge the proximity of the apical constriction. Unfortunately, the apex locator is sometimes not able to give an accurate reading in a blocked canal, and because of the continued "sticky" feel that occurs even as the instrument bypasses the foramen and penetrates apical

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tissues, an overextension may result. In order to prevent this complication with its attendant risk of a painful postoperative flare-up, when the estimated working length is reached, a working length radiograph is necessary.[165] Once apical working length is achieved, apical patency should be confirmed, and gentle, short amplitude 1 to 2 mm push-pull strokes should be made until the file can pass freely to the apical constriction (Fig. 10.71).

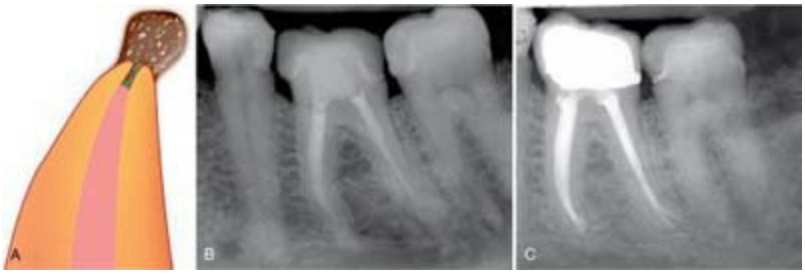

FIG. 10.71 **A,** Diagrammatic representation of a canal block. Fibrotic and/or calcified pulp and debris that is potentially infected remains in the apical segment of the canal when the canal is instrumented short of the apical constriction. **B,** Preoperative radiograph showing obturation short of ideal length. The patient was symptomatic and the canals were blocked. **C,** Three months posttreatment. Treatment took a total of 3.5 hours over three appointments due to the time-consuming nature of bypassing blocked canals.

A) Diagram of a tooth with root canal and circular infected debris at the tip of the root.

B) Radiograph of tooth shows a radiolucent crown with radiopaque upper canals and radiolucent lower canals.

C) Radiograph of tooth shows a radiopaque crown with radiolucent neck, radiopaque canals.

Source: (**A,** Courtesy Dentsply Endodontics.)

If, after a reasonable amount of time, no sticky spot can be found, the clinician must consider the possible presence of a ledge despite possibly not detecting it on the preoperative radiograph. The main problem with ledges is that instruments will invariably find their way to the ledge while finding the original canal is many times impossible. They feel like a hard brick wall, short of length when encountered, and care must be used to prevent worsening the ledge by indiscriminately burrowing into it.[286] In order to

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manage a ledge, the tip of a small #08 or #10 file has a small bend placed in it 1 to 2 mm from the end,[129,277] so the tip of the file forms an approximately 45 degree angle with the shaft of the instrument. The directional stop is oriented to the bend, and the file is carefully negotiated to the level of the ledge. Since ledges form mainly on the outside of curvatures, the directional stop (and thus the bent tip of the file) is turned in the direction of the suspected apical curvature away from the ledge (Fig. 10.72). The file tip is slowly scraped along the internal wall of the canal curve slightly coronal to the level of the ledge[277] looking for another sticky spot. This spot will be the entrance to the apical canal segment and gentle reciprocal rotation will usually allow the file to be negotiated to the canal terminus. Confirm with a radiograph. Once the ledge has been bypassed, short amplitude push-pull and rotational forces keeping the file tip apical to the ledge will be needed to clean and enlarge the apical canal space. When the file can be easily negotiated around the ledge, anti-curvature filing will enable the clinician to blend the ledge into the canal preparation (Fig. 10.73). Many times this cannot be completely accomplished,[286] but as long as the apical segment can be cleaned and obturated, the prognosis should not be adversely affected.

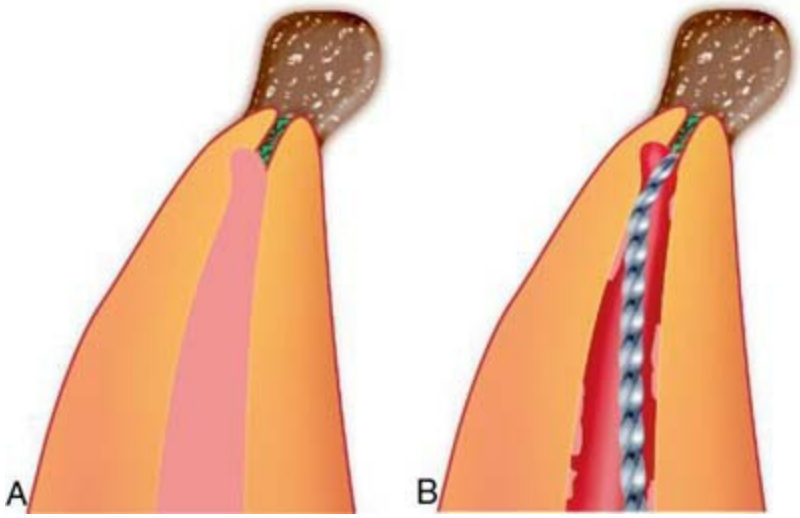

FIG. 10.72 **A,** Diagrammatic representation of a ledged canal. Potentially infected debris remaining in the apical segment can result in posttreatment disease. **B,** Attempting to bypass the ledge with a small file having a 45-degree bend in the tip. Note that the opening to the apical canal segment is on the inside of the canal curvature and coronal to the level of the ledge.

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A) Diagram of a tooth with root canal blocked with circular infected debris at the tip of the root.

B) Diagram of a tooth with root canal blocked with circular infected debris at the tip of the root. A spirally textured file with a 45 degree tip bend inserted in the canal is used to remove the debris.

Source: (Courtesy Dentsply Endodontics.)

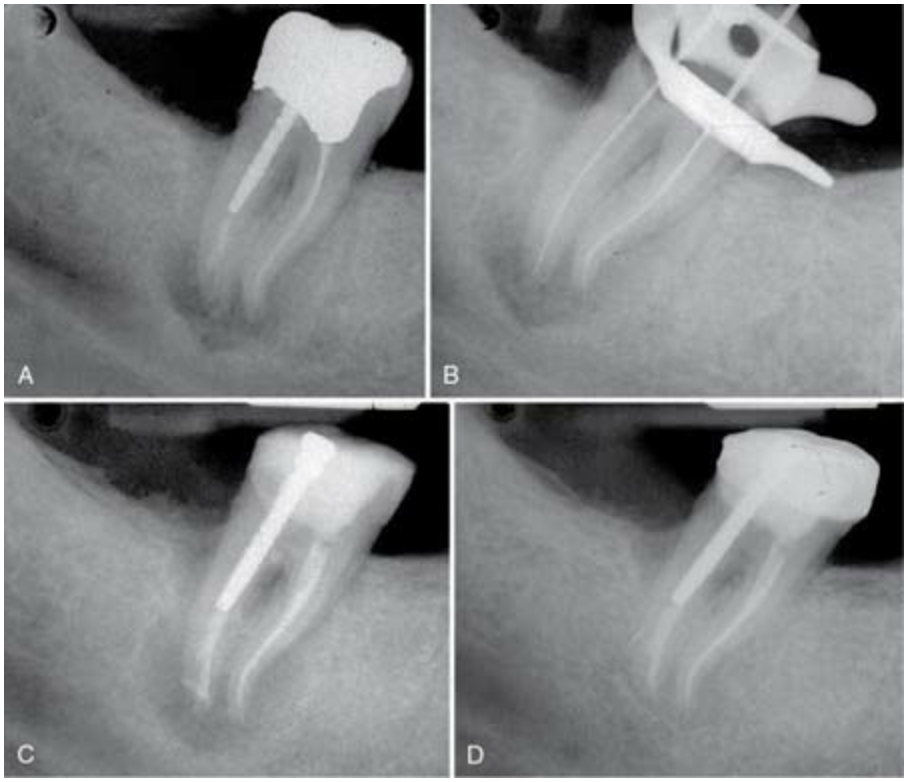

FIG. 10.73 **A,** Preoperative radiograph showing a distal canal ledge with a small amount of sealer that has entered the apical segment. The ledge prevented proper cleaning and sealing of the canal system resulting in posttreatment disease. **B,** The ledge has been bypassed. The attempt is made to blend the ledge into the contour of the prepared canal wall. **C,** Final obturation showing the filled ledge and apical segment. **D,** Thirteen-month recall showing healing. The patient was then directed to have a definitive coronal restoration placed.

A) Radiograph of tooth shows a radiopaque crown and right canal. An radiopaque outline of spirally textured ledge is visible in upper canal and lower canal shows radiolucency.

B) Radiograph of tooth shows a thread-like radiopacity in the canals with radiopaque outline of the clamp.

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C) Radiograph of tooth shows a partially radiopaque crown and right canal. A total radiopaque outline of the filled ledge with a broad head is visible in upper canal and lower canal shows radiolucency.

D) Radiograph of tooth shows a radiopaque crown and right canal. A radiopaque outline of the filled ledge with a broad head is visible in upper canal and lower canal shows radiolucency.

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The use of Greater Taper (GT) NiTi hand files (Dentsply Tulsa Dental) for the blending of ledges has been advocated.[214] The advantage these instruments have is that they are non-end cutting, and their rate of taper is two to six times that of conventional 0.02 tapered files, so they can do the work of multiple 0.02 tapered hand files. Once the ledge has been bypassed and the canal can be negotiated with a conventional size #15 or #20 K-file, a GT hand file is selected. The K-file creates a pilot hole so that the tip of the GT file can passively follow this glide path beyond the ledge. The GT file must have a tip diameter of 0.2 mm (#20) and a taper that will vary depending on the requirements of the preparation. The largest taper that will enter the apical segment is used; however, these instruments must be precurved, which presents a challenge since they are made from nickel-titanium alloy. In order to precurve this superelastic shape memory alloy, a file-bending tool, such as the Endo Bender Pliers (Kerr Corporation, Orange, CA), is needed. The pliers grasp the tip of the instrument, and the file is over curved between 180 and 270 degrees to plastically deform the alloy. At this time, the appropriate tapered GT file is then carried into the canal, and the rubber stop is oriented so that the instruments precurved, working end can bypass and move apical to the ledge. The GT file is then worked to length, and the ledge is either reduced or eliminated (Fig. 10.74).

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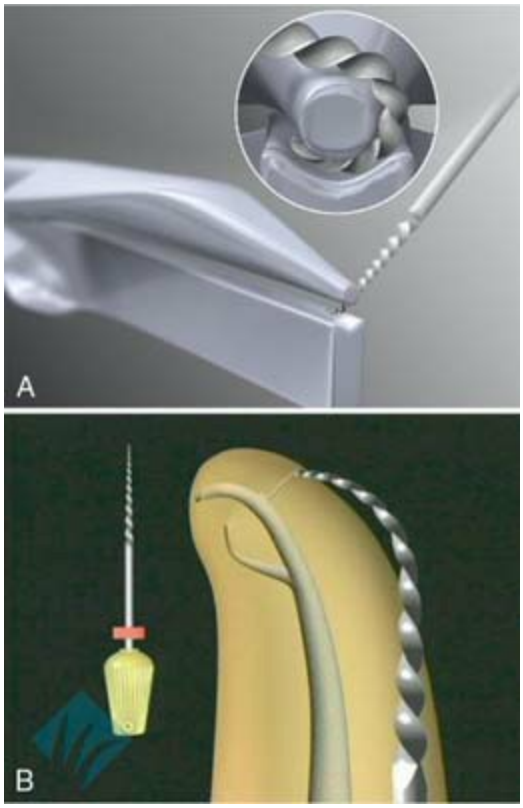

FIG. 10.74 **A,** Endobender Pliers (Kerr Corporation) used to overbend a nickel-titanium hand GT file. **B,** GT hand file can hold a bend to allow it to bypass ledges.

A) An endobender pliers bending a nickel titanium tip. A focused image shows the curving of tip in the pliers.

B) A three-dimensional diagram shows a tooth tip with a branched canal. A curved nickel titanium tip is shown reaching up to the canal.

Source: (Courtesy Dr. Steve Buchanan.)

If the canal blockage or ledge cannot be negotiated, then the canal space coronal to the impediment should be cleaned, shaped, obturated, and coronally sealed. The patient must be informed of this complication, the guarded prognosis, and the need for regular reevaluation (Fig. 10.75). If symptoms of posttreatment disease arise subsequently, apical surgery or extraction will be needed.[214,277]

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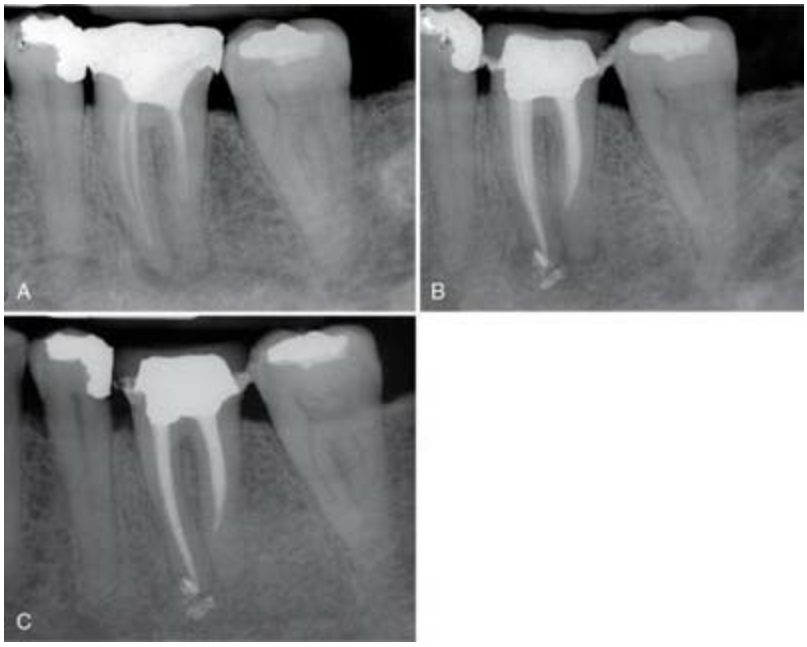

FIG. 10.75 **A,** Preoperative radiograph showing mesial canal blockages and potential distal root ledging with accompanying posttreatment disease. **B,** Final film showing mesial blockages bypassed but inability to negotiate beyond the distal ledge. The patient elected to pursue no further treatment at this time. **C,** One-year recall. Despite not achieving all the aims of conventional endodontic therapy, periradicular healing is apparent. The patient is asymptomatic and will now begin the final restoration with the knowledge that apical surgery may be needed in the future.

A) Radiograph of tooth shows a radiopaque crown with irregularly spread white patch on the crown. The radiopaque patch projects into another tooth.

B) Radiograph of tooth shows a radiopaque crown with irregular white patch on the crown. The radiopaque patch connects to the neighboring teeth through thread-like radiopaque structures.

C) Radiograph of tooth shows a radiopaque crown with irregular white patch on the crown, radiolucent left canal and radiolucent partial right canal. The radiopaque patch is visibly disconnected from the neighboring teeth.

Finishing the retreatment

After regaining the apical extent of the canal system, routine endodontic procedures are instituted to complete the retreatment. Any missed canals must be found using magnification, microexcavation techniques, and most importantly, the knowledge of canal anatomy that is discussed in another

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section of this text (Fig. 10.76). One cannot find a canal unless one suspects that it is there. Cleaning and shaping procedures must focus on a crown-down approach to minimize extrusion of irritants into the periradicular tissues, and also must emphasize enlargement of the apical portion of the preparation to ensure complete removal of apical debris. These aims are best accomplished using technique hybridization during instrumentation, keeping the goals of the retreatment procedure in mind. These matters are covered in detail in another chapter of this text. Canal disinfection procedures are, however, paramount after the completion of cleaning and shaping. Since the primary cause of posttreatment disease is usually microbial[73,182] and these microbes (i.e., *Enterococcus faecalis*) are frequently resistant to traditional canal disinfection regimes,[21] every effort must be made to eliminate these organisms from the canal system. This effort is complicated by the fact that no instrumentation regime can predictably remove the entire previous root filling from the canal space after retreatment.[16] This leaves areas where microbes can reside underneath fragments of root filling materials and remain protected from standard antimicrobial canal irrigants such as sodium hypochlorite. Whether the canal space can be adequately disinfected when completing treatment in one visit or whether an interappointment medicament such as calcium hydroxide is needed is still a matter of debate, and the reader is directed to the appropriate chapter of this text for details of the problem. It is important to keep in mind, however, that teeth that require retreatment also require the highest level of disinfection possible to ensure the most favorable outcome.

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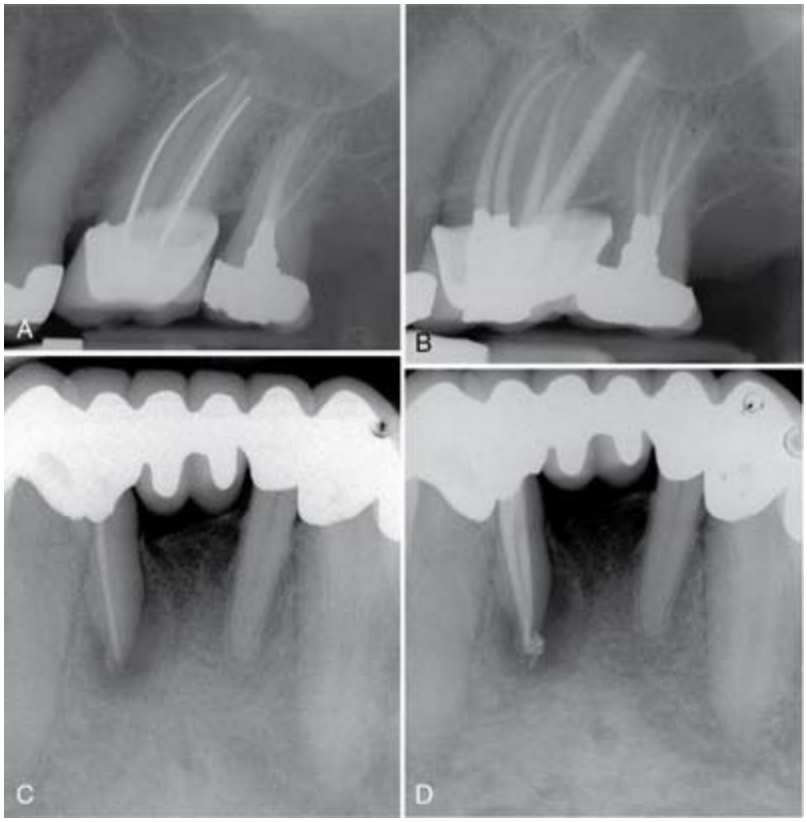

FIG. 10.76 Frequently missed canals that may result in posttreatment disease. **A,** Missed second mesiobuccal canal (MB2) in an upper molar. **B,** Final obturation showing cleaned, shaped, and filled MB2 canal. **C,** Missed lingual canal on a lower incisor results in posttreatment disease. **D,** Immediate postoperative film showing management of the missed canal.

A) Radiograph of tooth shows a radiopaque crown and canals.

B) Radiograph of tooth shows a radiopaque crown and neck branching into three radiopaque canals while lower canals show radiolucency.

C) Radiograph of tooth shows a radiopaque crown and a single canal with an arrow pointed to a gray circular dot.

D) Radiograph of tooth shows a radiopaque crown and a two canals with an arrow pointed to a white circular dot.

Repair of perforations

Occasionally, posttreatment endodontic disease will be the result of root perforation.[125] Root perforations are created pathologically by resorption and

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caries, and iatrogenically during root canal therapy (zip, strip, and furcation perforations) or its aftermath (e.g., post preparation perforation) (Fig. 10.77).[208] When they are present, perforations may usually be found during the diagnostic phase as areas where the root filling materials or possibly restorative materials such as posts are found to radiographically leave the confines of the presumed canal space and approach or cross the radiographic interface between the dentin and the periodontal ligament. Angled radiographs are of paramount importance in determining whether a perforation exists and locating which surface or surfaces of the root have been perforated. This information is necessary when deciding upon treatment options. The use of CBCT to evaluate potential perforations may also be useful, but if the canal space has already been obturated, the presence of beam hardening and other artifacts created by the radiopaque material in close proximity to the perforation site may limit the usefulness of this 3D imaging technology. Frequently, cervical and occasionally mid-root perforations are associated with epithelial downgrowth and subsequent periodontal defects, so thorough periodontal assessment is required (Fig. 10.78).[163,234] If there were no evidence of posttreatment disease associated with the defect or tooth, then no treatment would be indicated. If, however, there is evidence of periradicular periodontitis, repair may be instituted in one of two ways, either nonsurgically by approaching the defect internally through the tooth or surgically by using an external approach through the periradicular tissues.[208] In general, if all other factors are considered equal, internal nonsurgical perforation repair will be the preferred method since it is usually less invasive, produces less destruction of periradicular tissues via the surgical access wound needed, and isolation from microbes and disinfection is usually enhanced. If, however, the defect is readily accessible surgically, and disassembly of the existing restorations would impose an unacceptably high cost and long treatment time to the patient, surgical repair should be selected. If a longstanding defect has a periodontal lesion that has formed around it, surgery perhaps with guided tissue regeneration will usually be needed.[14] However, in most of these cases, nonsurgical retreatment and internal perforation repair prior to surgery will be beneficial to the treatment outcome. A multidisciplinary approach will be required, usually in consultation with the restorative dentist, a periodontist, and perhaps an

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orthodontist.[214,291]

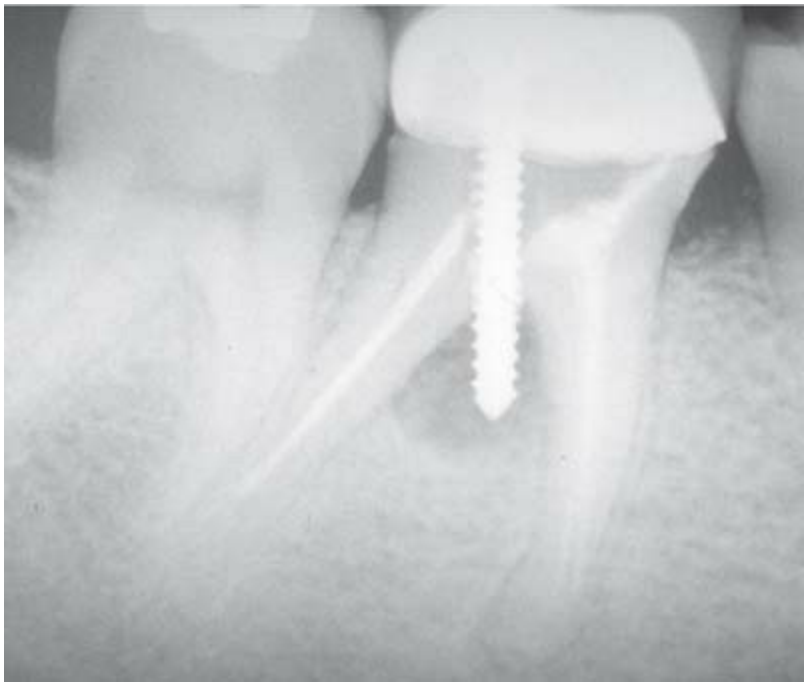

FIG. 10.77 Furcal post perforation resulting in persistent infection and furcal bone loss.

Radiograph of tooth shows a radiopaque crown and canals with radiolucent neck. A radiopaque outline of the spirally textured post is visibly perforated in the periodontal ligament.

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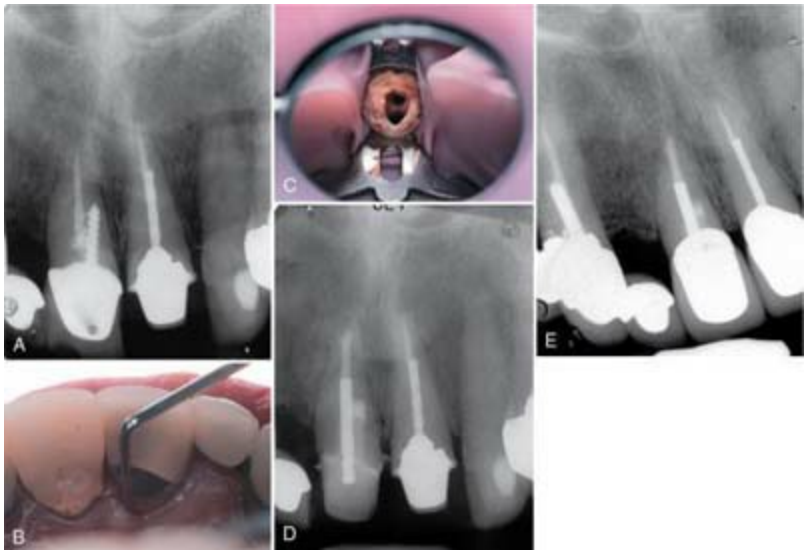

FIG. 10.78 **A,** Mesially angulated preoperative radiograph showing a palatally oriented post perforation in an upper incisor. **B,** An 8 mm narrow-based probing defect on the mesiopalatal corner of the tooth. **C,** Following coronal disassembly, the true canal can be seen lying in a facial direction relative to the palatal post preparation. **D,** The perforation was repaired with an external matrix of Colla-Cote and MTA. Subsequently, in conjunction with a periodontist, periodontal flap surgery was used to remove periodontal disease etiology from the long-standing pocket, and guided tissue regeneration procedures were instituted. **E,** Three-year reevaluation. The tooth is asymptomatic and mesiopalatal probing depth is 4 mm.

A) Radiograph of tooth shows a radiopaque crown with a black spot on the crown and radiolucent canal. An outline of a radiopaque spiral textured post is visible.

B) Close-up view of the posterior lower jaw shows a cracked tooth examined by a periodontal probe.

C) Close-up of the horizontal section of a molar held in a dam clamp shows an open crown with black spots visible in the cavity.

D) Radiograph of tooth shows a radiolucent crown and canal. An outline of the radiopaque rod-like dressing material is visible.

E) Radiograph of tooth shows a radiopaque crown and radiolucent canal. An outline of the radiopaque rod-like dressing material is visible.

Factors that affect the prognosis of perforation repair include location of perforation, time delay before perforation repair, ability to seal the defect, and previous contamination with microorganisms.[143,234,241] Generally, the more

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apical the perforation site, the more favorable the prognosis; however, the converse is true for the repair procedure itself. The difficulty of the repair will be determined by the level at which the perforation occurred. If the defect is in the furcal floor of a multirooted tooth, or in the coronal one third of a straight canal (access perforation), it is considered to be easily accessible. If it is in the middle third of the canal (strip or post perforations) difficulty increases, and in the apical third of the canal (instrumentation errors) predictable repair is most challenging and, frequently, apical surgery will be needed.

Immediate repair is better than delayed repair, since delay can cause breakdown of the periodontium, resulting in endoperio lesions that are difficult to manage,[131,240] and elimination of microbial contamination of the defect and sealing it properly is critical to success. Many materials have been advocated for the repair of perforations in the past; however, none provided predictable healing after treatment. Commonly used materials include amalgam, Super EBA cement (Bosworth, Skokie, IL), various bonded composite materials, and more recently, bioceramic materials such as mineral trioxide aggregate (Pro-Root MTA, Dentsply, York, PA and MTA Angelus, Londrina, PR, Brazil),[214] Biodentine (Septodont, Saint Maur des Fosses, France), and EndoSequence Root Repair Material (Brasseler USA, Savannah, GA) (Fig. 10.79).

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FIG. 10.79 Bioceramics are the materials of choice for perforation repair. **A,** ProRoot MTA (Dentsply Endodontics) is a medical grade of Portland cement that has had the arsenic removed so that it can be used in the human body. **B,** Brasseler Endosequence Root Repair Material (Brasseler USA). This material has better handling characteristics than MTA and minimizes discoloration of the tooth. **C**, Biodentine (Septodont) is another bioceramic that minimizes tooth discoloration.

Set of three images show the materials used for the repair of dental perforations. These are labeled from A through C.

When mineral trioxide aggregate was introduced for perforation repair, the choice of which repair material to use became more clear.[163,208] MTA has many advantages over previous restoratives when being used for perforation repair. The material seals well,[186,302] even when the cavity preparation is contaminated with blood.[273] It is very biocompatible,[193,201,274,276] rarely

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eliciting any response from the periradicular tissues, and a cementum-like material has been consistently shown to grow directly on the material after placement.[113,201] MTA has also been shown to have a high degree of clinically favorable long-term outcomes when used as a perforation repair material.[163,208] The main disadvantage of MTA is the long time required for setting[275] that makes this material inappropriate for transgingival defects such as those associated with cervical resorption. If the material is in contact with oral fluids, it will wash out of the defect prior to setting so a more rapid setting resin-ionomer such as Geristore (Den-Mat, Santa Maria, CA) is recommended for lesions that cross the gingival margin (Fig. 10.80).[23,56,225] MTA is available in the original gray formulation and an off-white option. Their sealing ability seems comparable,[69] but questions remain as to whether white MTA exhibits the same biocompatibility[199] and will have the same long-term success as the older variety. Also, both types of MTA cause significant tooth discoloration. This is an effect that is seen in all the bioceramics, but it is much less pronounced in the newer ones like Biodentine and EndoSequence Root Repair Material.[178]

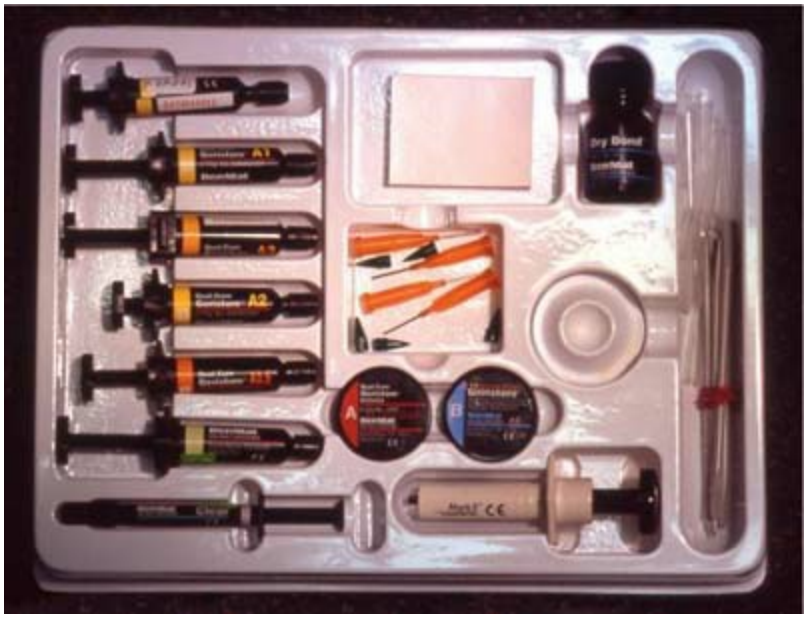

FIG. 10.80 Geristore Kit. This and other resin-ionomers have been advocated for cervical perforation repair due to good biocompatibility and the shorter more controlled setting times that make them useful for transgingival root fillings.

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A display of Geristore kit shows components including syringes, auto-mix dispensing tips, intra-oral tips, light cure test rings and instructions.

If the perforation is to be repaired nonsurgically through the tooth, coronal-radicular access to the defect is prepared as stated previously (Fig. 10.81). First, the root canals are located and preliminarily instrumented to create enough coronal shape to allow them to be protected from blockage by the repair material. The defect is cleaned and sometimes enlarged with the use of ultrasonics or appropriate rotary drills, such as the Gates-Glidden, to remove any potentially contaminated dentin surrounding the perforation. Use of a disinfectant irrigating solution such as sodium hypochlorite should be considered if the perforation is not so large as to allow the irrigant to significantly damage the periradicular tissues. If the perforation is large, then sterile saline should be used as an irrigant and disinfection of the margins of the defect is performed using mechanical dentin removal. Arens and Torabinejad[12] have advocated the use of copious flushing of the defect with 2.5% hypochlorite, but in light of the potential severe complications of hypochlorite overextension through a perforation,[84] extreme care should be used. After the defect has been cleaned, vigorous bleeding may result. Hemostasis should be undertaken using collagen (Colla-Cote, Integra Life Sciences, Plainsboro, NJ) (see Fig. 10.8, *B*), calcium sulfate (Capset, Lifecore Biomedical, Chaska, MN), or calcium hydroxide[214]; however, astringents such as ferric sulfate should be avoided, since the coagulum they leave behind may promote bacterial growth and compromise the seal of the repair.[148]

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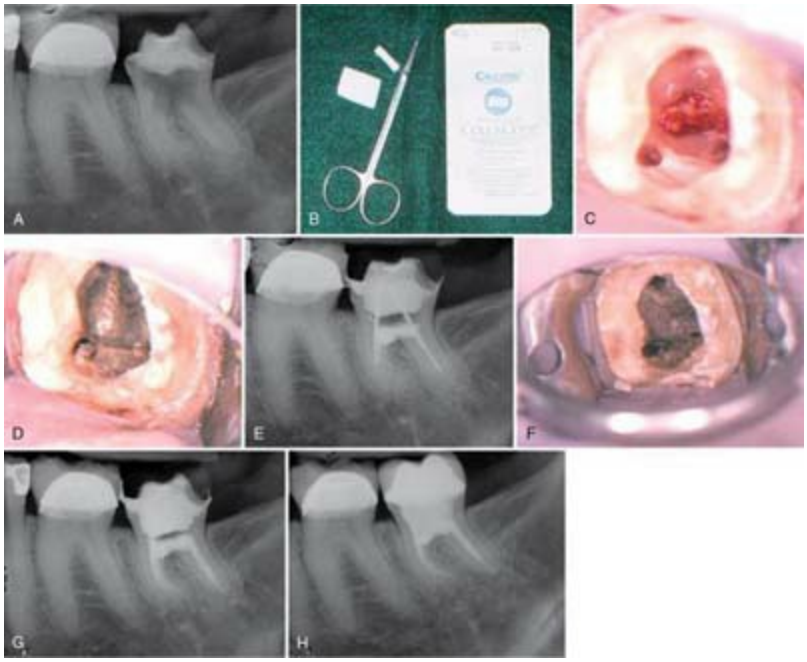

FIG. 10.81 **A,** Large furcal perforation created during an attempt at endodontic access. **B,** Colla-Cote (Integra Life Sciences, Plainsboro, NJ) to be used as an external matrix material to recreate the external root contour. **C,** Canals have been found, preliminarily instrumented, and the external matrix has been placed. **D,** Canals are protected from being blocked using large endodontic files cut off above the orifice. MTA has been placed into the defect. **E,** Radiograph showing the initial repair with MTA recreating the vault of the furcation. **F,** On the second appointment, the blocking files are removed with difficulty since the MTA has flowed into the flutes and set. At this time, endodontic therapy is completed as normal. **G,** Postobturation radiograph. Note the radiolucency in the furcal vault, which represents the Colla-Cote external matrix material. **H,** Nineteen-month reevaluation. The patient is asymptomatic and there is evidence of healing in the furcal vault area.

A) Radiograph of tooth shows a radiopaque uneven crown with a black patch around the neck and radiolucent pulp.

B) An image of a colla-cote with a pair of scissors on the left.

C) Close-up of the horizontal section of a molar shows an open crown exposing red cavity with holes around.

D) Close-up of the horizontal section of a molar shows an open crown exposing a textured cavity with an intensified hole.

E) Radiograph of tooth shows a radiopaque crown and radiolucent pulp. An 'A' shaped radiopaque outline of root repair material is visible.

F) Close-up of the horizontal section of a molar held in a dam clamp shows an

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open crown exposing a dark and textured cavity with intensified holes.

G) Radiograph of tooth shows a radiopaque crown, radiopaque segmented neck filled with root repair material and radiopaque canals.

H) Radiograph of tooth shows a radiopaque crown and radiolucent, radiopaque neck filled with root repair material and radiopaque canals.

When the bleeding has been controlled, some easily removable material should be placed over the entrances to the deeper portion of the canals to prevent the repair material from blocking reaccess to the apical terminus. The canals may be protected with cotton, gutta-percha cones, paper points, or shredded collagen. The use of severed files is not recommended, because removal of the files after placement of the repair material is very difficult since the material tends to lock into the instrument's flutes (see Fig. 10.81, *F*). After protecting the canals, the perforation site is inspected to determine if an external matrix is needed to ensure a proper contour of the restoration.[147] If the surrounding bone is closely adapted to the defect margins, minimal to no matrix material will be necessary; however, if the perforation is associated with a large osseous defect, this must be filled with an external matrix to minimize overcontouring of the repair restoration. The matrix material should be a biocompatible, usually absorbable material such as collagen, freeze-dried demineralized bone allograft (FDDB), hydroxylapatite, Gelfoam, or calcium sulfate.[208,214] Care must be exercised so that the external matrix material is not condensed so forcefully that it damages adjacent vital structures, such as the mental nerve or the floor of the sinus.

Following the preparation of the defect, the repair material is placed. It may be carried in a small syringe or amalgam carrier, and it is condensed with pluggers or microspatulas. In the case of MTA in an accessible defect, the butt end of paper points make an excellent condenser since they can wick some of the water out of the material giving it a firmer consistency, aiding condensation. Upon reentry, the material should be set hard and well retained in the perforation site (see Fig. 10.81, *F*).[245] If there is an overextension of the material beyond the normal external root contour, it seems not to affect the prognosis of the repair.[12,208] While the other bioceramics like Biodentine and EndoSequence Root Repair Material handle more easily than MTA and also set well in both moist and dry environments, the set materials are not as

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hard as set MTA.[39]

If the perforation is deeper in the canal, the objectives and principles or repair as just outlined all apply, except that access to the defect is more complicated (Fig. 10.82). Protecting the canal from blockage is somewhat more difficult and placement of the repair material requires the enhanced vision that is provided by the surgical operating microscope. Ideally, the canal should be fully shaped prior to the repair attempt,[214] and a canal patency protector should be placed apical to the defect. In some of these instances, protecting the canal can be accomplished by using a severed file, notwithstanding the previous warning, since not only can it protect the canal from blockage, but it can also be used as an indirect carrier for transmitting ultrasonic energy to the MTA, causing it to "slump" into the defect when direct condensation is impossible. The file is placed into the canal to a level well below the defect, and the MTA is carried to place. Once condensation has been performed as well as possible, the coronal extent of the file is touched with an ultrasonic tip to vibrate the MTA into the defect. After this is accomplished, the file must be vigorously instrumented in a short 1 to 2 mm amplitude push-pull motion to free it from the placed MTA so that it can be easily removed after the material is set (Fig. 10.83).[214] There is some evidence that ultrasonic placement of MTA may enhance the seal against bacteria in an apexification model,[144] although other researchers have not agreed with this conclusion, and found poorer canal wall adaptation when filling the apical extent of canals using ultrasonic condensation compared with hand filling.[10] Further study of ultrasonic MTA placement is warranted, but clinical observation suggests that this method has merit.

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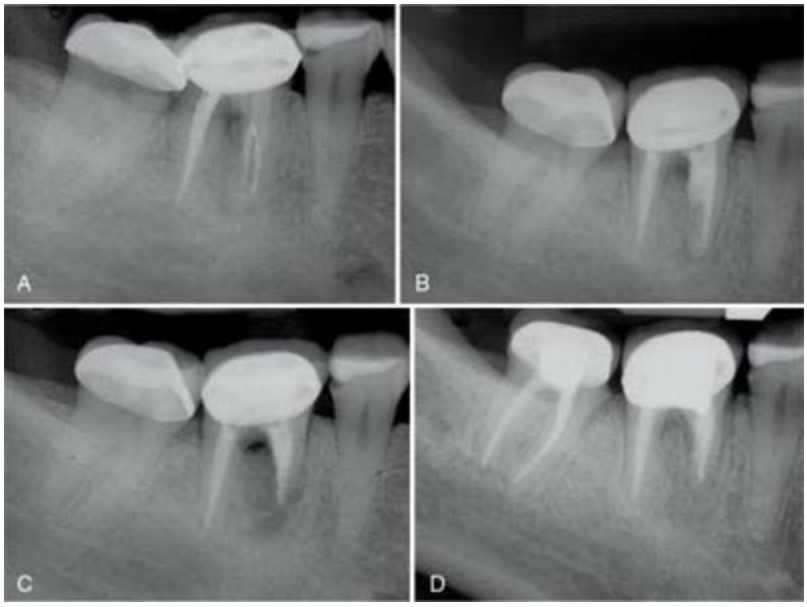

FIG. 10.82 Mid-root level perforation repair. **A,** Preoperative film showing mesial strip perforation with bone loss. **B,** Nonsurgical internal repair with inability to negotiate canals to the apical terminus and MTA overextension into the furca. **C,** Apical and perforation repair surgery performed. **D,** One-year follow-up showing complete healing.

A) Radiograph of tooth shows a radiopaque crown with radiopaque left canal and radiolucent right canal segmented vertically.

B) Radiograph of tooth shows a radiopaque crown with radiopaque left canal and radiopaque right canal with outline of root repair material.

C) Radiograph of tooth shows a radiopaque crown with radiopaque canals.

D) Radiograph of tooth shows a radiopaque crown with radiopaque canals. A radiopaque outline of the root repair material is slightly visible in the upper right canal.

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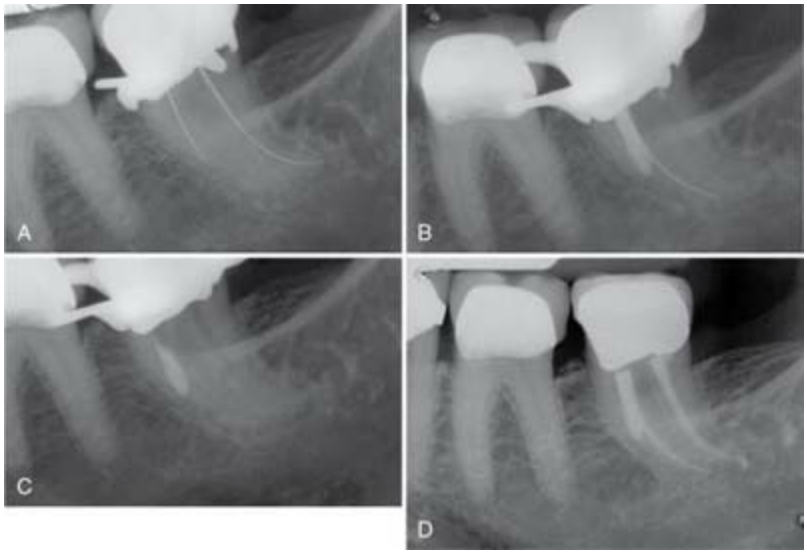

FIG. 10.83 **A,** This patient was in extreme pain following initial endodontic instrumentation by her dentist. Mid-root level perforation found on access. **B,** The original canal was found and protected with an endodontic file. MTA was vibrated using ultrasonic energy applied to the file and it flowed into the defect. Then the file is moved in a push-pull manner to dislodge it from the MTA prior to closure. Note that the defect was intentionally enlarged to allow for more predictable application of the MTA. **C,** On the second appointment, the file is withdrawn easily since it was detached from the MTA repair material. Now the endodontic therapy is concluded as normal. The patient has been asymptomatic since the end of the first appointment. **D,** Twenty-seven–month follow-up showing complete healing.

A) Radiograph of tooth shows a radiopaque crown with radiopaque thread-like structures in the canals.

B) Radiograph of tooth shows a radiopaque crown with outline of the root repair material in the upper left canal.

C) Radiograph of tooth shows a radiopaque crown with a radiopaque stain in the upper left canal.

D) Radiograph of tooth shows a radiopaque crown with radiopaque curved canals.

If the perforation is in the apical portion of the canal, it is usually due to a procedural accident during instrumentation of a curved canal and is invariably accompanied by a block or ledge. This type of perforation is the most difficult to repair, since repair not only involves cleaning and sealing the defect, but also finding, cleaning, and filling the apical canal segment. All

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the aforementioned techniques for managing blocks and ledges are required to find and clean the apical canal segment. When this has been accomplished, the decision is made as to whether the canal should be filled with a bioceramic or with gutta-percha and sealer. The bioceramic is undoubtedly more effective in sealing the canal (especially if it cannot be dried) and is much more biocompatible but carrying it predictably to the apical extent of a curved canal is problematic. If a holding file is placed in the apical canal segment to anticipate eventual gutta-percha placement after the bioceramic repair material is set, then the presence of the file precludes consistent extension of the bioceramic into the apical end of the defect. If a holding file is not placed, the bioceramic can be placed into the prepared apical segment, which may not be completely three-dimensionally obturated. Generally, whichever choice is made, the outcome will be unpredictable, and so the patient must be advised that regular reevaluation is necessary and apical surgery or extraction may ultimately be needed.

Prognosis of retreatment

When the proper diagnosis has been made, and all the technical aspects of retreatment are carefully performed, orthograde retreatment can be highly successful (Fig. 10.84). The prognosis depends to a large extent on whether apical periodontitis exists prior to retreatment.[188,189] In a systematic review of outcomes studies,[75] Friedman and Mor report that in the absence of prior apical periodontitis, the incidence of healed cases after both initial treatment and orthograde retreatment ranges from 92% to 98% up to 10 years after treatment. When prior apical periodontitis is present, the incidence of healing drops to 74% to 86%, regardless of whether initial treatment or orthograde retreatment was performed. The authors state that this “similar potential to heal after initial treatment and orthograde retreatment challenges the historic perception of the latter having a poorer prognosis than the former.”[75]

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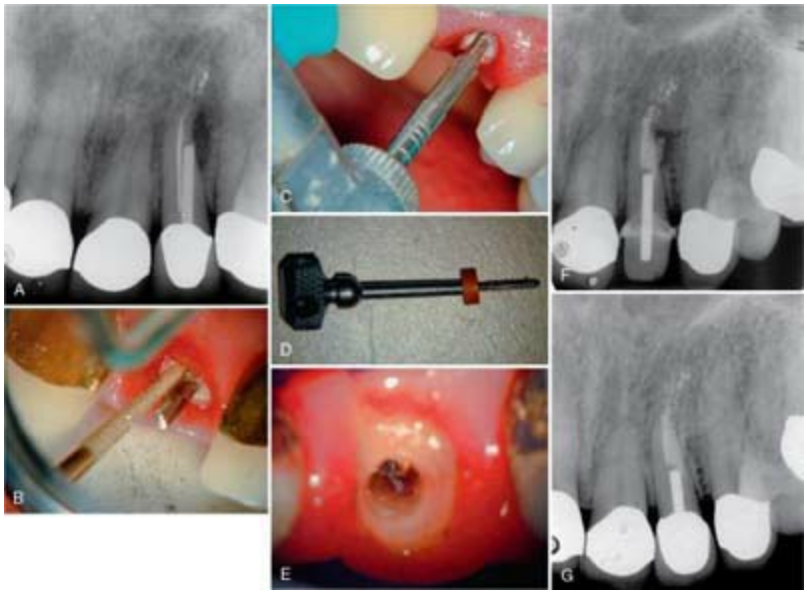

FIG. 10.84 **A,** Preoperative radiograph showing mid-root level post perforation and associated periradicular periodontitis. **B,** The crown was removed and ultrasonic energy was applied to the post. **C,** Using the trephine bur from the Ruddle Post Removal kit to mill the end of the post. **D,** The screw post is removed using the wrench from the Ruddle kit. **E,** After removal of the post and gutta-percha, a plastic solid core carrier is found in the canal and removed using the techniques described in this chapter. **F,** Postoperative radiograph showing MTA perforation repair, canal seal with gutta-percha, and post and core fabrication. **G,** Thirteen-month recall showing healing around the perforation repair site.

A) Radiograph of tooth shows a radiopaque crown with an outline of a radiopaque post in the canal.

B) Close-up of an open mouth with an ultrasonic bit shows periodontal ligament exposing the post from a crown-less tooth.

C) Close-up of an open mouth shows a trephine bit milling the exposed post.

D) Close-up of a wrench with extracted post.

E) Close-up of the horizontal section of a molar shows an open crown exposing a plastic carrier in the cavity.

F) Radiograph of tooth shows a radiolucent crown with an outline of a radiopaque root repair material in the canal and radiolucent lower canal.

G) Radiograph of tooth shows a radiopaque crown with an outline of a radiopaque root repair material in the canal and radiolucent lower canal.

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Unfortunately, these numbers mean that the desired outcome will not occur in potentially one-quarter of retreatment cases. Many techniques and devices for endodontic retreatment have been mentioned here to aid the clinician. However, none of this will guarantee success. Even when strict endodontic principles and fundamentals are followed, the result may be persistent posttreatment disease. When healing does not occur, the clinician is faced with the decision of what to do next. The choice is between four treatment options: observation, endodontic surgery, extraction-replantation, or extraction.

Many times, a tooth that has persistent apical periodontitis may remain in asymptomatic function for an extended period of time, a state that has been referred to as functional retention of the tooth.[75] If the patient's goal of treatment is not necessarily complete healing of the tooth, but simply to retain it in function and without pain, then regular evaluation by the clinician is warranted. If signs and symptoms of worsening infection such as progressive enlargement of a periapical radiolucency, pain, periodontal pocket formation, or sinus tract eruption occur, then further treatment may be needed. However, many teeth classified early on as uncertain healing may indeed be retained for many years.[174]

Endodontic surgery (Fig. 10.85) is a very predictable procedure[75,100,236] that can be performed on most teeth; however, there are some anatomical and medical concerns regarding treatment planning for this procedure that are covered in detail in another chapter. Extraction-replantation (Fig. 10.86), also referred to as intentional replantation,[190] is another treatment option. This involves extraction of the tooth and performing the apicoectomy and root-end filling while the tooth is out of the patient's mouth, followed by replantation and splinting if indicated. This procedure is also discussed in detail in another chapter. Extraction and replacement should be the treatment of last resort, to be selected only when the tooth has been shown to be nonrepairable. If the decision is made to extract the tooth, usually replacement will be necessary to prevent shifting of the dentition with its attendant problems. Replacement can be with an implant, a fixed partial-denture, or a removable partial-denture.

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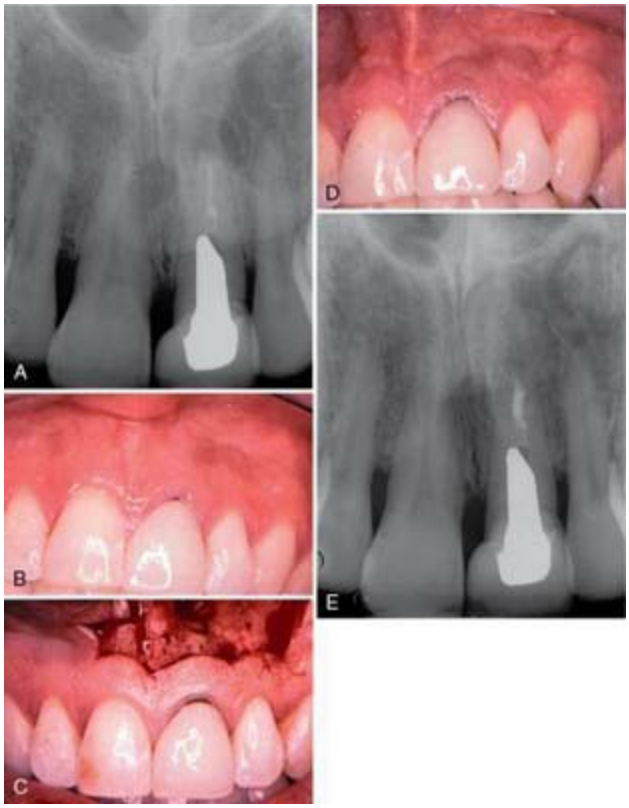

FIG. 10.85 **A** and **B,** Preoperative images showing posttreatment disease in the upper left central incisor and a large custom cast post. The patient elected to leave the post and perform surgery rather than risk damage to his new crown. **C,** Submarginal rectangular flap design. **D,** Three-week follow-up showing excellent soft-tissue healing. **E,** Eighteen-month follow-up showing excellent healing of the periradicular tissues.

A) Radiograph of tooth shows a radiopaque outline of the cast post in the crown and neck.

B) Close-up of frontal upper jaw shows breakage of tissue between the central incisor and the gingiva.

C) Close-up of frontal upper jaw shows a lesion in the gingival surface.

D) Close-up of frontal upper jaw shows a slit between the central incisor and the gingiva.

E) Radiograph of tooth shows a radiopaque outline of the cast post in the crown and neck.

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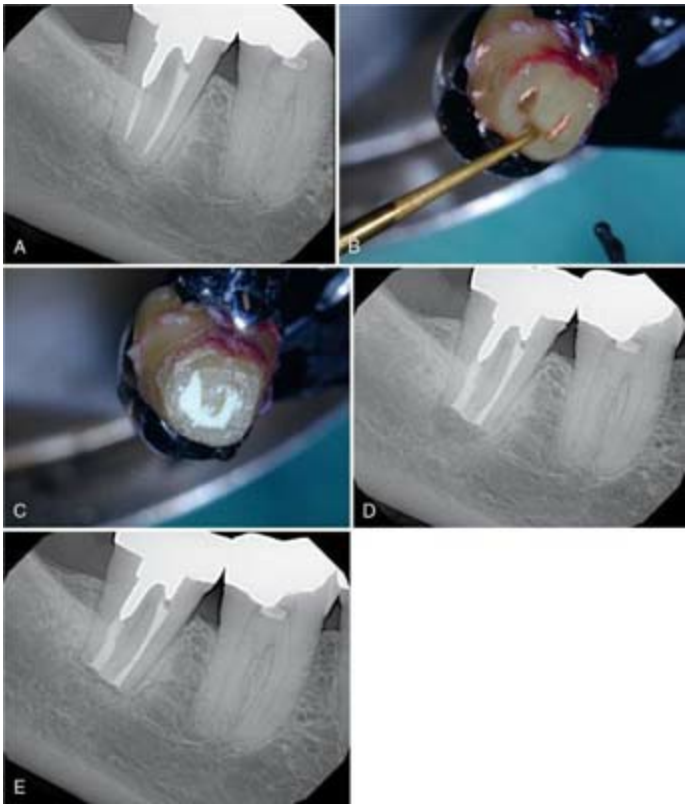

FIG. 10.86 **A,** This lower second molar became symptomatic many years after nonsurgical treatment. Nonsurgical retreatment had a guarded prognosis due to the large multiroot cast custom post and core. Surgery was precluded by the poor access and proximity of the inferior alveolar canal. **B,** Ultrasonic root end preparation is made in the extracted tooth. **C,** A white MTA retrograde filling was placed. Note the C-shape. **D,** Immediate post-implantation radiograph. **E,** Seven-month reevaluation showing apical healing. The patient was asymptomatic.

A) Radiograph of tooth shows a radiopaque white patch on the projecting towards the canals. Radiolucency is visible in lower canals.

C) Close-up of ultrasonic bit and the horizontal section of extracted tooth shows unfilled root-ends.

D) Close-up of the horizontal section of extracted tooth shows filled root-ends.

E) Radiograph of tooth shows a radiopaque white patch on the projecting towards the canals. Radiopacity is visible in lower canals.

F) Radiograph of tooth shows a radiopaque white patch on the projecting towards the canals. Radiopacity is visible in lower canals.

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Conclusion

Posttreatment endodontic disease does not preclude saving the involved tooth. In fact, most of these teeth can be returned to health and long-term function by current retreatment procedures. In most instances, the retreatment option provides the greatest advantage to the patient since there is no replacement that functions as well as a natural tooth, and it can significantly improve a patient's quality of life and chewing ability over time.[110] Armed with the information in the preceding section, appropriate armamentaria, and the desire to do what is best for the patient, the clinician will provide the foundation for long-term restorative success.

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References

1. AAE/AAOMR: *Use of cone-beam-computed tomography in endodontics*, 2009.
2. AAE/AAOMR: *Use of cone beam computed tomography in endodontics—2015/2016 update*, 2016.
3. Abbott PV. *Incidence of root fractures and methods used for post removal*, Int Endod J 2002;35: 63.
4. Abramovitz I, Relles-Bonar S, Baransi B, et al: *The effectiveness of a self-adjusting file to remove residual gutta-percha after retreatment with rotary files*, Int Endod J 2012;45: 386.
5. Coucil on Scientific Affairs ADA. *Dental endosseous implants: an update*, J Am Dent Assoc 2004;135: 92.
6. Alfouzan K, Jamleh A. *Fracture of nickel titanium rotary instrument during root canal treatment and re-treatment: a 5-year retrospective study*, Int Endod J 2018;51: 157.
7. Allard U, Andersson L. *Exposure of dental personnel to chloroform in root-filling procedures*, Endod Dent Traumatol 1992;8: 155.
8. Altshul JH, Marshall G, Morgan LA, et al: *Comparison of dentinal crack incidence and of post removal time resulting from post removal by ultrasonic or mechanical force*, J Endod 1997;23: 683.
9. American Association of Endodontists. Guide to clinical endodontics 2004; Chicago.
10. Aminoshariae A, Hartwell GR, Moon PC. *Placement of mineral trioxide aggregate using two different techniques*, J Endod 2003;29: 679.
11. Amsterdam M. *Periodontal prosthesis. Twenty-five years in retrospect*, Alpha Omegan 1974;67: 8.

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www.konkur.in

12. Arens DE, Torabinejad M. *Repair of furcal perforations with mineral trioxide aggregate: two case reports*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 84.
13. Atrizadeh F, Kennedy J, Zander H. *Ankylosis of teeth following thermal injury*, J Periodontal Res 1971;6: 159.
14. Azim AA, Lloyd A, Huang GT. *Management of longstanding furcation perforation using a novel approach*, J Endod 2014;40: 1255.
15. Azim AA, Wang HH, Tarrosh M, et al: *Comparison between single-file rotary systems: part 1-efficiency, effectiveness, and adverse effects in endodontic retreatment*, J Endod 2018;44: 1720.
16. Baldassari-Cruz LA, Wilcox LR. *Effectiveness of gutta-percha removal with and without the microscope*, J Endod 1999;25: 627.
17. Baratto Filho F, Ferreira EL, Fariniuk LF. *Efficiency of the 0.04 taper ProFile during the re-treatment of gutta-percha-filled root canals*, Int Endod J 2002;35: 651.
18. Barborka BJ, Woodmansey KF, Glickman GN, et al: *Long-term clinical outcome of teeth obturated with resilon*, J Endod 2017;43: 556.
19. Barbosa SV, Burkard DH, Spångberg LS. *Cytotoxic effects of gutta-percha solvents*, J Endod 1994;20: 6.
20. Barrieshi-Nusair KM. *Gutta-percha retreatment: effectiveness of nickel-titanium rotary instruments versus stainless steel hand files*, J Endod 2002;28: 454.
21. Basrani B, Tjaderhane L, Santos JM, et al: *Efficacy of chlorhexidine- and calcium hydroxide-containing medicaments against Enterocaucus faecalis in vitro*, Oral Surg 2003;96: 618.
22. Beasley RT, Williamson AE, Justman BC, et al: *Time*

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www.konkur.in

required to remove GuttaCore, Thermafil Plus, and Thermoplasticized Gutta-percha from moderately curved root canals with ProTaper files, J Endod 2013;39: 125.

23. Behnia A, Strassler HE, Campbell R. *Repairing iatrogenic root perforations*, J Am Dent Assoc 2000;131: 196.
24. Berbert A, Filho MT, Ueno AH, et al: *The influence of ultrasound in removing intraradicular posts*, Int Endod J 1995;28: 100.
25. Bergeron BE, Murchison DF, Schindler WG, et al: *Effect of ultrasonic vibration and various sealer and cement combinations on titanium post removal*, J Endod 2001;27: 13.
26. Bertrand MF, Pellegrino JC, Rocca JP, et al: *Removal of thermafil root canal filling material*, J Endod 1997;23: 54.
27. Betti LV, Bramante CM. *Quantec SC rotary instruments versus hand files for gutta-percha removal in root canal retreatment*, Int Endod J 2001;34: 514.
28. Bhaskar SN. *Periapical lesion - types, incidence, and clinical features*, Oral Surg 1966;21: 657.
29. Bhaskar SN, Rappaport HM. *Histologic evaluation of endodontic procedures in dogs*, Oral Surg Oral Med Oral Pathol 1971;31: 526.
30. Block RM, Lewis RD, Hirsch J, et al: *Systemic distribution of N2 paste containing 14C paraformaldehyde following root canal therapy in dogs*, Oral Surg Oral Med Oral Pathol 1980;50: 350.
31. Brady JM, del Rio CE. *Corrosion of endodontic silver cones in humans: a scanning electron microscope and X-ray microprobe study*, J Endod 1975;1: 205.
32. Bramante CM, Betti LV. *Efficacy of Quantec rotary instruments for gutta-percha removal*, Int Endod J

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www.konkur.in

2000;33: 463.
33. Brodin P. *Neurotoxic and analgesic effects of root canal cements and pulp-protecting dental materials*, Endod Dent Traumatol 1988;4: 1.
34. Budd JC, Gekelman D, White JM. *Temperature rise of the post and on the root surface during ultrasonic post removal*, Int Endod J 2005;38: 705.
35. Buhler H. *Evaluation of root-resected teeth. Results after 10 years*, J Periodontol 1988;59: 805.
36. Buoncristiani J, Seto BG, Caputo AA. *Evaluation of ultrasonic and sonic instruments for intraradicular post removal*, J Endod 1994;20: 486.
37. Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 1985;1: 170.
38. Capar ID, Uysal B, Ok E, et al: *Effect of the size of the apical enlargement with rotary instruments, single-cone filling, post space preparation with drills, fiber post removal, and root canal filling removal on apical crack initiation and propagation*, J Endod 2015;41: 253.
39. Caronna V, Himel V, Yu Q, et al: *Comparison of the surface hardness among 3 materials used in an experimental apexification model under moist and dry environments*, J Endod 2014;40: 986.
40. Castrisos T, Abbott PV. *A survey of methods used for post removal in specialist endodontic practice*, Int Endod J 2002;35: 172.
41. Castrisos TV, Palamara JE, Abbott PV. *Measurement of strain on tooth roots during post removal with the Eggler post remover*, Int Endod J 2002;35: 337.

Telegram: @dental_k

www.konkur.in

42. Chenail BL, Teplitsky PE. *Orthograde ultrasonic retrieval of root canal obstructions*, J Endod 1987;13: 186.
43. Chugal NM, Clive JM, Spangberg LS. *Endodontic infection: some biologic and treatment factors associated with outcome*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 81.
44. Chutich MJ, Kaminski EJ, Miller DA, et al: *Risk assessment of the toxicity of solvents of gutta-percha used in endodontic retreatment*, J Endod 1998;24: 213.
45. Cohen AS, Brown DC. *Orofacial dental pain emergencies: Endodontic diagnosis and management*, Cohen S Burns RC Pathways of the pulp 8 2002; Mosby St. Louis 31.
46. Cohen S, Liewehr F. *Diagnostic procedures*, Cohen S Burns RC Pathways of the pulp ed 8 2002; Mosby St. Louis 3.
47. Cohen S, Schwartz S. *Endodontic complications and the law*, J Endod 1987;13: 191.
48. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J Endod 2007;33: 1121.
49. Crump MC, Natkin E. *Relationship of broken root canal instruments to endodontic case prognosis: a clinical investigation*, J Am Dent Assoc 1970;80: 1341.
50. Cunha RS, De Martin AS, Barros PP, et al: *In vitro evaluation of the cleansing working time and analysis of the amount of gutta-percha or Resilon remnants in the root canal walls after instrumentation for endodontic retreatment*, J Endod 2007;33: 1426.
51. da Silveira PF, Vizzotto MB, Liedke GS, et al: *Detection of vertical root fractures by conventional radiographic examination and cone beam computed tomography - an in*

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www.konkur.in

vitro analysis, Dent Traumatol 2013;29: 41.
52. de Oliveira DP, Barbizam JV, Trope M, et al: *Comparison between gutta-percha and resilon removal using two different techniques in endodontic retreatment*, J Endod 2006;32: 362.
53. de Rijk WG. *Removal of fiber posts from endodontically treated teeth*, Am J Dent 2000;13: 19B.
54. de Siqueira Zuolo A, Zuolo ML, da Silveira Bueno CE, et al: *Evaluation of the efficacy of TRUShape and reciproc file systems in the removal of root filling material: an ex vivo micro-computed tomographic study*, J Endod 2016;42: 315.
55. Dominici JT, Clark S, Scheetz J, et al: *Analysis of heat generation using ultrasonic vibration for post removal*, J Endod 2005;31: 301.
56. Dragoo MR. *Resin-ionomer and hybrid-ionomer cements: part II, human clinical and histologic wound healing responses in specific periodontal lesions*, Int J Periodontics Restorative Dent 1997;17: 75.
57. Edlund M, Nair MK, Nair UP. *Detection of vertical root fractures by using cone-beam computed tomography: a clinical study*, J Endod 2011;37: 768.
58. Ee J, Fayad MI, Johnson BR. *Comparison of endodontic diagnosis and treatment planning decisions using cone-beam volumetric tomography versus periapical radiography*, J Endod 2014;40: 910.
59. Endodontic Treatment Statistics 2007; American Association of Endodontists Chicago.
60. Erdemir A, Eldeniz AU, Belli S. *Effect of gutta-percha solvents on mineral contents of human root dentin using ICP-AES technique*, J Endod 2004;30: 54.
61. Erdemir A, Eldeniz AU, Belli S, et al: *Effect of solvents*

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www.konkur.in

on bonding to root canal dentin [abstract], J Dent Res (Spec Iss A) 2002;81: 241.

62. Eriksson AR, Albrektsson T. *Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit*, J Prosthet Dent 1983;50: 101.
63. Estrela C, Bueno MR, De Alencar AH, et al: *Method to evaluate inflammatory root resorption by using cone beam computed tomography*, J Endod 2009;35: 1491.
64. Ettrich CA, Labossiere PE, Pitts DL, et al: *An investigation of the heat induced during ultrasonic post removal*, J Endod 2007;33: 1222.
65. Ezzie E, Fleury A, Solomon E, et al: *Efficacy of retreatment techniques for a resin-based root canal obturation material*, J Endod 2006;32: 341.
66. Farzaneh M, Abitbol S, Friedman S. *Treatment outcome in endodontics: the Toronto study. Phases I and II: Orthograde retreatment*, J Endod 2004;30: 627.
67. Fava LR, Dummer PM. *Periapical radiographic techniques during endodontic diagnosis and treatment*, Int Endod J 1997;30: 250.
68. Ferreira JJ, Rhodes JS, Ford TR. *The efficacy of gutta-percha removal using ProFiles*, Int Endod J 2001;34: 267.
69. Ferris DM, Baumgartner JC. *Perforation repair comparing two types of mineral trioxide aggregate*, J Endod 2004;30: 422.
70. Fors UG, Berg JO. *Endodontic treatment of root canals obstructed by foreign objects*, Int Endod J 1986;19: 2.
71. Frajlich SR, Goldberg F, Massone EJ, et al: *Comparative study of retreatment of Thermafil and lateral condensation endodontic fillings*, Int Endod J 1998;31: 354.
72. Friedman S. *Treatment outcome and prognosis of*

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www.konkur.in

endodontic therapy, Orstavik D Pitt-Ford TR Essential endodontology. Prevention and treatment of apical periodontitis 1998; Blackwell Science Ltd. London 367.

73. Friedman S. *Orthograde retreatment*, Walton RE Torabinejad M Principles and practice of endodontics ed 3 2002; W.B. Saunders Philadelphia 345.
74. Friedman S, Abitbol S, Lawrence HP. *Treatment outcome in endodontics: The Toronto study. Phase 1: Initial treatment*, J Endod 2003;29: 787.
75. Friedman S, Mor C. *The success of endodontic therapy - Healing and functionality*, J Calif Dent Assoc 2004;32: 493.
76. Friedman S, Moshonov J, Trope M. *Efficacy of removing glass ionomer cement, zinc oxide eugenol, and epoxy resin sealers from retreated root canals*, Oral Surg Oral Med Oral Pathol 1992;73: 609.
77. Friedman S, Stabholz A. *Endodontic retreatment—case selection and technique. Part 1: Criteria for case selection*, J Endod 1986;12: 28.
78. Friedman S, Stabholz A, Tamse A. *Endodontic retreatment—case selection and technique. 3. Retreatment techniques*, J Endod 1990;16: 543.
79. Fristad I, Molven O, Halse A. *Nonsurgically retreated root-filled teeth - radiographic findings after 20 - 27 years*, Int Endod J 2004;37: 12.
80. Fukushima H, Yamamoto K, Hirohata K, et al: *Localization and identification of root canal bacteria in clinically asymptomatic periapical pathosis*, J Endod 1990;16: 534.
81. Gambrel MG, Hartwell GR, Moon PC, et al: *The effect of endodontic solutions on resorcinol-formalin paste in teeth*, J Endod 2005;31: 25.

Telegram: @dental_k

www.konkur.in

82. Garg H, Grewal MS. *Cone-beam computed tomography volumetric analysis and comparison of Dentin structure loss after retrieval of separated instrument by using Ultrasonic EMS and ProUltra tips*, J Endod 2016;42: 1693.
83. Garrido ADB, Fonseca TS, Alfredo E, et al: *Influence of ultrasound, with and without water spray cooling, on removal of posts cemented with resin or zinc phosphate cements*, J Endod 2004;30: 173.
84. Gernhardt CR, Eppendorf K, Kozlowski A, et al: *Toxicity of concentrated sodium hypochlorite used as an endodontic irrigant*, Int Endod J 2004;37: 272.
85. Gesi A, Magnolfi S, Goracci C, et al: *Comparison of two techniques for removing fiber posts*, J Endod 2003;29: 580.
86. Gettleman BH, Spriggs KA, ElDeeb ME, et al: *Removal of canal obstructions with the Endo extractor*, J Endod 1991;17: 608.
87. Giuliani V, Cocchetti R, Pagavino G. *Efficacy of ProTaper universal retreatment files in removing filling materials during root canal retreatment*, J Endod 2008;34: 1381.
88. Glick DH, Frank AL. *Removal of silver points and fractured posts by ultrasonics*, J Prosthet Dent 1986;55: 212.
89. Glickman GN, Dumsha TC. *Problems in canal cleaning and shaping*, Gutmann JL Dumsha TC Lovdahl PE. Problem solving in endodontics. Prevention, identification, and management ed 3 1997; Mosby St. Louis 91.
90. Glickman GN, Pileggi R. *Preparation for treatment*, Cohen S Burns RC Pathways of the pulp 8 2002; Mosby St. Louis 103.

Telegram: @dental_k

www.konkur.in

91. Gluskin AH, Peters CI, Wong RDM, et al: *Retreatment of non-healing endodontic therapy and management of mishaps*, Ingle JI Bakland LK Baumgartner JC Endodontics ed 6 2008; BC Decker Hamilton 1088.
92. Gluskin AH, Ruddle CJ, Zinman EJ. *Thermal injury through intraradicular heat transfer using ultrasonic devices: precautions and practical preventive strategies*, J Am Dent Assoc 2005;136: 1286.
93. Goldberg RA, Kuttler S, Dorn SO. *The properties of Endocal 10 and its potential impact on the structural integrity of the root*, J Endod 2004;30: 159.
94. Gomes AP, Kubo CH, Santos RA, et al: *The influence of ultrasound on the retention of cast posts cemented with different agents*, Int Endod J 2001;34: 93.
95. Goodacre CJ, Bernal G, Rungcharassaeng K, et al: *Clinical complications with implants and implant prostheses*, J Prosthet Dent 2003;90: 121.
96. Goodacre CJ, Kan JY, Rungcharassaeng K. *Clinical complications of osseointegrated implants*, J Prosthet Dent 1999;81: 537.
97. Gound TG, Marx D, Schwandt NA. *Incidence of flare-ups and evaluation of quality after retreatment of resorcinol-formaldehyde resin ('Russian Red Cement') endodontic therapy*, J Endod 2003;29: 624.
98. Grobecker-Karl T, Christian M, Karl M. *Effect of endodontic access cavity preparation on monolithic and ceramic veneered zirconia restorations*, Quintessence Int 2016;47: 725.
99. Gutierrez JH, Villena F, Gigoux C, et al: *Microscope and scanning electron microscope examination of silver points corrosion caused by endodontic materials*, J Endod 1982;8: 301.

Telegram: @dental_k

www.konkur.in

100. Gutmann JL, Harrison JW. Surgical endodontics ed 2 1994; Ishiyaku EuroAmerica, p 468 St. Louis.
101. Gutmann JL, Lovdahl PE. *Problems in the assessment of success and failure, quality assurance, and their integration into endodontic treatment planning*, Gutmann JL Dumsha TC Lovdahl PE. Problem solving in endodontics. Prevention, identification, and management ed 3 1997; Mosby St. Louis 1.
102. Gutmann JL, Witherspoon DE. *Obturation of the cleaned and shaped root canal system*, Cohen S Burns RC Pathways of the pulp 2002; Mosby St. Louis 293.
103. Hammad M, Qualtrough A, Silikas N. *Three-dimensional evaluation of effectiveness of hand and rotary instrumentation for retreatment of canals filled with different materials*, J Endod 2008;34: 1370.
104. Hansen MG. *Relative efficiency of solvents used in endodontics*, J Endod 1998;24: 38.
105. Haselton DR, Lloyd PM, Johnson WT. *A comparison of the effects of two burs on endodontic access in all-ceramic high lucite crowns*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 486.
106. Hashem AA. *Ultrasonic vibration: temperature rise on external root surface during broken instrument removal*, J Endod 2007;33: 1070.
107. Hassan B, Metska ME, Ozok AR, et al: *Detection of vertical root fractures in endodontically treated teeth by a cone beam computed tomography scan*, J Endod 2009;35: 719.
108. Hassanloo A, Watson P, Finer Y, et al: *Retreatment efficacy of the Epiphany soft resin obturation system*, Int Endod J 2007;40: 633.
109. Hauman CHJ, Chandler NP, Purton DG. *Factors*

Telegram: @dental_k

www.konkur.in

influencing the removal of posts, Int Endod J 2003;36: 687.

110. He J, White RK, White CA, et al: *Clinical and patient-centered outcomes of nonsurgical root canal retreatment in first molars using contemporary techniques*, J Endod 2017;43: 231.
111. Hiraishi N, Yau JY, Loushine RJ, et al: *Susceptibility of a polycaprolactone-based root canal-filling material to degradation. III. Turbidimetric evaluation of enzymatic hydrolysis*, J Endod 2007;33: 952.
112. Holland R, De Souza V, Nery MJ, et al: *Tissue reactions following apical plugging of the root canal with infected dentin chips. A histologic study in dogs' teeth*, Oral Surg Oral Med Oral Pathol 1980;49: 366.
113. Holland R, Filho JA, de Souza V, et al: *Mineral trioxide aggregate repair of lateral root perforations*, J Endod 2001;27: 281.
114. Horan BB, Tordik PA, Imamura G, et al: *Effect of dentin thickness on root surface temperature of teeth undergoing ultrasonic removal of posts*, J Endod 2008;34: 453.
115. Hulsmann M. *Removal of fractured instruments using a combined automated/ultrasonic technique*, J Endod 1994;20: 144.
116. Hulsmann M, Bluhm V. *Efficacy, cleaning ability and safety of different rotary NiTi instruments in root canal retreatment*, Int Endod J 2004;37: 468.
117. Hulsmann M, Stotz S. *Efficacy, cleaning ability and safety of different devices for gutta- percha removal in root canal retreatment*, Int Endod J 1997;30: 227.
118. Hunter KR, Doblecki W, Pelleu GB Jr. *Halothane and eucalyptol as alternatives to chloroform for softening gutta-percha*, J Endod 1991;17: 310.

Telegram: @dental_k

www.konkur.in

119. Huttula AS, Tordik PA, Imamura G, et al: *The effect of ultrasonic post instrumentation on root surface temperature*, J Endod 2006;32: 1085.
120. Ibarrola JL, Knowles KI, Ludlow MO. *Retrievability of Thermafil plastic cores using organic solvents*, J Endod 1993;19: 417.
121. Imura N, Kato AS, Hata GI, et al: *A comparison of the relative efficacies of four hand and rotary instrumentation techniques during endodontic retreatment*, Int Endod J 2000;33: 361.
122. Imura N, Zuolo ML, Ferreira MO, et al: *Effectiveness of the canal finder and hand instrumentation in removal of gutta-percha root fillings during root canal retreatment*, Int Endod J 1996;29: 382.
123. Imura N, Zuolo ML, Kherlakian D. *Comparison of endodontic retreatment of laterally condensed gutta-percha and Thermafil with plastic carriers*, J Endod 1993;19: 609.
124. Ingle JI, Heithersay GS, Hartwell GR, et al: *Endodontic diagnostic procedures*, Ingle JI Bakland LK Endodontics ed 5 2002; BC Decker Hamilton 203.
125. Ingle JI, Simon JH, Machtou P, et al: *Outcome of endodontic treatment and retreatment*, Ingle JI Bakland LK Endodontics ed 5 2002; BC Decker Hamilton 747.
126. Iqbal MK, Johansson AA, Akeel RF, et al: *A retrospective analysis of factors associated with the periapical status of restored, endodontically treated teeth*, Int J Prosthodont 2003;16: 31.
127. Iqbal MK, Kohli MR, Kim JS. *A retrospective clinical study of incidence of root canal instrument separation in an endodontics graduate program: a PennEndo database study*, J Endod 2006;32: 1048.

Telegram: @dental_k

www.konkur.in

128. Iqbal MK, Rafailov H, Kratchman SI, et al: *A comparison of three methods for preparing centered platforms around separated instruments in curved canals*, J Endod 2006;32: 48.
129. Jafarzadeh H, Abbott PV. *Ledge formation: review of a great challenge in endodontics*, J Endod 2007;33: 1155.
130. Jeng HW, ElDeeb ME. *Removal of hard paste fillings from the root canal by ultrasonic instrumentation [published erratum appears in J Endod 1987;13(12):565]*, J Endod 1987;13: 295.
131. Jew RC, Weine FS, Keene JJ Jr, et al: *A histologic evaluation of periodontal tissues adjacent to root perforations filled with Cavit*, Oral Surg Oral Med Oral Pathol 1982;54: 124.
132. Johnson WT, Leary JM, Boyer DB. *Effect of ultrasonic vibration on post removal in extracted human premolar teeth*, J Endod 1996;22: 487.
133. Kang M, In Jung H, Song M, et al: *Outcome of nonsurgical retreatment and endodontic microsurgery: a meta-analysis*, Clin Oral Investig 2015;19: 569.
134. Kaplowitz GJ. *Using rectified turpentine oil in endodontic retreatment*, J Endod 1996;22: 621.
135. Kaufman D, Mor C, Stabholz A, et al: *Effect of gutta-percha solvents on calcium and phosphorus levels of cut human dentin*, J Endod 1997;23: 614.
136. Khedmat S, Rouhi N, Drage N, et al: *Evaluation of three imaging techniques for the detection of vertical root fractures in the absence and presence of gutta-percha root fillings*, Int Endod J 2012;45: 1004.
137. Kim E, Lee SJ. *Electronic apex locator*, Dent Clin North Am 2004;48: 35.
138. Koch K. *The microscope. Its effect on your practice*, Dent

Telegram: @dental_k

www.konkur.in

Clin North Am 1997;41: 619.

139. Koppang HS, Koppang R, Solheim T, et al: *Cellulose fibers from endodontic paper points as an etiological factor in postendodontic periapical granulomas and cysts*, J Endod 1989;15: 369.
140. Krell KV, Neo J. *The use of ultrasonic endodontic instrumentation in the re-treatment of a paste-filled endodontic tooth*, Oral Surg Oral Med Oral Pathol 1985;60: 100.
141. Ladley RW, Campbell AD, Hicks ML, et al: *Effectiveness of halothane used with ultrasonic or hand instrumentation to remove gutta-percha from the root canal*, J Endod 1991;17: 221.
142. Langer B, Stein SD, Wagenberg B. *An evaluation of root resections. A ten-year study*, J Periodontol 1981;52: 719.
143. Lantz B, Persson PA. *Periodontal tissue reactions after root perforations in dog's teeth. A histologic study*, Odontol Tidskr 1967;75: 209.
144. Lawley GR, Schindler WG, Walker WA, et al: *Evaluation of ultrasonically placed MTA and fracture resistance with intracanal composite resin in a model of apexification*, J Endod 2004;30: 167.
145. Lazarski MP, Walker WA III, Flores CM, et al: *Epidemiological evaluation of the outcomes of nonsurgical root canal treatment in a large cohort of insured dental patients*, J Endod 2001;27: 791.
146. Lee FS, Van Cura JE, BeGole E. *A comparison of root surface temperatures using different obturation heat sources*, J Endod 1998;24: 617.
147. Lemon RR. *Nonsurgical repair of perforation defects. Internal matrix concept*, Dent Clin North Am 1992;36: 439.

Telegram: @dental_k

www.konkur.in

148. Lemon RR, Steele PJ, Jeansonne BG. *Ferric sulfate hemostasis: effect on osseous wound healing. Left in situ for maximum exposure*, J Endod 1993;19: 170.
149. Leonard JE, Gutmann JL, Guo IY. *Apical and coronal seal of roots obturated with a dentine bonding agent and resin*, Int Endod J 1996;29: 76.
150. Levin A, Shemesh A, Katzenell V, et al: *Use of cone-beam computed tomography during retreatment of a 2-rooted maxillary central incisor: case report of a complex diagnosis and treatment*, J Endod 2015;41: 2064.
151. Liang YH, Jiang L, Chen C, et al: *The validity of cone-beam computed tomography in measuring root canal length using a gold standard*, J Endod 2013;39: 1607.
152. Lin LM, Skribner JE, Gaengler P. *Factors associated with endodontic treatment failures*, J Endod 1992;18: 625.
153. Lindemann M, Yaman P, Dennison JB, et al: *Comparison of the efficiency and effectiveness of various techniques for removal of fiber posts*, J Endod 2005;31: 520.
154. Lipski M, Wozniak K. *In vitro infrared thermographic assessment of root surface temperature rises during thermafil retreatment using system B*, J Endod 2003;29: 413.
155. Lovdahl PE. *Endodontic retreatment*, Dent Clin North Am 1992;36: 473.
156. Lovdahl PE, Gutmann JL. *Problems in nonsurgical root canal retreatment*, Gutmann JL Dumsha TC Lovdahl PE. Problem solving in endodontics. Prevention, identification, and management ed 3 1997; Mosby St. Louis 157.
157. Ludlow JB. *Dose and risk in dental diagnostic imaging: with emphasis on dosimetry of CBCT*, Korean J Oral Maxillofac Radiol 2009;39: 175.
158. Luks S. *Gutta percha vs. silver points in the practice of*

Telegram: @dental_k

www.konkur.in

endodontics, N Y State Dent J 1965;31: 341.

159. Maalouf EM, Gutmann JL. *Biological perspectives on the non-surgical endodontic management of periradicular pathosis*, Int Endod J 1994;27: 154.
160. Machtou P, Sarfati P, Cohen AG. *Post removal prior to retreatment*, J Endod 1989;15: 552.
161. Madarati AA, Qualtrough AJ, Watts DC. *Factors affecting temperature rise on the external root surface during ultrasonic retrieval of intracanal separated files*, J Endod 2008;34: 1089.
162. Madison S, Swanson K, Chiles SA. *An evaluation of coronal microleakage in endodontically treated teeth. Part II. Sealer types*, J Endod 1987;13: 109.
163. Main C, Mirzayan N, Shabahang S, et al: *Repair of root perforations using mineral trioxide aggregate: a long-term study*, J Endod 2004;30: 80.
164. Mancini M, Palopoli P, Iorio L, et al: *Accuracy of an electronic apex locator in the retreatment of teeth obturated with plastic or cross-linked gutta-percha carrier-based materials: an ex vivo study*, J Endod 2014;40: 2061.
165. Mandel E, Friedman S. *Endodontic retreatment: a rational approach to root canal reinstrumentation*, J Endod 1992;18: 565.
166. Martin JA, Bader JD. *Five-year treatment outcomes for teeth with large amalgams and crowns*, Oper Dent 1997;22: 72.
167. Mason AG, Sutton A, Turkyilmaz I. *An investigation of heat transfer to the implant-bone interface when drilling through a zirconia crown attached to a titanium or zirconia abutment*, J Prosthet Dent 2014;112: 1119.
168. Masserann J. *Entfernen metallischer Fragmente aus*

Telegram: @dental_k

www.konkur.in

Wurzelkanalen (Removal of metal fragments from the root canal), J Br Endod Soc 1971;5: 55.

169. Matherne RP, Angelopoulos C, Kulild JC, et al: *Use of cone-beam computed tomography to identify root canal systems in vitro*, J Endod 2008;34: 87.
170. McDonald MN, Vire DE. *Chloroform in the endodontic operatory*, J Endod 1992;18: 301.
171. Messer HH. *Permanent restorations and the dental pulp*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Books Chicago 345.
172. Metska ME, Aartman IH, Wesselink PR, et al: *Detection of vertical root fractures in vivo in endodontically treated teeth by cone-beam computed tomography scans*, J Endod 2012;38: 1344.
173. Metzger Z, Ben-Amar A. *Removal of overextended gutta-percha root canal fillings in endodontic failure cases*, J Endod 1995;21: 287.
174. Molven O, Halse A, Fristad I, et al: *Periapical changes following root-canal treatment observed 20–27 years postoperatively*, Int Endod J 2002;35: 784.
175. Moshonov J, Peretz B, Ben-Zvi K, et al: *Effect of gutta-percha solvents on surface microhardness of IRM fillings*, J Endod 2000;26: 142.
176. Moshonov J, Trope M, Friedman S. *Retreatment efficacy 3 months after obturation using glass ionomer cement, zinc oxide-eugenol, and epoxy resin sealers*, J Endod 1994;20: 90.
177. Mota de Almeida FJ, Knutsson K, Flygare L. *The impact of cone beam computed tomography on the choice of endodontic diagnosis*, Int Endod J 2015;48: 564.
178. Mozynska J, Metlerski M, Lipski M, et al: *Tooth discoloration induced by different calcium silicate-based*

Telegram: @dental_k

www.konkur.in

cements: a systematic review of in vitro studies, J Endod 2017;43: 1593.

179. Mulvay PG, Abbott PV. *The effect of endodontic access cavity preparation and subsequent restorative procedures on molar crown retention*, Aust Dent J 1996;41: 134.
180. Nagai O, Tani N, Kayaba Y, et al: *Ultrasonic removal of broken instruments in root canals*, Int Endod J 1986;19: 298.
181. Nair PN. *New perspectives on radicular cysts: do they heal?*, Int Endod J 1998;31: 155.
182. Nair PN, Sjogren U, Krey G, et al: *Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study*, J Endod 1990;16: 580.
183. Nair PN, Sjogren U, Krey G, et al: *Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth*, J Endod 1990;16: 589.
184. Nair PN, Sjogren U, Schumacher E, et al: *Radicular cyst affecting a root-filled human tooth: a long-term post-treatment follow-up*, Int Endod J 1993;26: 225.
185. Nair PNR, Schroeder HE. *Periapical actinomycosis*, J Endod 1984;10: 567.
186. Nakata TT, Bae KS, Baumgartner JC. *Perforation repair comparing mineral trioxide aggregate and amalgam using an anaerobic bacterial leakage model*, J Endod 1998;24: 184.
187. Nearing MV, Glickman GN. *Comparitive efficacy of various rotary instrumentation systems for gutta-percha removal [Abstract]*, J Endod 1999;24: 295.
188. Ng YL, Mann V, Gulabivala K. *Outcome of secondary root canal treatment: a systematic review of the literature*,

Telegram: @dental_k

www.konkur.in

Int Endod J 2008;41: 1026.
189. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health*, Int Endod J 2011;44: 583.
190. Niemczyk SP. *Re-inventing intentional replantation: a modification of the technique*, Pract Proced Aesthet Dent 2001;13: 433.
191. Niemi TK, Marchesan MA, Lloyd A, et al: *Effect of instrument design and access outlines on the removal of root canal obturation materials in oval-shaped canals*, J Endod 2016;42: 1550.
192. Nudera WJ. *Selective root retreatment: a novel approach*, J Endod 2015;41: 1382.
193. Osorio RM, Hefti A, Vertucci FJ, et al: *Cytotoxicity of endodontic materials*, J Endod 1998;24: 91.
194. Ozgoz M, Yagiz H, Cicek Y, et al: *Gingival necrosis following the use of a paraformaldehyde-containing paste: a case report*, Int Endod J 2004;37: 157.
195. Parashos P, Messer HH. *Questionnaire survey on the use of rotary nickel-titanium endodontic instruments by Australian dentists*, Int Endod J 2004;37: 249.
196. Parker H, Glickman GN. *Solubility of plastic Thermafil carriers*, J Dent Res 1993;72: 188.
197. Patel S. *New dimensions in endodontic imaging: Part 2. Cone beam computed tomography*, Int Endod J 2009;42: 463.
198. Patel S, Dawood A, Ford TP, et al: *The potential applications of cone beam computed tomography in the management of endodontic problems*, Int Endod J 2007;40: 818.
199. Perez AL, Spears R, Gutmann JL, et al: *Osteoblasts and*

Telegram: @dental_k

www.konkur.in

MG-63 osteosarcoma cells behave differently when in contact with ProRoot MTA and White MTA, Int Endod J 2003;36: 564.

200. Peters SB, Canby FL, Miller DA. *Removal of a carbon-fiber post system [abstract PR35]*, J Endod 1996;22: 215.
201. Pitt-Ford TR, Torabinejad M, McKendry DJ, et al: *Use of mineral trioxide aggregate for repair of furcal perforations*, Oral Surg Oral Med Oral Pathol 1995;79: 756.
202. Ray H, Seltzer S. *A new glass ionomer root canal sealer*, J Endod 1991;17: 598.
203. Ray HA, Trope M. *Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration*, Int Endod J 1995;28: 12.
204. Regezi JA, Sciubba JJ. *Cysts of the oral region*, Regezi JA Sciubba JJ Oral pathology. Clinical pathologic correlations ed 3 1999; WB Saunders Philadelphia 288.
205. Ricucci D, Siqueira JF Jr, Lopes WS, et al: *Extraradicular infection as the cause of persistent symptoms: a case series*, J Endod 2015;41: 265.
206. Rocas IN, Siqueira JFJ, Santos KR. *Association of enterococcus faecalis with different forms of periradicular diseases*, J Endod 2004;30: 315.
207. Roda R. *Clinical showcase - unintentional replantation: a technique to avoid*, J Can Dent Assoc 2006;72: 133.
208. Roda RS. *Root perforation repair: surgical and nonsurgical management*, Pract Proced Aesthet Dent 2001;13: 467.
209. Roda RS. *Can use of cone beam computed tomography have an effect on endodontic treatment?*, J Calif Dent Assoc 2018;46: 237.

Telegram: @dental_k

www.konkur.in

210. Rodriguez G, Patel S, Duran-Sindreu F, et al: *Influence of cone-beam computed tomography on endodontic retreatment strategies among general dental practitioners and endodontists*, J Endod 2017;43: 1433.
211. Roig-Greene JL. *The retrieval of foreign objects from root canals: a simple aid*, J Endod 1983;9: 394.
212. Rossi-Fedele G, Ahmed HM. *Assessment of root canal filling removal effectiveness using micro-computed tomography: a systematic review*, J Endod 2017;43: 520.
213. Royzenblat A, Goodell GG. *Comparison of removal times of Thermafil plastic obturators using ProFile rotary instruments at different rotational speeds in moderately curved canals*, J Endod 2007;33: 256.
214. Ruddle CJ. *Non-surgical endodontic retreatment*, Cohen S Burns RC Pathways of the pulp ed 8 2002; Mosby St. Louis 875.
215. Ruddle CJ. *Nonsurgical retreatment*, J Endod 2004;30: 827.
216. Sabourin CR, Flinn BD, Pitts DL, et al: *A novel method for creating endodontic access preparations through all-ceramic restorations: air abrasion and its effect relative to diamond and carbide bur use*, J Endod 2005;31: 616.
217. Sae-Lim V, Rajamanickam I, Lim BK, et al: *Effectiveness of ProFile.04 taper rotary instruments in endodontic retreatment*, J Endod 2000;26: 100.
218. Sakkal S, Gauthier G, Milot P, et al: *A clinical appraisal of the Gonon post-pulling system*, J Can Dent Assoc 1994;60: 537.
219. Sanchez-Sanhueza G, Bello-Toledo H, Gonzalez-Rocha G, et al: *Metagenomic study of bacterial microbiota in persistent endodontic infections using next-generation sequencing*, Int Endod J 2018;51: 1336.

Telegram: @dental_k

www.konkur.in

220. Satterthwaite JD, Stokes AN, Frankel NT. *Potential for temperature change during application of ultrasonic vibration to intra-radicular posts*, Eur J Prosthodont Restor Dent 2003;11: 51.
221. Saunders EM. *In vivo findings associated with heat generation during thermomechanical compaction of gutta-percha. 1. Temperature levels at the external surface of the root*, Int Endod J 1990;23: 263.
222. Saunders EM. *In vivo findings associated with heat generation during thermomechanical compaction of gutta-percha. 2. Histological response to temperature elevation on the external surface of the root*, Int Endod J 1990;23: 268.
223. Saunders EM, Saunders WP. *The heat generated on the external root surface during post space preparation*, Int Endod J 1989;22: 169.
224. Saunders WP, Saunders EM. *Coronal leakage as a cause of failure in root-canal therapy: a review*, Endod Dent Traumatol 1994;10: 105.
225. Scherer W, Dragoo MR. *New subgingival restorative procedures with Geristore resin ionomer*, Pract Periodontics Aesthet Dent 1995;7: 1.
226. Schilder H. *Filling root canals in three dimensions*, Dent Clin North Am 1967;723:.
227. Schirrmeister JF, Meyer KM, Hermanns P, et al: *Effectiveness of hand and rotary instrumentation for removing a new synthetic polymer-based root canal obturation material (Epiphany) during retreatment*, Int Endod J 2006;39: 150.
228. Schwandt NW, Gound TG. *Resorcinol-formaldehyde resin 'Russian Red' endodontic therapy*, J Endod 2003;29: 435.

Telegram: @dental_k

www.konkur.in

229. Schwartz RS, Robbins JW. *Post placement and restoration of endodontically treated teeth: a literature review*, J Endod 2004;30: 289.
230. Seltzer S. Endodontology: Biologic considerations in endodontic procedures ed 2 1988; Lea & Febiger Philadelphia x.
231. Seltzer S, Bender IB, Ziontz BA. *The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp*, Oral Surg 1963;16: 846.
232. Seltzer S, Bender IB. *Cognitive dissonance in endodontics*, Oral Surg Oral Med Oral Pathol 1965;20: 505.
233. Seltzer S, Green DB, Weiner N, et al: *A scanning electron microscope examination of silver cones removed from endodontically treated teeth*, Oral Surg Oral Med Oral Pathol 1972;33: 589.
234. Seltzer S, Sinai I, August D. *Periodontal effects of root perforations before and during endodontic procedures*, J Dent Res 1970;49: 332.
235. Serper A, Ucer O, Onur R, et al: *Comparative neurotoxic effects of root canal filling materials on rat sciatic nerve*, J Endod 1998;24: 592.
236. Setzer FC, Shah SB, Kohli MR, et al: *Outcome of endodontic surgery: a meta-analysis of the literature—part 1: comparison of traditional root-end surgery and endodontic microsurgery*, J Endod 2010;36: 1757.
237. Shen Y, Peng B, Cheung GS. *Factors associated with the removal of fractured NiTi instruments from root canal systems*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 605.
238. Shipper G, Orstavik D, Teixeira FB, et al: *An evaluation*

Telegram: @dental_k

www.konkur.in

of microbial leakage in roots filled with a thermoplastic synthetic polymer-based root canal filling material (Resilon), J Endod 2004;30: 342.

239. Simon JH, Chimenti RA, Mintz GA. *Clinical significance of the pulse granuloma*, J Endod 1982;8: 116.
240. Simon JH, Glick DH, Frank AL. *The relationship of endodontic-periodontic lesions*, J Periodontol 1972;43: 202.
241. Sinai IH. *Endodontic perforations: their prognosis and treatment*, J Am Dent Assoc 1977;95: 90.
242. Siqueira JF, Sen BH. *Fungi in endodontic infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 632.
243. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498.
244. Sjogren U, Happonen RP, Kahnberg KE, et al: *Survival of Arachnia propionica in periapical tissue*, Int Endod J 1988;21: 277.
245. Sluyk SR, Moon PC, Hartwell GR. *Evaluation of setting properties and retention characteristics of mineral trioxide aggregate when used as a furcation perforation repair material*, J Endod 1998;24: 768.
246. So MV, Saran C, Magro ML, et al: *Efficacy of ProTaper retreatment system in root canals filled with gutta-percha and two endodontic sealers*, J Endod 2008;34: 1223.
247. Solomonov M, Paque F, Kaya S, et al: *Self-adjusting files in retreatment: a high-resolution micro-computed tomography study*, J Endod 2012;38: 1283.
248. Somma F, Cammarota G, Plotino G, et al: *The effectiveness of manual and mechanical instrumentation for the retreatment of three different root canal filling*

Telegram: @dental_k

www.konkur.in

materials, J Endod 2008;34: 466.
249. Song M, Nam T, Shin SJ, et al: *Comparison of clinical outcomes of endodontic microsurgery: 1 year versus long-term follow-up*, J Endod 2014;40: 490.
250. Souter NJ, Messer HH. *Complications associated with fractured file removal using an ultrasonic technique*, J Endod 2005;31: 450.
251. Spatafore CM, Griffin JA Jr, Keyes GG, et al: *Periapical biopsy report: an analysis of over a 10-year period*, J Endod 1990;16: 239.
252. Spili P, Parashos P, Messer HH. *The impact of instrument fracture on outcome of endodontic treatment*, J Endod 2005;31: 845.
253. Spriggs K, Gettleman B, Messer HH. *Evaluation of a new method for silver point removal*, J Endod 1990;16: 335.
254. Stabholz A, Friedman S. *Endodontic retreatment—case selection and technique. Part 2: treatment planning for retreatment*, J Endod 1988;14: 607.
255. Stamos DE, Gutmann JL. *Revisiting the post puller*, J Endod 1991;17: 466.
256. Stamos DE, Gutmann JL. *Survey of endodontic retreatment methods used to remove intraradicular posts*, J Endod 1993;19: 366.
257. Studebaker B, Hollender L, Mancl L, et al: *The incidence of second mesiobuccal canals located in maxillary molars with the aid of cone-beam computed tomography*, J Endod 2018;44: 565.
258. Sundqvist G. *Ecology of the root canal flora*, J Endod 1992;18: 427.
259. Sundqvist G, Figdor D. *Endodontic treatment of apical periodontitis*, Orstavik D Pitt-Ford TR Essential endodontology: prevention and treatment of apical

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periodontitis 1998; Blackwell Science Ltd. London 242.
260. Sundqvist G, Reuterving CO. *Isolation of Actinomyces Israelii from periapical lesion*, J Endod 1980;6: 602.
261. Suter B. *A new method for retrieving silver points and separated instruments from root canals*, J Endod 1998;24: 446.
262. Suter B, Lussi A, Sequeira P. *Probability of removing fractured instruments from root canals*, Int Endod J 2005;38: 112.
263. Sutherland JK, Teplitsky PE, Moulding MB. *Endodontic access of all-ceramic crowns*, J Prosthet Dent 1989;61: 146.
264. Swanson K, Madison S. *An evaluation of coronal microleakage in endodontically treated teeth. Part I. Time periods*, J Endod 1987;13: 56.
265. Talwar S, Utneja S, Nawal RR, et al: *Role of cone-beam computed tomography in diagnosis of vertical root fractures: a systematic review and meta-analysis*, J Endod 2016;42: 12.
266. Tamse A, Fuss Z, Lustig J, et al: *An evaluation of endodontically treated vertically fractured teeth*, J Endod 1999;25: 506.
267. Tamse A, Kaffe I, Lustig J, et al: *Radiographic features of vertically fractured endodontically treated mesial roots of mandibular molars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 797.
268. Tasdemir T, Er K, Yildirim T, et al: *Efficacy of three rotary NiTi instruments in removing gutta-percha from root canals*, Int Endod J 2008;41: 191.
269. Tasdemir T, Yildirim T, Celik D. *Comparative study of removal of current endodontic fillings*, J Endod 2008;34: 326.

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270. Teixeira FB, Teixeira EC, Thompson JY, et al: *Fracture resistance of roots endodontically treated with a new resin filling material*, J Am Dent Assoc 2004;135: 646.
271. Teplitsky PE, Rayner D, Chin I, et al: *Gutta percha removal utilizing GPX instrumentation*, J Can Dent Assoc 1992;58: 53.
272. Topcuoglu HS, Demirbuga S, Tuncay O, et al: *The bond strength of endodontic sealers to root dentine exposed to different gutta-percha solvents*, Int Endod J 2014;47: 1100.
273. Torabinejad M, Higa RK, McKendry DJ, et al: *Dye leakage of four root end filling materials: effects of blood contamination*, J Endod 1994;20: 159.
274. Torabinejad M, Hong C, Pitt Ford TR. *Tissue reaction to implanted Super-EBA and mineral trioxide aggregate in the mandible of guinea pigs: a preliminary report*, J Endod 1995;21: 569.
275. Torabinejad M, Hong CU, McDonald F, et al: *Physical and chemical properties of a new root-end filling material*, J Endod 1995;21: 349.
276. Torabinejad M, Hong CU, Pitt Ford TR, et al: *Cytotoxicity of four root end filling materials*, J Endod 1995;21: 489.
277. Torabinejad M, Lemon RR. *Procedural accidents*, Walton RE Torabinejad M Principles and practice of endodontics ed 3 2002; W.B. Saunders Philadelphia 310.
278. Torabinejad M, Ung B, Kettering JD. *In vitro bacterial penetration of coronally unsealed endodontically treated teeth*, J Endod 1990;16: 566.
279. Tronstad L, Barnett F, Cervone F. *Periapical bacterial plaque in teeth refractory to endodontic treatment*, Endod Dent Traumatol 1990;6: 73.
280. Trope M, Maltz DO, Tronstad L. *Resistance to fracture of*

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restored endodontically treated teeth, Endod Dent Traumatol 1985;1: 108.

281. Tsesis I, Faivishevsky V, Kfir A, et al: *Outcome of surgical endodontic treatment performed by a modern technique: a meta-analysis of literature*, J Endod 2009;35: 1505.
282. United States Drug Administration: *Chloroform used as an ingredient (active or inactive) in drug products*. Federal Register No. 26845, US Government Printing Office, Washington DC, 1976.
283. Uzun O, Topuz O, Tinaz C, et al: *Accuracy of two root canal length measurement devices integrated into rotary endodontic motors when removing gutta-percha from root-filled teeth*, Int Endod J 2008;41: 725.
284. Valderhaug J, Jokstad A, Ambjornsen E, et al: *Assessment of the periapical and clinical status of crowned teeth over 25 years*, J Dent 1997;25: 97.
285. Viducic D, Jukic S, Karlovic Z, et al: *removal of gutta-percha from root canals using an Nd:YAG laser*, Int Endod J 2003;36: 670.
286. Walton RE, Rivera EM. *Cleaning and shaping*, Walton RE Torabinjad M Principles and practice of endodontics ed 3 2002; Saunders Philadelphia 206.
287. Walton RE, Torabinejad M. *Diagnosis and treatment planning*, Walton RE Torabinejad M Principles and practice of endodontics ed 3 2002; W.B. Saunders Philadelphia 49.
288. Weiger R, Manncke B, Werner H, et al: *Microbial flora of sinus tracts and root canals of non-vital teeth*, Endod Dent Traumatol 1995;11: 15.
289. Welk ARDMD, Baumgartner JCDDSP, Marshall JGDMD. *An in vivo comparison of two frequency-based*

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electronic apex locators, J Endod 2003;29: 497.

290. Wennberg A, Orstavik D. *Evaluation of alternatives to chloroform in endodontic practice*, Endod Dent Traumatol 1989;5: 234.
291. White C, Bryant N. *Combined therapy of mineral trioxide aggregate and guided tissue regeneration in the treatment of external root resorption and an associated osseous defect*, J Periodontol 2002;73: 1517.
292. Whitworth JM, Boursin EM. *Dissolution of root canal sealer cements in volatile solvents*, Int Endod J 2000;33: 19.
293. Wilcox LR. *Thermafil retreatment with and without chloroform solvent*, J Endod 1993;19: 563.
294. Wilcox LR. *Endodontic retreatment with halothane versus chloroform solvent*, J Endod 1995;21: 305.
295. Wilcox LR, Juhlin JJ. *Endodontic retreatment of Thermafil versus laterally condensed gutta-percha*, J Endod 1994;20: 115.
296. Wilcox LR, Krell KV, Madison S, et al: *Endodontic retreatment: evaluation of gutta-percha and sealer removal and canal reinstrumentation*, J Endod 1987;13: 453.
297. Wohlgemuth P, Cuocolo D, Vandrangi P, et al: *Effectiveness of the GentleWave system in removing separated instruments*, J Endod 2015;41: 1895.
298. Wolcott J, Ishley D, Kennedy W, et al: *A 5 yr clinical investigation of second mesiobuccal canals in endodontically treated and retreated maxillary molars*, J Endod 2005;31: 262.
299. Wolcott JF, Himel VT, Hicks ML. *Thermafil retreatment using a new "System B" technique or a solvent*, J Endod 1999;25: 761.

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300. Wolcott S, Wolcott J, Ishley D, et al: *Separation incidence of protaper rotary instruments: a large cohort clinical evaluation*, J Endod 2006;32: 1139.
301. Wourms DJ, Campbell AD, Hicks ML, et al: *Alternative solvents to chloroform for gutta-percha removal*, J Endod 1990;16: 224.
302. Yatsushiro JD, Baumgartner JC, Tinkle JS. *Longitudinal study of the microleakage of two root-end filling materials using a fluid conductive system*, J Endod 1998;24: 716.
303. Yeo JF, Loh FC. *Retrograde removal of fractured endodontic instruments*, Ann Acad Med Singapore 1989;18: 594.
304. Yilmaz F, Kamburoglu K, Senel B. *Endodontic working length measurement using cone-beam computed tomographic images obtained at different voxel sizes and field of views, periapical radiography, and apex locator: a comparative ex vivo study*, J Endod 2017;43: 152.
305. Yoldas O, Oztunc H, Tinaz C, et al: *Perforation risks associated with the use of Masserann endodontic kit drills in mandibular molars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 513.
306. Zakariasen KL, Brayton SM, Collinson DM. *Efficient and effective root canal retreatment without chloroform*, J Can Dent Assoc 1990;56: 509.
307. Zinman EJ. *Records and legal responsibilities*, Cohen S Burns RC Pathways of the pulp ed 8 2002; Mosby St. Louis 365.
308. Zou X, Liu D, Yue L, et al: *The ability of cone-beam computerized tomography to detect vertical root fractures in endodontically treated and nonendodontically treated teeth: a report of 3 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111: 797.

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www.konkur.in

309. Zuolo ML, Imura N, Ferreira MO. *Endodontic retreatment of thermafil or lateral condensation obturations in post space prepared teeth*, J Endod 1994;20: 9.
310. Zuolo ML, Kherlakian N, Imura N. *Effectiveness of nickel titanium rotary and hand instrumentation in endodontic retreatment [Abstract]*, J Endod 1996;22: 209.

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11: Periradicular surgery

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CHAPTER OUTLINE

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Although nonsurgical endodontic treatment is a highly predictable option in most cases, surgery may be indicated for teeth with persistent periradicular pathoses that have not responded to nonsurgical approaches. Surgical root canal therapy, including root-end resection, has been practiced since at least the mid-1800s.[211] In 1906, Schamberg[473] described using radiographs to assist diagnosis and the use of surgical burs to perform a rapid osteotomy and root-end "ablation."

Perhaps the single most important development in dental practice in the

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early 20th century was the introduction of safe, effective local anesthesia, which allowed for more meticulous and comfortable surgical treatment. Formal recognition of endodontics as a specialty in 1963 ushered in a new era of basic and clinical research focused on the prevention and treatment of pulpal and periradicular pathosis.

Since the 1990s, periradicular surgery has continued to evolve into a precise, biologically based adjunct to nonsurgical root canal therapy. The parallel development of new instruments and materials, along with a better understanding of the biology of wound healing, has made surgical treatment a viable alternative to extraction and tooth replacement, rather than a treatment of last resort.

Periradicular surgery, when indicated, should be considered an extension of nonsurgical treatment, because the underlying etiology of the disease process and the objectives of treatment are the same: prevention or elimination of apical periodontitis. Surgical root canal treatment should not be considered as somehow separate from nonsurgical treatment, although the instruments and techniques are obviously quite different. Surgical treatment accounts for about 3% to 10% of the typical endodontic specialty practice.[1,75,374] A web-based survey found that 91% of active endodontists perform some type of root-end surgery, and almost all are using a dental operating microscope and ultrasonics.[115] Endodontists perform almost 78% of surgical root canal treatments; general dentists and other specialists perform 15.5% and 6.6%, respectively.[242] Although appropriately trained general dentists and other dental specialists may perform periradicular surgery, individuals with advanced training in endodontics have developed most of the current periradicular surgery techniques and science presented in this chapter. We believe that endodontists must continue to include periradicular surgery as a routine part of clinical endodontic practice and not abrogate this treatment option to others, who may not possess the same background, skills, or values.[343,424,439] When patient preferences and quality of life measures are considered, patients can be expected to place high value on endodontic treatment and the retention of a natural tooth.[153,180]

Indications for periradicular surgery

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Etiology of persistent periradicular disease

The first and arguably most important step in treatment decision making is attempting to determine the cause of persistent periradicular disease. Treatment then is directed at eliminating the etiology, which most often is the presence of bacteria and other microbial irritants in the root canal space.[492] Nonsurgical retreatment, when possible, is often the first choice for attempting to correct obvious deficiencies in the previous treatment.[564] However, microorganisms can survive even in apparently well-treated teeth in dentinal tubules, canal irregularities, deltas, and isthmus areas.[275,307,568] If residual microorganisms remain completely entombed in the root canal system, periradicular healing should occur. Sealing off all potential routes of microbial escape from the root canal system is the goal of both nonsurgical and surgical treatment. When microorganisms of sufficient pathogenicity and number gain access to the periradicular tissues, pathosis develops.

Enterococcus faecalis is commonly isolated from failing root canal–treated teeth and is known to be especially difficult to eliminate with standard instrumentation and irrigation techniques.[510] Unlike primary endodontic infections, which predominantly are associated with mixed anaerobic microbiota, it has been commonly accepted that treatment failures are more frequently associated with one or two microorganisms.[492] However, research using more sophisticated techniques (16S ribosomal RNA gene clone library analysis) has determined that multiple, previously uncultivated phylotypes can be identified in the majority of teeth with persistent apical periodontitis.[464] Fungi and viruses have also emerged as potential causes of root canal failure and may play either a primary or secondary role in persistent periradicular pathosis.[369,399,462,575]

Established extraradicular colonies of microorganisms also may be a reason for failure of some teeth to respond to nonsurgical treatment. When microorganisms are able to arrange in an extraradicular biofilm, they may be particularly resistant to elimination by host defense mechanisms and antimicrobial agents.[490] Persistent extraradicular root surface colonization cannot be diagnosed by noninvasive methods but may be suspected in well-treated cases that are refractory to nonsurgical treatment.[166,218,465,495,511] Although the presence of extraradicular microbial colonies has been

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somewhat controversial, studies using DNA-DNA hybridization techniques have confirmed the persistence of microorganisms in the periradicular tissues of some root canal–treated teeth.[187,509]

Overextended filling materials may contribute to treatment failure, presumably as a result of a chronic inflammatory response.[369] Although this is possible and even likely with certain toxic materials (e.g., pastes containing formaldehyde),[376] the role of relatively inert materials such as gutta-percha and set sealer is less clear, and these materials probably become a significant contributing factor only if microorganisms are present. If the root apex is close to the buccal cortical plate, apical fenestration may occur, leading to persistent symptoms, especially tenderness to palpation over the root apex.[72] Some have suggested that overextension of filling materials may contribute to endodontic failure because certain dental materials may induce periodontal ligament (PDL) cell apoptosis.[467] This specific interaction between filling materials and periradicular tissues is not fully understood and deserves further research. The poorer prognosis often reported with overextended root canal fillings also may be related simply to lack of an adequate apical seal and subsequent egress of microorganisms from the root canal space. Regardless, minor overextension of filling material is rarely a sole indication for surgery except when symptoms or periradicular pathosis develops. Significant overextension of filling material, especially when important anatomic areas and possibly toxic materials are involved, is an indication for referral to an endodontist or oral surgeon for evaluation and, possibly, treatment.

The presence of periradicular cholesterol crystals may interfere with healing after nonsurgical root canal treatment.[365] Although relatively uncommon, true periradicular cysts (completely enclosed, epithelium-lined cavities) may not be expected to resolve after nonsurgical treatment.[364] As with other extraradicular causes of failure, surgery is indicated, because definitive diagnosis and treatment require an excisional biopsy and removal of the periradicular tissue.

Vertical root fractures are a significant cause of failure and may be difficult to diagnose in the early stages.[90] Exploratory surgery often is required to confirm a root fracture. Cone-beam computed tomography (CBCT) is a promising tool for noninvasive diagnosis of root fractures[155,165] and is

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discussed in greater detail later in this chapter and in Chapter 2. Although several promising approaches to the management of vertical root fractures have been proposed,[226,268,506] the prognosis generally is believed to be poor. Extraction is usually the treatment of choice, especially if appropriate tooth replacement options are available. Root amputation or hemisection may be considered in a multirooted tooth if the remaining tooth structure is uninvolved and sufficient periodontal support is present.

The relationship between systemic disease and periradicular healing is not completely understood. The possible influence of certain systemic medications on wound healing is discussed later in the chapter. Compromised host healing capacity may be a contributing factor in delayed healing and failure of some root canal–treated teeth. For example, complete healing after nonsurgical root canal treatment is less likely in diabetic patients with preoperative periradicular pathosis.[78,175] Patients undergoing immunosuppressive therapy may be at greater risk for delayed healing, treatment failure, or acute exacerbation of a subclinical infection, although two studies involving subgroups of immunocompromised patients (bone marrow transplant and acquired immunodeficiency syndrome [AIDS]) did not find these patients to be at higher risk for complications related to endodontic treatment.[197,406]

Rationale for surgical treatment

Although nonsurgical retreatment generally is believed to be the preferred first approach in the management of persistent apical periodontitis,[77,492,510,564] periradicular surgery is indicated when nonsurgical retreatment is impractical or unlikely to improve on the previous result. In particular, a surgical approach may be the first choice for managing teeth with long posts or irretrievable separated instruments, non-negotiable ledges and canal blockages or transportation, hard cement filling materials, failure of previous nonsurgical retreatment, and suspected vertical root fracture, or when a biopsy is indicated (Figs. 11.1–11.4). Even when surgical treatment is the likely definitive approach, nonsurgical therapy before the procedure may be recommended to help reduce the number of microorganisms in the root canal system and ensure a more favorable long-term prognosis.[230] On the other hand, surgery may be the first choice even if a tooth can be treated

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nonsurgically if the risks and costs of retreatment are considered excessive. For example, disassembling a recently restored bridge abutment tooth to allow endodontic retreatment may be technically possible but not economically feasible. One study found that endodontic microsurgery may be the most cost-effective option for management of persistent periapical disease, compared to nonsurgical retreatment, extraction and placement of a fixed partial denture, and extraction and placement of a single tooth implant.[284] Case-specific variables and clinical judgment are key elements in the decision-making process, as current evidence supports the contention that the prognosis for surgical treatment is approximately the same as that for nonsurgical retreatment.[127,128,135,293,468,577]

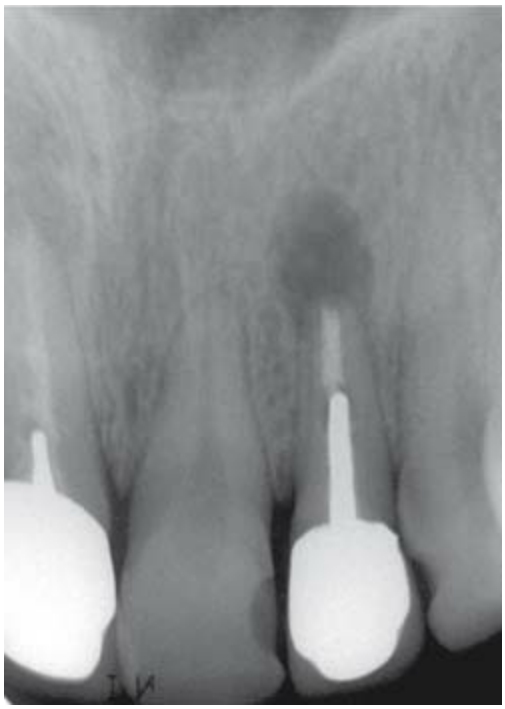

FIG. 11.1 Previously treated maxillary lateral incisor with persistent periradicular disease. Nonsurgical retreatment is possible but would involve disassembly of an otherwise adequate coronal restoration. Periradicular surgery is a reasonable option.

Radiograph shows four teeth with first and third shows radiopaque dentin. A gray patch is present at the root of maxillary lateral incisor.

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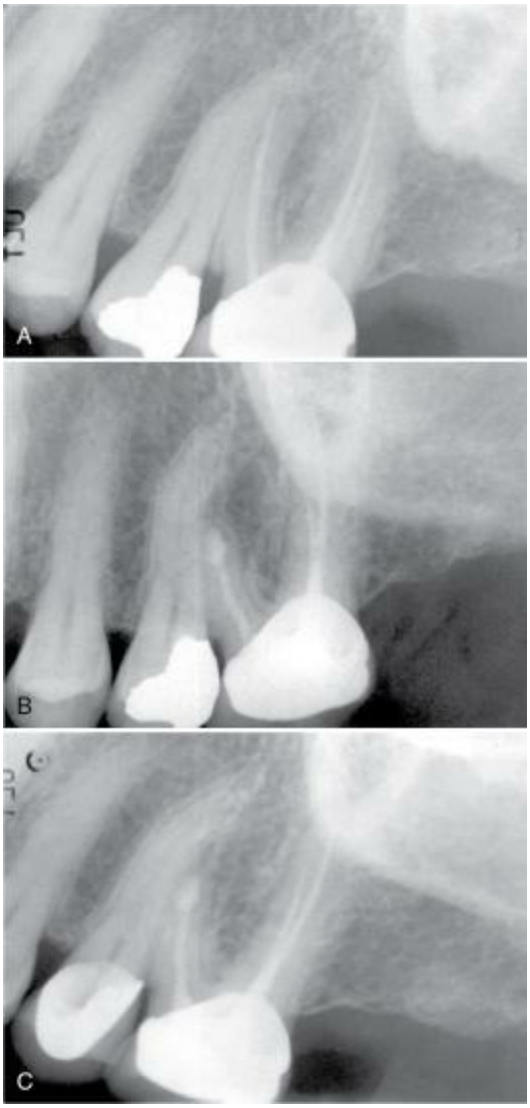

FIG. 11.2 **A,** Previously treated maxillary first molar with persistent periradicular disease and obvious transportation of the main mesiobuccal (MB-1) canal. Nonsurgical retreatment is unlikely to correct this iatrogenic condition, and surgery is the preferred choice. **B,** Immediate postsurgery radiograph. The root-end preparation and fill was extended from the MB-1 canal in a palatal direction to include the isthmus area and the second (MB-2) canal. **C,** One-year follow-up examination: the tooth is asymptomatic, and periradicular healing is apparent radiographically. Although surgery was the first-choice treatment in this case and the outcome was favorable, a good argument could be made for nonsurgical retreatment before surgery to ensure disinfection of the canal and to attempt to locate a MB-2 canal.

Set of three radiographs depict pre-treatment and post-treatment changes observed in the maxillary first molar. The radiograph shows healing of tooth on the left post-treatment depicted as radiopaque crown.

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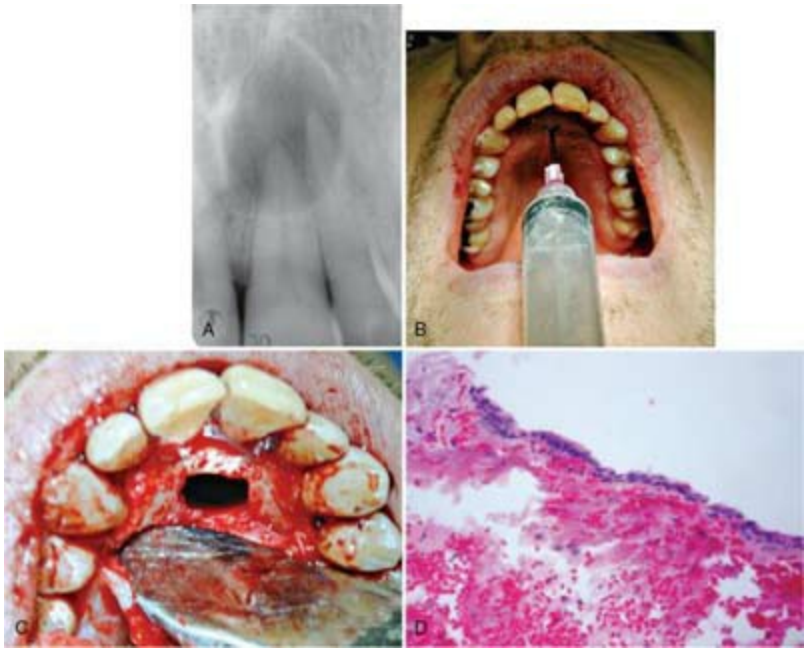

FIG. 11.3 **A,** Indication for surgery (biopsy): large radiolucent lesion in the area of the maxillary left central and lateral incisors was detected on routine radiographic examination. All anterior teeth responded within normal limits to pulp vitality testing. **B,** After administration of a local anesthetic but before surgery, the lesion was aspirated with a large-gauge needle to rule out a vascular lesion. **C,** Buccal and palatal flaps were reflected. The lesion was directly accessible from the palate. An excisional biopsy was performed, and the sample was submitted for evaluation. **D,** Light microscopic section of the biopsy specimen; the lesion was diagnosed as a nasopalatine duct cyst (×400).

Set of four figures shows steps involved in the surgical treatment of lesion in the area of the maxillary left central and lateral incisors.

Source: (Courtesy Dr. Vince Penesis.)

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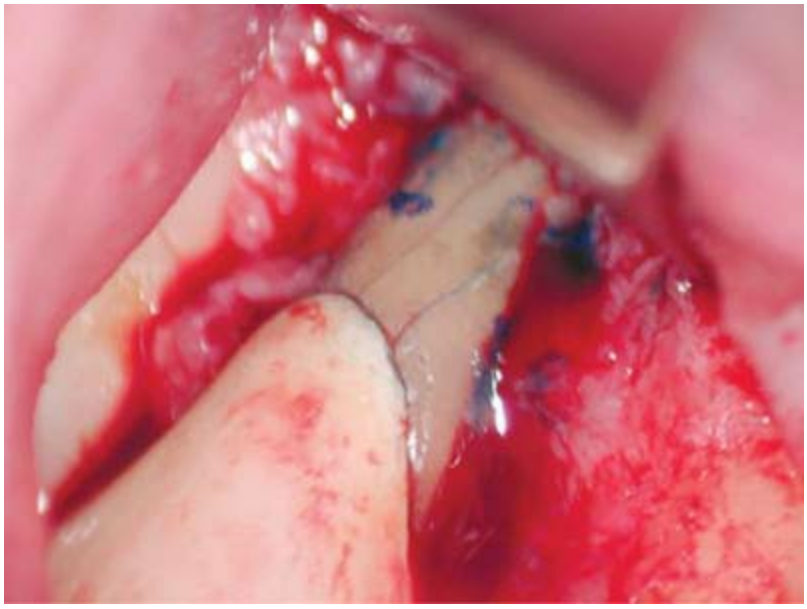

FIG. 11.4 Exploratory surgery was indicated to rule out or confirm a root fracture. Magnification and staining with methylene blue dye confirmed the presence of multiple root fractures. The tooth was subsequently extracted.

Close-up of interior of mouth shows blue lines of stains on the tooth depicting root fractures.

Source: (Courtesy Dr. Martin Rogers.)

Clinical decision making

Clinical decision making is a process that combines the best available evidence, clinical judgment, and patient preferences. Treatment choices are always made under conditions of at least some uncertainty. Surgical root canal treatment rarely is the only possible choice. Clinicians and patients must weigh the relative benefits, risks, and costs of two or more acceptable alternatives. Patients and clinicians can be expected to hold different attitudes about the value of potential treatment outcomes.[269] In a prognosis study of single-tooth dental implants, Gibbard and Zarb[193] reported that factors considered significant by health care professionals may not be important to patients. Even among groups of dentists and dental specialists, the threshold for treatment varies widely and treatment recommendations may depend more on personal values and experience than objective analysis of treatment costs, prognosis, risks, and alternatives.[56,343,439]

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Shared decision making, in which the patient and clinician consider outcome probabilities and patient preferences and agree on the appropriate treatment, is preferable to a clinician-directed treatment decision.[46,179] Such a two-way exchange allows the clinician to provide the best available current evidence and case-specific clinical judgment while encouraging a decision that considers the patient's personal values and preferences. This decision-making model has been shown to increase patient knowledge about and satisfaction with the treatment choice.[262,386,387] In general, most patients prefer to be actively involved in the decision-making process but want to leave the specifics of the treatment to the clinician.[179] That is, the risks, benefits, and costs of treatment alternatives are important to patients, but the details of the procedure usually are not. Shared decision making is particularly relevant in light of the current trend in some areas to recommend implants as an alternative to root canal treatment.

Comparative probabilities for a successful outcome after surgical root canal treatment, nonsurgical retreatment, or extraction and replacement with a fixed prosthesis or implant are difficult to project. Many treatment variables are complex and not easily quantified, such as the affected location in the mouth, bone quality, practitioner's skill, possible influence of systemic disease on healing, periodontal support, bulk of the remaining tooth structure and resistance to fracture, quality of the coronal restoration, patient susceptibility to recurrent caries, materials used, and other factors. In addition, the definition of success varies considerably and is inconsistent from study to study.

Boioli and colleagues[66] performed a meta-analysis of implant studies and reported a 93% survival rate at 5 years. The 5- to 10-year success rate for implant retention routinely is reported to be 90% to 97%, depending on the location in the mouth and other variables.[302,309] Surgical root canal treatment was previously reported to have a lower success rate than implant placement.[230,546] However, most of these older studies would be considered weak by today's evidence-based standards and, more important, do not reflect the use of many of the newer surgical materials and techniques. With careful case selection, surgical skill, and the use of materials and techniques described later in this chapter, many studies have demonstrated success rates over 90% for surgical root canal treatment.[326,448,500,501,546,609] von Arx

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reported that the 5-year success for teeth receiving endodontic microsurgery was 8% less than observed at 1 year.[566] A similar study found an 85% success rate after 10 years.[500] A systematic review and meta-analysis comparing traditional root-end surgery to modern endodontic microsurgery (ultrasonic root-end preparation; root-end filling with intermediate restorative material [Denstply/Sirona, York, PA], Super EBA [ethoxybenzoic acid; Keystone Industries, Gibbstown, NJ], or mineral trioxide aggregate [MTA]; and high power [>×10] magnification and illumination) found that the outcome for endodontic microsurgery using current materials and techniques was 94%, compared to 59% success with traditional root-end surgery.[483]

In broad terms and under ideal conditions, the prognoses for nonsurgical retreatment, surgical treatment, and implant placement should be roughly equal. The choice of treatment should be based on the best available evidence, case-specific clinical judgment, and the patient's preferences. As a stronger evidence base continues to develop, we predict that algorithms encompassing multiple patient and treatment variables will be devised to assist clinical decision making.

General biologic principles of wound healing

Wound healing varies from region to region in the body and depends on several factors, including the type of tissue, the type of wound, and the type of healing. In periradicular surgery, the tissues include free and attached gingiva, the alveolar mucosa, periosteum, bone, the PDL, and cementum. The wound may be intentional surgical trauma, which includes incision, blunt dissection, and excision (surgical), or pathologic or traumatic wounds. Healing occurs by primary or secondary intention.[212]

An incisional blunt dissectional wound, for example, can be considered to heal typically by primary intention, whereas a dissectional wound involving the resected root surface and osseous crypt heals by secondary intention. An important concept of the wound healing process in general is the difference between regeneration and repair. The goal of all surgical procedures should be *regeneration*, which returns the tissues to their normal microarchitecture and function, rather than *repair*, a healing outcome in which tissues do not return to normal architecture and function. Repair typically results in the

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formation of scar tissue.

The wounding process varies, depending on the types of tissue and injury. However, all wounds progress through three broad, overlapping phases in the process of healing; these are the inflammatory phase, the proliferative phase, and the maturation phase.[240,570] Although these phases can be identified in healing tissue, none of them has a clear beginning or end. Furthermore, in a wound such as an endodontic surgical site, which involves more than one tissue type, these phases progress at different rates in each type of tissue.

Soft-tissue wound healing

Inflammatory phase

Broadly speaking, the inflammatory phase of healing is similar for all tissues.[543] This phase can be broken down further into clot formation, early inflammation, and late inflammation.

Clot formation

Clot formation begins with three events: (1) blood vessel contraction is initiated by platelet degranulation of serotonin, which acts on the endothelial cell and increases the permeability of the vessel, allowing a protein-rich exudate to enter the wound site; (2) a plug composed of platelets forms, primarily through intravascular platelet aggregation; and (3) both the extrinsic and intrinsic clotting mechanisms are activated. Several other events occur simultaneously, including activation of the kinin, complement, and fibrinolytic systems and the generation of plasmin.[34,247,521] These events stabilize hemostasis, begin the production of a number of mitogens and chemoattractants, and initiate the process of wound decontamination. The result is a coagulum consisting of widely spaced, haphazardly arranged fibrin strands with serum exudate, erythrocytes, tissue debris, and inflammatory cells. Compression of the surgical flap with sterile iced gauze immediately after surgery is designed to minimize the thickness of the fibrin clot and thereby accelerate optimal wound healing.

Early inflammation: Polymorphonuclear neutrophil organization

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As a result of production of chemoattractants by the various components of the clot, polymorphonuclear neutrophils (PMNs) begin to enter the wound site within 6 hours of clot stabilization. The number of PMNs increases steadily, peaking at about 24 to 48 hours after the injury. Three key steps mark PMN migration into the wound site: (1) *pavementing*, in which the red blood cells undertake intravascular agglutination, allowing the PMNs to adhere to the endothelial cells; (2) *emigration*, in which the PMNs actively pass through the vascular wall; and (3) *migration*, in which the PMNs use ameboid motion, under the influence of the various chemotactic mediators, to move into the injured tissues.[212]

The principal role of the PMNs is wound decontamination by means of phagocytosis of bacteria. The high number of PMNs in the wound site is relatively short lived; the number drops rapidly after the third day.

Late inflammation: Macrophage organization

About the time the PMN population is declining (48 to 96 hours after injury), macrophages begin to enter the wound site. They reach a peak concentration by approximately the third or fourth day. These cells, which are derived from circulating monocytes, leave the bloodstream under the influence of the chemoattractants in the wound site. The monocytes subsequently evolve into macrophages. Macrophages have a much longer life span than PMNs; they remain in the wound until healing is complete. Similar to PMNs, macrophages play a major role in wound decontamination through phagocytosis and digestion of microorganisms and tissue debris.

Macrophages are considerably more bioactive than PMNs and can secrete a vast array of cytokines. A key action of many of these bioactive substances is initiation of the proliferative phase of wound healing, which is accomplished by prompting the formation of granulation tissue. Two other major functions of macrophages are ingestion and processing of antigens for presentation to T lymphocytes, which enter the wound after the macrophages. Unlike PMNs, macrophages play an essential role in the regulation of wound healing.[109,224] A reduction in the number of macrophages in the wound site delays healing, because the wound does not progress to the next phase. For example, the age-related reduction in healing potential appears to be partly the result of loss of estrogen regulation of macrophages in healing tissues.[33]

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Proliferative phase

The proliferative phase is characterized by the formation of granulation tissue in the wound. Two key cell types, fibroblasts and endothelial cells, have a primary role in the formation of granulation tissue. Granulation tissue is a fragile structure composed of an extracellular matrix of fibrin, fibronectin, glycosaminoglycans, proliferating endothelial cells, new capillaries, and fibroblasts mixed with inflammatory macrophages and lymphocytes. Epithelial cells also are active during this phase of soft-tissue healing and are responsible for initial wound closure. Guided tissue regeneration (GTR) procedures are based on control of the epithelial cell growth rate during this phase.

Fibroblasts: Fibroplasia

Undifferentiated mesenchymal stem cells in the perivascular tissue and fibroblasts in the adjacent connective tissue migrate into the wound site on the third day after injury and achieve their peak numbers by approximately the seventh day. This action is stimulated by a combination of cytokines (e.g., fibroblast growth factor [FGF], insulin-like growth factor-1 [IGF-1], and 2-15 platelet-derived growth factor [PDGF]), which are produced initially by platelets and subsequently by macrophages and lymphocytes. As the number of macrophages declines and the fibroblast population increases, the tissue in the wound transforms from a *granulomatous* tissue to a *granulation* tissue.

Fibroblasts are the crucial reconstructive cell in the progression of wound healing because they produce most of the structural proteins forming the extracellular matrix (e.g., collagen). Collagen is first detected in the wound about the third day after injury. The fibroblasts produce type III collagen initially and then, as the wound matures, type I collagen. As this network of collagen fibers is laid down, endothelial and smooth muscle cells begin to migrate into the wound. Subsequently, as wound healing progresses, the collagen fibers become organized by cross-linking. Regularly aligned bundles of collagen begin to orient so as to resist stress in the healing wound.[108,286,413,516]

A focused type of fibroblast, known as a *myofibroblast*, plays a significant role in wound contraction, particularly in incisional-type wounds.[316,524] Myofibroblasts align themselves parallel with the wound surface and then

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contract, drawing the wound edges together. These cells are eliminated by apoptosis after wound closure.[137,138]

Endothelial cells: Angiogenesis

Capillary buds originate from the vessels at the periphery of the wound and extend into the wound proper. This occurs concurrently with fibroblast proliferation and can begin as early as 48 to 72 hours after injury. Without angiogenesis, the wound would not have the blood supply needed for further active healing. The capillary sprouts eventually join to form a network of capillary loops *(capillary plexuses)* throughout the wound.

In addition to a low oxygen concentration in the wound proper,[198,289] several factors have been identified as potent stimulators of angiogenesis, including vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), acidic FGF (aFGF), transforming growth factor–alpha (TGF-alpha) and transforming growth factor–beta (TGF-beta), epidermal growth factor (EGF), interleukin-1 (IL-1), and tumor necrosis factor–alpha (TNF-alpha),[145] as well as lactic acid.[248,258,503] All of these have been shown to stimulate new vessel development.

Epithelium

The first step in epithelial healing is the formation of an epithelial seal on the surface of the fibrin clot. This process begins at the edge of the wound, where the basal and suprabasal prickle cells rapidly undergo mitosis. The cells then migrate across the fibrin clot at a remarkable rate (0.5 to 1 mm/day). This monolayer of epithelial cells continues to migrate by contact guidance along the fibrin scaffold of the clot below. Migration stops as a result of contact inhibition of the epithelial cells from the opposing wound edge. Once the epithelium from both sides of the wound is in contact, an epithelial seal is achieved. In wounds healing by primary intention, formation of an epithelial seal typically takes 21 to 28 hours after reapproximation of the wound margins.[222]

Maturation phase

Under ideal conditions, maturation of the wound begins 5 to 7 days after injury. A reduction in fibroblasts, vascular channels, and extracellular fluids

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marks the transition to this phase of healing. During the early stages of wound maturation, the wound matrix is chiefly composed of fibronectin and hyaluronic acid. As the tensile strength of the wound increases, significant upregulation of collagen fibrogenesis occurs. Collagen remodeling ensues, with the formation of larger collagen bundles and alteration of intermolecular cross-linking. The result is a conversion of granulation tissue to fibrous connective tissue and a decreased parallelism of collagen to the plane of the wound. Aggregated collagen fibril bundles increase the tensile strength of the wound. As healing progresses in the wound, the collagen gradually reorganizes; this requires degradation and reaggregation of the collagen. The degradation of collagen is controlled by a variety of collagenase enzymes. Remodeling results in a gradual reduction in the cellularity and vascularity of the reparative tissue; the degree to which this occurs determines the extent of scar tissue formation. Active remodeling of scar tissue can continue very slowly for life.[138,247]

Maturation of the epithelial layer quickly follows formation of the epithelial seal. The monolayer of cells forming the epithelial seal differentiates and undergoes mitosis and maturation to form a definitive layer of stratified squamous epithelium. In this way an epithelial barrier is formed that protects the underlying wound from further invasion by oral microbes. The epithelial barrier typically forms by 36 to 42 hours after suturing of the wound and is characterized by a significant increase in wound strength.[222]

Hard-tissue healing: Excisional dentoalveolar wound

The inflammatory and proliferative phases of healing for hard tissue are similar to those for soft tissue. A clot forms in the bony crypt, and an inflammatory process ensues that involves PMNs initially and macrophages subsequently. This is followed by the formation of granulation tissue with an angiogenic component. However, the maturation phase of hard-tissue healing differs markedly from that for soft tissues, primarily because of the tissues involved: cortical bone, cancellous bone, alveolar bone proper, endosteum, PDL, cementum, dentin, and inner mucoperiosteal tissue.

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Osteoblasts: Osteogenesis

The healing of an excisional osseous wound that is approximately 1 cm in diameter is similar to that of a fractured long bone. It progresses from hematoma to inflammation, eradication of nonvital debris, proliferation of granulation tissue, callus formation, conversion of woven bone to lamellar bone, and, finally, remodeling of the united bone ends. The coagulum that initially forms delays healing and must be removed to allow wound healing to progress.

A major difference between soft- and hard-tissue wound healing is found in the role of the osteoclast. Functionally, osteoclasts act as organizational units to debride necrotic bone from the wound margin, much as macrophages remove tissue debris from the clot. Granulation tissue begins to proliferate from the severed PDL by 2 to 4 days after root-end resection.[223] This tissue rapidly encapsulates the root end. Simultaneously, endosteal proliferation into the coagulum occurs from the deep surface of the bony wound edge. The coagulum in the bony crypt is quickly converted into a mass of granulation tissue. In addition to those already discussed, several cell types migrate into the coagulum, including osteoprogenitor cells, preosteoblasts, and osteoblasts. These cells begin the formation of woven bone within the mass of granulation tissue. New bone formation is apparent about 6 days after surgery.[223]

Bone formation can be categorized into two types, each having several phases. The phases differ, depending on which type of formation is involved. One type of bone formation is a matrix vesicle–based process, and the other type is based on osteoid secretion. In both processes, osteoblasts produce the bone matrix. They secrete a collagen-rich ground substance that is essential for mineralization. Osteoblasts also cause calcium and phosphorus to precipitate from the blood.

In the formation of woven bone, which occurs by the matrix vesicle–based process, osteoblasts produce matrix vesicles through exocytosis (the release of substances contained in a vesicle within a cell by a process in which the membrane surrounding the vesicle unites with the membrane forming the outer wall of the cell) of their plasma membranes. As hydroxyapatite crystals accrue in the vesicles, they become enlarged and eventually rupture. This process begins with the deposition and growth of hydroxyapatite crystals in

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the pore regions. The crystals then amalgamate to form structures known as *spherulites.* Union of the separate spherulites results in mineralization.

The formation of lamellar bone does not require the production of matrix vesicles; rather, it proceeds by the osteoblast secretion process. Osteoblasts secrete an organic matrix composed of longitudinally arranged collagen matrix fibrils (mainly type I collagen). Mineralization occurs by mineral deposition directly along the collagen fibrils.[239,240] This stage has been associated with a rise in pH, most likely because of the enzyme alkaline phosphatase, which is secreted by osteoblasts and other cells and plays an important role in mineralization. The exact role of alkaline phosphatase during mineralization is unclear. The balance of evidence favors a positive catalytic role.[19,20] Some hypothesize[71,70,552,553] that alkaline phosphatase promotes mineralization through a combination of mechanisms involving various cells, extracellular matrix proteins, and elements. The interaction of alkaline phosphatase and phosphoproteins in both bone and dentin appears to be especially critical to the mineralization process.[71,72,557,558]

Inhibitor molecules, such as pyrophosphate and acidic noncollagenous bone proteins, regulate mineralization. Several growth factors also have been identified as key components in the production of osseous tissue. These include TGF-beta, bone morphogenic protein (BMP), PDGF, FGF, and IGF.[97,191,476,513] One clinical study demonstrated that the addition of autologous platelet concentrate to the surgical site decreased postsurgical pain and may accelerate the healing process.[134]

Three to 4 weeks after surgery, an excisional osseous wound is filled from 75% to 80% with trabeculae surrounded by intensely active osteoid and osteoblastic cells. A reforming periosteum can be seen on the outer surface of the wound; it is highly cellular and has more fibrous connective tissue oriented parallel to the plane of the former cortical plate. At 8 weeks after surgery, the trabeculae are larger and denser and the osteoblasts are less active; these cells occupy about 80% of the original wound. Also, fewer osteoid cells are associated with the maturing trabeculae. The overlying periosteum has reformed and is in contact with the newly developed bone. The osseous defect typically is filled with bone tissue by 16 weeks after surgery. However, the cortical plate has not yet totally reformed. Maturation and remodeling of the osseous tissue continues for several more months.[223]

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Local healing is also influenced systemically by the endocrine system and its three general categories of hormones: polypeptide regulators (parathyroid hormone, calcitonin, insulin, and growth hormone), steroid hormones (vitamin D_3, glucocorticoids, and the sex hormones), and thyroid hormones.[219]

Cementoblasts: Cementogenesis

During regeneration of the periradicular tissues, cementum forms over the surface of surgically resected root ends.[114] The exact spatial and temporal sequences of events leading to this formation of new cementum remains undefined; however, cementogenesis is important, because cementum is relatively resistant to resorption (osteoclasts have little affinity for attaching to cementum).

Cementogenesis begins 10 to 12 days after root-end resection. Cementoblasts develop at the root periphery and proceed centrally toward the root canal.[24] Considerable evidence indicates that the cells that regulate cementogenesis are derived from ectomesenchymal cells in the tooth germ proper rather than from bone or other surrounding tissues. The migration and attachment of precementoblasts to the root surface dentin is monitored by mediators from within the dentin itself.[140,210] Cementum covers the resected root end in approximately 28 days. Newly formed PDL fibers show a functional realignment that involves reorientation of fibers perpendicular to the plane of the resected root end, extending from the newly formed cementum to the woven bone trabeculae. This occurs about 8 weeks after surgery.[212,319,320]

Systemic medications and wound healing

Bisphosphonates

Bisphosphonates are commonly used for the treatment of osteopenia, osteoporosis, Paget disease of bone, multiple myeloma, and metastatic bone, breast, and prostate cancer. The potential association between bisphosphonate use and osteonecrosis of the jaw was first reported in 2003.[332,351] The current preferred term for this condition is antiresorptive agent-induced osteonecrosis

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of the jaw (ARONJ)[229] or medication-related osteonecrosis of the jaw (MRONJ). The change in terminology reflects the finding that, in addition to bisphosphonates, other antiresorptive agents can increase the risk for osteonecrosis of the jaw (e.g., denosumab). ARONJ can occur spontaneously but is more commonly associated with dental procedures that involve bone trauma. Patient variables that increase the risk for ARONJ include age (over 65 years old), chronic use of corticosteroids, use of bisphosphonates for more than 2 years, smoking, diabetes, and obesity.[278,334,340,583] It is important to note that the risk for ARONJ with commonly prescribed oral bisphosphonates appears to be very low, whereas the overall benefits of this drug class for reducing morbidity and mortality related to hip, vertebral, and other bone fractures are significant.[229] The estimated incidence for ARONJ in patients taking oral bisphosphonates ranges from zero to 1 in 2260 cases, although dental extractions may quadruple the risk of developing ARONJ.[7,340] A reasonable upper estimate of the risk of developing ARONJ in a patient who does not have cancer is about 0.1%.[229] One clinical study reported a much higher 4% risk for developing ARONJ in patients taking a specific oral bisphosphonate (alendronate sodium),[479] although this was a retrospective study using an electronic patient record at a dental school and may not have adequately controlled for other relevant variables. The majority of ARONJ cases have been reported in patients taking intravenous (IV) bisphosphonates (e.g., zoledronic acid and pamidronate). According to one report, approximately 20% of patients taking IV bisphosphonates may develop ARONJ.[7,340] A systematic review found that there was a strong association between cancer and risk of ARONJ, with the prevalence ranging from 0.7% to 13.3%.[352] Interestingly, this same review found that the higher-quality studies reported the highest prevalence of ARONJ in cancer patients.

Conservative and timely management of apical periodontitis is essential to reduce the risk of ARONJ in patients taking bisphosphonates. Because periapical pathosis may exacerbate or increase the risk for ARONJ, the “no treatment” option is not a viable choice. The potential risk for ARONJ should be thoroughly discussed with all patients that are taking bisphosphonates, as well as treatment options. Nonsurgical retreatment should usually be considered the first choice, especially for patients with a history of IV bisphosphonate use or other risk factors. Even so, surgical treatment may be

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indicated to manage chronic or acute apical periodontitis. When the only viable treatment options for the management of persistent periradicular inflammation are surgical root canal therapy or extraction, the question of which option is more or less likely to lead to ARONJ in at-risk patients remains unanswered. In general, the procedure that could most predictably eliminate the periradicular inflammation with the least amount of surgical trauma would be preferred. Conservative surgical technique, primary tissue closure, and use of chlorhexidine mouth rinses preoperatively and during the healing stage are recommended.[229] There is some limited evidence to support the use of prophylactic antibiotics and chlorhexidine mouth rinse to decrease the risk of ARONJ for patients undergoing surgical dental treatment.[229] Because bisphosphonates have a substantial half-life when incorporated into bone, discontinuation of bisphosphonates prior to dental treatment is unlikely to provide any measurable benefit and may place the patient at greater risk for complications that the drug is intended to prevent.

The possibility of predicting which patients may be at greater risk for ARONJ based on serum levels of C-terminal cross-linking telopeptide of type I collagen (cross-linking telopeptide [CTX]) has been suggested based on clinical observations.[333] This observation may provide a useful clue for future risk assessment but requires additional refinement and validation prior to widespread acceptance. In fact, one study found that although serum CTX levels were lower in patients taking bisphosphonates (as expected), there was no association between lower CTX levels and increased risk of ARONJ.[208] This same study found that patients taking a once-a-year dose of 5 mg zoledronic acid were at no greater risk for ARONJ than a control group (1 case in 5903 patients). Once ARONJ develops, treatment options are limited. A possible treatment strategy proposed in a series of case reports is the use of teriparatide, an anabolic drug that stimulates osteoblast formation and is used for the management of osteoporosis.[505]

Glucocorticoids

Glucocorticoid therapy has been shown to induce rapid bone loss within the first 3 months of treatment. Even inhaled steroids have been implicated as a cause of bone loss. Bone formation is inhibited partly through a decrease in osteoblast life span and function, a reduction in the mineral apposition rate,

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and a prolonged mineralization lag time. Biochemical markers of bone formation (i.e., osteocalcin and bone-specific alkaline phosphatase) are suppressed. In addition to this primary suppressive effect, glucocorticoids cause accelerated bone resorption. The number and activity of osteoclasts increase during early glucocorticoid exposure. With continued use of glucocorticoids, the rapid rate of osteoclast-mediated bone resorption slows, but suppression of bone formation continues as the overriding skeletal activity.[272] Bone loss is therefore progressive because bone resorption chronically exceeds bone formation.

The adverse effects of glucocorticoids on bone are mediated by sex steroid deficiency and expression of locally produced growth factors and related proteins, such as reduced production of IGF-1 and alterations in IGF-binding proteins in osteoblasts. The direct effect on calcium metabolism through alteration of vitamin D metabolism leads to a state of secondary hyperparathyroidism.[447,581,582]

Nonsteroidal antiinflammatory drugs

Bone homeostasis is regulated by many factors, including prostaglandins (PGs).[267] PGs are important to both normal and pathologic bone turnover. PGs modulate osteoblast proliferation and differentiated functions.[426,427] The levels of prostaglandin E (PGE) and prostaglandin F (PGF) are elevated in the early phase of fracture healing, and administration of PGE_2 has increased the rate of osseous repair in several animal studies.[272] Nonsteroidal antiinflammatory drug (NSAID) inhibition of the enzyme cyclooxygenase (COX), which is involved in the synthesis of PGs, is the same mechanism by which NSAIDs control pain. By inhibiting the COX enzymes and the subsequent production of PGs, NSAIDs accomplish the desired antiinflammatory effects—but also prevent the increased production of PGs required for bone healing. In vitro studies using animal models have shown that NSAIDs inhibit osteoblast proliferation and stimulate protein synthesis.[235] These drugs also have been shown to delay fracture healing and to affect bone formation adversely in animals and humans.[15,192] In one study, the use of NSAIDs reduced the amount of bony ingrowth into an orthopedic implant.[220]

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In other studies, NSAIDs reduced bone resorption associated with experimentally induced periapical lesions,[18,390] and systemic NSAIDs may play a positive role in maintaining bone height around endosseous titanium dental implants.[257] Additional research is needed to clarify the specific influence of NSAIDs on healing after periradicular surgery.

Cyclooxygenase-2 inhibitors

Although both COX-1 and COX-2 have been identified in osteoblasts, the different roles of the two COXs in bone formation remain unclear. A study using COX-1 and COX-2 knockout mice compared the roles of the two enzymes in fracture healing. COX-2 was shown to have an essential role in both endochondral and intramembranous bone formation during wound healing. COX-2 knockout mice showed a persistent delay in cartilage tissue ossification. No difference in fracture healing was observed between the COX-1 knockout mice and the wild-type control used in this study.[603] Additional studies have shown a persistent delay in healing with COX-2 NSAIDs[202,203]; however, this difference in effect was not apparent clinically in a study examining the healing of spinal fusion wounds.[182]

Preoperative evaluation of medically complex patients

The preoperative assessment must take into account both the type of procedure planned and the type of patient (i.e., physical health and psychological status). Healthy patients clearly can be expected to tolerate surgical procedures better than medically complex patients. Clinicians should anticipate and prepare for the inevitability of treating more medically complex patients as the population ages. It is beyond the scope of this brief section to cover all possible medical considerations; rather, the most common issues that may require modification of an endodontic surgical treatment plan are presented. Relatively few absolute contraindications to periradicular surgery exist for patients well enough to seek care in an ambulatory dental office. Nonetheless, if any question arises about a patient's ability to tolerate a surgical procedure, medical consultation is advised. A thorough medical history and assessment of vital signs are required parts of the presurgical

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evaluation.

The American Society of Anesthesiologists (ASA) has developed a widely used system for establishing surgical risk. Patients categorized as ASA 1 are healthy and usually require no modification of the surgical treatment plan. Patients classified as ASA 4 or ASA 5 are not treated in a dental office; these individuals have significant medical problems that take priority over dental considerations. Patients considered ASA 2 or ASA 3 are commonly seen on an outpatient basis and may require medical consultation and modification of the surgical treatment plan. Patients in the ASA 2 and ASA 3 categories have mild to moderate systemic disease and often are undergoing treatment with one or more prescription medications. The ASA classification system should only be used as a general guide because, when used alone, the ASA system is not a reliable predictor of operative risk.[201] In addition, even experienced anesthesiologists exhibit differences of opinion when using the ASA system to classify patients.[227,395] The patient's psychological status and anticipated procedural stress should be considered in addition to the ASA classification of physical health. A stress reduction protocol may be helpful for patients who report moderate dental anxiety with concurrent mild to moderate systemic disease.

Surgical procedures typically require a larger amount of local anesthetic with a vasoconstrictor than a nonsurgical root canal treatment. An essential part of the presurgical evaluation is an assessment of the patient's cardiovascular status and tolerance for local anesthetics that contain epinephrine. In particular, patients with advanced cardiovascular disease, geriatric patients, and patients taking certain medications may have a reduced tolerance for local anesthetics containing a vasoconstrictor. Treatment-induced stress associated with surgical procedures can cause a significant increase in heart rate and systolic blood pressure when compared to nonsurgical root canal therapy.[189] The use of local anesthetics with vasoconstrictors was addressed in the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7).[231] An updated version of this report (JNC 8) was issued in 2013. In patients with cardiovascular disease, 0.036 to 0.054 mg of epinephrine (approximately two to three cartridges of local anesthetic with 1:100,000 epinephrine) should be safe for most patients except those with

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severe cardiovascular disease or other specific risk factors.[38,231,594] Local anesthetics with vasoconstrictors should be avoided or used with extreme caution in patients with the following cardiovascular conditions: severe or poorly controlled hypertension, arrhythmias that are refractory to treatment, myocardial infarction within the past month, stroke within the past 6 months, coronary artery bypass graft within the past 3 months, and uncontrolled congestive heart failure.[311] Patients unable to tolerate vasoconstrictors may not be good candidates for periradicular surgery, because a local anesthetic with a vasoconstrictor is essential for obtaining adequate hemostasis and visibility during this type of procedure. Surgery can be performed using only local anesthetics without vasoconstrictors, but this is not recommended.

The potential for multiple drug interactions is increasing because of the aging population and the introduction of many new drugs. Many elderly patients have diminished liver and kidney function and therefore cannot metabolize and excrete drugs as efficiently as younger, healthy patients. The potential for drug interactions and decreased metabolism and excretion of drugs should be considered, even for commonly used agents such as local anesthetics and analgesics.

A transient bacteremia is a virtual certainty with periradicular surgery; therefore patients at high risk for bacterial endocarditis should receive appropriate antibiotic prophylaxis as recommended by the American Heart Association. New guidelines for prevention of infective endocarditis were published in 2007 and represent a significant change from previous American Heart Association guidelines.[588] For example, antibiotic prophylaxis is no longer recommended for patients with a history of mitral valve prolapse (with or without regurgitation), rheumatic heart disease, bicuspid valve disease, aortic stenosis, and certain congenital heart conditions. Antibiotic prophylaxis is now only recommended for patients with valvular disease associated with the highest risk of adverse outcomes from infective endocarditis. For patients in the highest risk category, antibiotic prophylaxis is recommended for dental procedures that involve manipulation of gingival tissues or the periapical region of teeth or perforation of the oral mucosa. For all other patients with valvular disease, the risks associated with routine antibiotic prophylaxis are greater than the potential benefits.[588] Although these guidelines represent the best available current evidence, practitioners

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should be aware that a recent study from England found a significant increase in cases of infective endocarditis after widespread adoption of the new guidelines in 2008. However, the authors point out that their data do not establish a causal relationship and recommend adherence to the current guidelines at this time.[130a]

New clinical practice guidelines for the management of patients with prosthetic joints were co-developed by the American Association of Orthopaedic Surgeons and the American Dental Association in December 2012.[578] The working group conducted a systematic review and presented three recommendations. Recommendation 1 states that "the practitioner might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures." The level of evidence to support this recommendation was classified as "limited," but that was the highest level of evidence assigned to any of the three recommendations. Two case-control studies provide evidence for this recommendation.[51,498] In recommendation 2, the group was unable to recommend for or against the use of topical antimicrobial agents (grade of recommendation = inconclusive). Recommendation 3 was a consensus recommendation to support the maintenance of appropriate oral hygiene (grade of recommendation = consensus). A recent American Dental Association Council on Scientific Affairs report added further clarification by concluding that: "In general, for patients with prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures to prevent prosthetic joint infection.[499a]

Management of patients undergoing anticoagulant therapy depends on the type of anticoagulant, the reason for anticoagulant therapy, and the type of oral surgery planned. Warfarin (Coumadin), a commonly prescribed anticoagulant used to treat or prevent thromboembolisms, blocks the formation of prothrombin and other clotting factors. The international normalized ratio (INR) value is the accepted standard for measuring prothrombin time (PT). The desired therapeutic range for the INR usually is 2 to 3.5, depending on the underlying medical indication for anticoagulant therapy. Limited oral surgery procedures, such as simple forceps extraction of one to three teeth, may be performed safely on patients with INR values

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within the normal therapeutic range.[8,89,255,478] Periradicular surgery, however, may present a greater challenge for hemostasis, even for patients well maintained within the therapeutic range. The clear field visibility normally required for proper surgical management of the root end may not be possible in patients undergoing anticoagulant therapy. The patient's physician must be consulted for assistance in developing an appropriate treatment plan. Some patients may be able to tolerate discontinuation of warfarin therapy 2 days before a planned surgical procedure, which allows the INR to drift downward. In a prospective cohort study, Russo and colleagues[461] reported that suspension of warfarin 2 days before a surgical procedure resulted in no bleeding problems and no thromboembolic events. They found that the average time spent at an INR below 2 (critical value) was 28 hours and that 90% of the patients returned to the desired therapeutic INR value within 7 days. However, this strategy may place certain patients at greater risk for a thromboembolic event; therefore discontinuation of anticoagulant therapy would not be recommended.

In general, patients undergoing anticoagulant therapy should present minimal risk for significant bleeding during or after oral surgery, and local measures to control bleeding should be adequate.[88,569] In a review prepared for the American Dental Association Council on Scientific Affairs and Division of Science, Jeske and Suchko[260] recommended against routine discontinuation of anticoagulant therapy before dental procedures, including surgical procedures. Regardless of the management approach selected, consultation with the patient's physician and an INR test on the day of surgery are strongly recommended. A retrospective study of an urban dental school population found that INR values for 43% of patients taking warfarin were not within the recommended therapeutic range.[266] To underscore the challenge of maintaining anticoagulation levels within an appropriate therapeutic range, warfarin has been implicated in more emergency hospital admissions than any other medication.[83]

Hospitalization and conversion to heparin therapy may be considered in special cases, but the patient, physician, and surgeon must carefully weigh the potential risks against the expected outcome and benefits. A new category of heparin anticoagulant, low-molecular-weight heparins (LMWHs), allows patient self-administration and may be an alternative for patients who need to

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maintain a high level of anticoagulation but want to reduce the cost and time required for traditional heparin conversion therapy.

Novel oral anticoagulant drugs (NOACs) have recently emerged as an alternative to warfarin for most indications (e.g., previous stroke, atrial fibrillation, and deep vein thrombosis) *except* for mechanical heart valves. Examples include: apixaban (Eliquis), dabigratran (Pradaxa), edoxaban (Lixiana), and rivaroxaban (Xarelto). NOACs have fewer interactions with foods and other drugs, and there is no need for routine blood work monitoring. However, although NOACs are known to prolong PT by inhibiting factor X to Xa conversion, there is currently no reliable test to accurately measure level of anticoagulation. Medical consultation may be indicated to help evaluate the relative benefits and risks of discontinuing NOAC drugs for 1 or 2 days prior to endodontic surgery.

Low-dose aspirin therapy is known to increase bleeding time by irreversibly inhibiting platelet aggregation. A common practice has been to advise patients to discontinue aspirin therapy for 7 to 10 days before oral surgery.[28] At low-dose therapeutic levels (<100 mg/day), aspirin may increase bleeding time and complicate surgical procedures. However, Ardekian and colleagues[28] concluded that low-dose aspirin therapy should *not* be discontinued before surgical procedures and that bleeding could be controlled by local measures. Higher-dose therapy may create a greater risk of bleeding during or after surgery.

Although a patient undergoing aspirin therapy may not be at high risk for significant bleeding during or after surgery, a concern in periradicular surgery is the visibility problems created by oozing blood. The clinician should consult the patient's physician about the medical reason for aspirin therapy and should weigh the risks and benefits of discontinuing aspirin before the proposed surgery. It should be possible to perform periradicular surgery without discontinuing aspirin therapy if necessary, but visibility during the procedure may be compromised, which may adversely affect the prognosis.

Herbs, dietary supplements, and vitamins can contribute to bleeding problems during surgery, and patients often fail to report use of these substances in the preoperative evaluation.[385] In a survey of surgical patients, approximately one third reported using a nonprescription medication that might inhibit coagulation or interact with anesthetics.[384] In particular, ginkgo

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biloba, ginger, garlic, ginseng, feverfew, and vitamin E inhibit platelet aggregation and can increase the risk of bleeding.[96] Ingredients in over-the-counter (OTC) weight loss products can potentiate the effect of epinephrine and increase cardiac stress, although the most obvious example of this phenomenon, ephedra, has been removed from the US market by a Food and Drug Administration (FDA) order.

Patients with inherited or acquired bleeding disorders are also at risk for excessive bleeding during and after periradicular surgery. Impaired liver function associated with past or current alcohol or drug abuse may also predispose a patient to excessive bleeding during surgery. A thorough medical history and patient interview should help identify these patients and, in consultation with the patient's physician, allow for any necessary treatment modifications.

Many other medical conditions, such as recent myocardial infarction, stroke, cardiac arrhythmias, diabetes, radiation therapy for head and neck cancer, immunocompromising conditions, seizure disorders, adrenal suppression, liver or kidney disease, and pregnancy may require modification of the treatment plan. However, these conditions usually present more general problems that are not unique to a surgical procedure. The value of a thorough medical history and patient interview cannot be overemphasized.

Anatomic considerations

Evaluating the access to the surgical site is one of the most important steps in case selection for periradicular surgery. Anatomic studies can provide some guidance, but individual variation is great, and there is no substitute for a complete clinical examination. A small oral opening, active facial muscles, shallow vestibule, and thick buccal alveolar bone all can significantly increase the difficulty of the procedure, even in cases that appear straightforward on radiographic examination.

Posterior mandible

The primary anatomic structure of concern for periradicular surgery in the posterior mandible is the neurovascular bundle that courses through the mandibular canal and exits through the mental foramen. The relationship

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among the mandibular canal, the mental foramen, and the root apices of mandibular teeth has been well studied; however, anthropometric averages have limited value in the treatment of individual patients. An understanding of the typical anatomic relationships is important, but even more important is an evaluation of the individual patient to develop a case-specific risk assessment.

The depth of the vestibular fornix generally is a good predictor of the possible difficulty involved in obtaining surgical access to the mandibular posterior teeth.[305] A shallow vestibule usually portends thicker alveolar bone and more difficult access to the root end.

The mental foramen, another key anatomic structure, is usually located between and apical to the mandibular first and second premolars[118,356]; however, this varies considerably, and the practitioner must examine each patient carefully to determine its location (Figs. 11.5 and 11.6). Vertical location of the mental foramen may vary even more than horizontal location. Moiseiwitsch[356] found that the average location was 16 mm inferior to the cementoenamel junction (CEJ) of the second premolar, although the range was 8 to 21 mm, which would place approximately 20% of the foramina at or coronal to the root apex. Fortunately, the mental foramen usually is easily visualized with standard periapical and panoramic radiographs. A vertically positioned periapical radiograph often may be more useful than a horizontally positioned one, especially in individuals with longer roots. Also, the mental foramen can usually be palpated.

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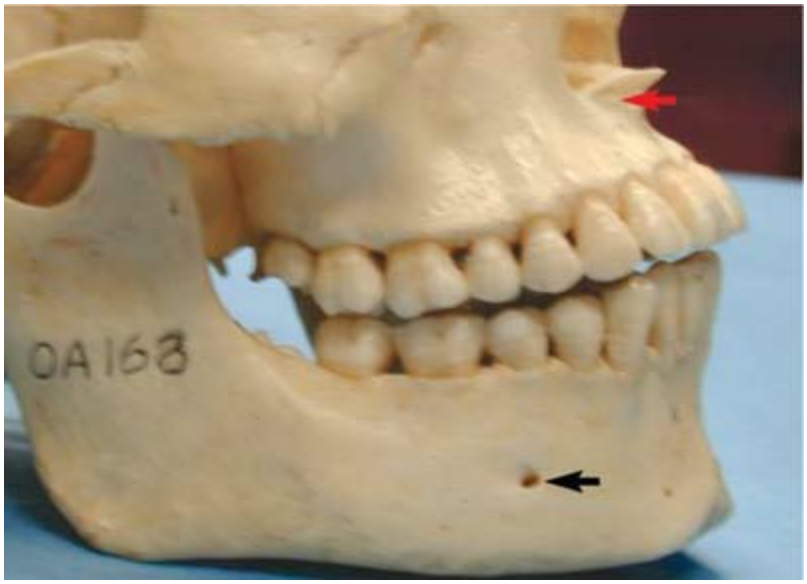

FIG. 11.5 Lateral view of the skull showing the anterior nasal spine and the proximity of the maxillary anterior root apices to the floor of the nose *(red arrow),* as well as the typical location of the mental foramen *(black arrow).*

Lateral view of model of skull shows a red arrow pointing the root apices present on the floor of the nose on top and a black arrow pointing at a hole present approximately 16milimeters inferior to the cementoenamel junction of the second premolar.

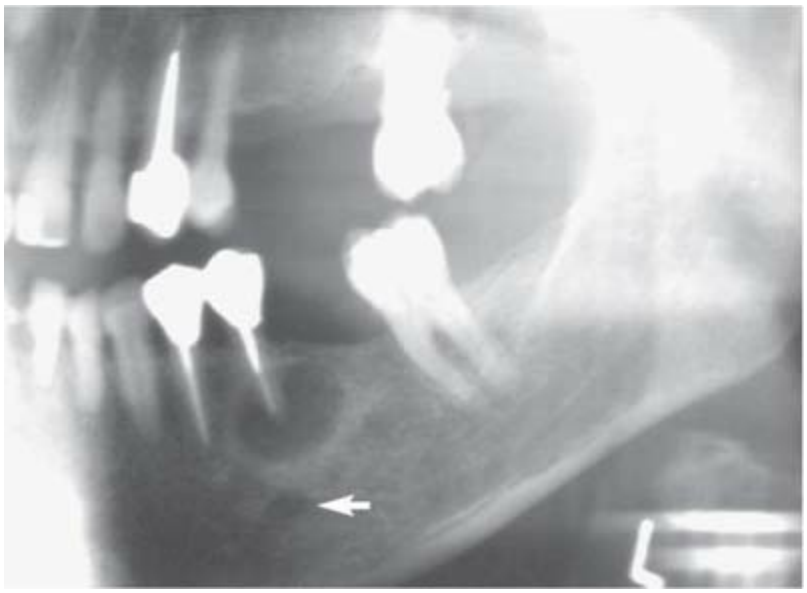

FIG. 11.6 Preoperative evaluation of a mandibular left second premolar included a panoramic radiograph to help locate the mental

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foramen *(white arrow),* which was not visible on a standard periapical radiograph.

Radiograph of lateral view of jaw shows a white arrow pointing at a dark spot present in the mandibular region.

When a vertical releasing incision is indicated, it typically is made at the mesial line angle of the mandibular canine. This location is always mesial to the mental foramen, because the foramen is located in the area ranging from the apex of the mandibular first premolar to slightly distal to the second premolar. The nerve bundle exits from the foramen in a distal direction. An alternative technique for gaining access to mandibular posterior teeth involves a distal releasing incision between the mandibular first and second molars.[355] This approach may be especially useful for access to the mandibular second premolar and first molar. Care must be taken to avoid the facial artery as it crosses the level of the inferior vestibular fornix near the mandibular first molar. Inadvertent contact with the facial artery is unlikely if the incision is not extended beyond the depth of the vestibule.

Regardless of the approach selected, it is obviously important to avoid making an incision in the immediate vicinity of the mental foramen. The mental nerve is encased in a relatively tough sheath, and permanent damage can be avoided if careful blunt dissection is used in the area. A misguided vertical releasing incision, however, may sever the nerve, causing permanent injury. Trauma to the nerve from blunt dissection in this area or from pressure from a misplaced retractor may cause temporary paresthesia but is much less likely to do permanent injury. (Documentation and management of nerve injuries are discussed later in the chapter.)

The borders of the mandibular canal often are more difficult to visualize with conventional radiographic techniques. A parallel periapical radiograph, either horizontally or vertically positioned, usually can provide a reasonably accurate image of the relationship between the superior border of the mandibular canal and the root apices. However, the mandibular canal sometimes cannot be readily visualized. Such cases should be approached with extreme caution, because an increased risk of paresthesia secondary to nerve injury may be an unacceptable risk for many patients. CBCT imaging can be very useful for identifying the location of the mandibular canal and determining its relationship to the root apices (see Chapter 2).[68,285,556]

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In the buccolingual dimension, the mandibular canal commonly follows a curving pathway from the buccal half of the mandible near the distal root of the second molar to the lingual half of the mandible near the first molar, then curving back to the buccal near the second premolar as it exits the mental foramen.[136] The average vertical distance from the superior border of the mandibular canal to the distal root apex of the mandibular second molar is approximately 3.5 mm; this increases gradually to approximately 6.2 mm for the mesial root of the mandibular first molar and to 4.7 mm for the second premolar.[136,312] This relationship typically provides a greater margin of safety for surgery on the mandibular first molar compared with the second premolar and especially the second molar. Surgery on the mandibular second molar may be further complicated by relatively thick overlying buccal bone, lingual inclination of the roots, and a more buccal location of the mandibular canal. This is not to say that periradicular surgery should not be performed on mandibular second molars; rather, it points out that the relative risks and benefits must be carefully considered. Often the most prudent choice for a mandibular second molar is an intentional replantation procedure or extraction and placement of an implant.

Posterior maxilla

The primary anatomic structure of concern in the posterior maxilla is the maxillary sinus. CBCT allows for a more precise preoperative three-dimensional evaluation of the relationship between the roots of maxillary posterior teeth and the sinus.[69,79] Perforation of the sinus during surgery is fairly common, with a reported incidence of about 10% to 50% of cases.[157,177,456] Even without periradicular pathosis, the distance between the root apices of the maxillary posterior teeth and the maxillary sinus sometimes is less than 1 mm.[225] An inflammatory periradicular lesion often increases the likelihood of sinus exposure during surgery. Fortunately, perforation of the maxillary sinus rarely results in long-term postoperative problems.[225] In a report of 146 cases of sinus exposure during periradicular surgery, Watzek and colleagues[579] found no difference in healing compared with similar surgical procedures without sinus exposure. The sinus membrane usually regenerates, and a thin layer of new bone often forms over the root end, although osseous regeneration is less predictable.[50,157,579] The general rule of

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placing a vertical releasing incision at least one tooth mesial and distal to the surgical site is especially important when sinus perforation is possible, because the exposure site should be completely covered with the mucoperiosteal flap to provide primary closure.

If the maxillary sinus is entered during surgery, special care must be taken to prevent infected root fragments and debris from entering the sinus. The most commonly used root resection technique involves grinding the root apex with a high-speed drill for approximately 3 mm in an apical to coronal direction; therefore an opening could allow infected debris into the sinus. A sinus opening can be temporarily occluded with a material such as Telfa gauze, although the gauze should be secured so as to prevent inadvertent displacement into the sinus. A suture can be placed through the packing material to prevent displacement and aid retrieval. Jerome and Hill[259] suggested securing the apical root segment by drilling a small hole in the root tip and threading a piece of suture material through the hole. The root is then resected at the appropriate level, and the root-end fragment is removed in one piece. If a root fragment or other foreign object is displaced into the sinus, it should be removed. An orascope or endoscope may be useful for visualizing the foreign object, but referral for evaluation and surgical removal may be indicated if the fragment cannot be located and removed.

The palatal roots of maxillary molars present a special challenge for surgical access. Palatal roots may be reached from either a buccal (transantral) or palatal approach. Wallace[572] described a transantral approach in which a buccal flap is reflected, the buccal roots are resected, the osteotomy access into the sinus is enlarged to approximately 1 by 1.5 cm, and the palatal root tip is resected, ultrasonically prepared, and filled. The sinus may be packed with moist gauze to catch debris, and it should be irrigated with sterile saline upon completion of the surgery. In some cases, careful reflection and retraction of the sinus membrane may be possible, allowing access to the palatal root end without direct invasion of the sinus.[16] Many who have tried this approach find it more challenging than it might initially seem. The enhanced illumination and magnification provided by a dental operating microscope, endoscope, or orascope are essential aids in this type of surgery.[40]

A palatal approach to the palatal root of maxillary molars may seem more

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direct than a transantral approach, but it can present certain difficulties. Visibility in the surgical field is reduced and manipulation of instruments is more difficult than with most routine buccal approaches. Patients with a deep, vertical palatal vault are better candidates for this approach than individuals with a wide, shallow palate. A sinus tract or a large lesion on the palatal root may allow easier access and better visualization of the palatal root because only limited bone removal would be required. The position of the anterior palatine artery must be carefully considered when the incision is made and the flap reflected. This artery emerges from the greater palatine foramen distal to the maxillary second molar at the junction of the vertical section of the alveolar process and the flat portion of the palate and continues anteriorly (Fig. 11.7). A vertical releasing incision can be placed between the maxillary first premolar and canine, where the artery is relatively narrow and branches off into smaller arteries. If needed, a short distal vertical releasing incision may be made distal to the second molar, but it should not approach the junction of the alveolar process and roof of the palate. If the anterior palatine artery is severed, local clamping and pressure may not stop the hemorrhage, and ligation of the external carotid artery may be necessary. Because of the palatal vault's concave shape, repositioning the flap may be challenging. An acrylic surgical stent may be fabricated before surgery to assist repositioning of the flap and to help prevent pooling of blood under the flap.

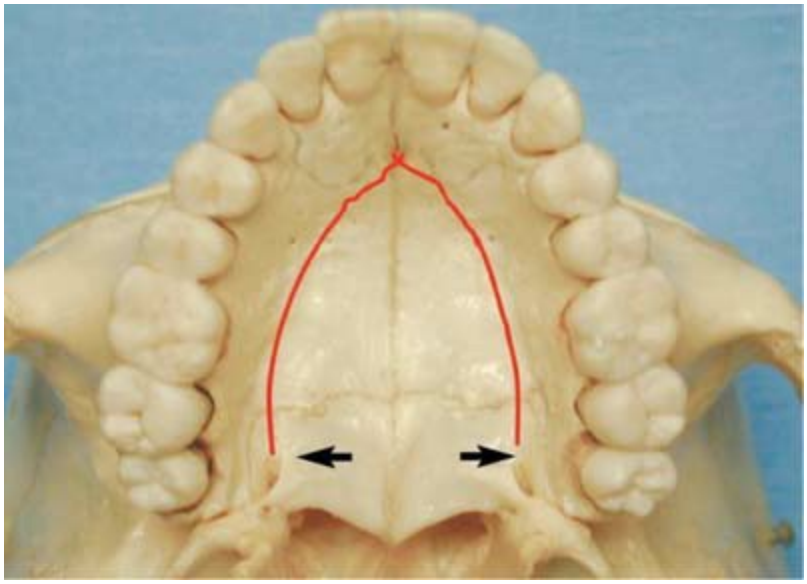

FIG. 11.7 Palatal view showing the position of the greater palatine

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foramen *(arrows)*. The approximate location of the anterior palatine artery is marked in *red*.

Close-view of palatal view shows red arc marked in between depicting anterior palatine artery, with two opposite arrows pointing at two holes at the bottom of arc representing greater palatine foramen.

Anterior maxilla and mandible

Periradicular surgery on anterior teeth generally involves fewer anatomic hazards and potential complications than in posterior teeth. Nonetheless, access to the root apex in some patients may be unexpectedly difficult because of long roots, a shallow vestibule, or lingual inclination of the roots. As shown in Fig. 11.5, the root apices of maxillary central and lateral incisors can be quite close to the floor of the nose and the bony anterior nasal spine. Fig. 11.8 demonstrates isolation and protection of the nasopalatine neurovascular bundle during maxillary anterior surgery. The average maxillary canine is about 26 mm long and usually presents no difficulty for surgical access; however, the combination of a shallow vestibule and a longer than average root length could complicate access to the root apex area. In such cases, the creation of an osteotomy apical to the root end may be impossible. An alternative approach for long-rooted teeth and root tips in the vicinity of critical anatomic structures is to enter the bone and resect the root at a level approximately 3 mm coronal to the apex. After the root tip is removed, the area apical to the root can be inspected and curetted as needed.

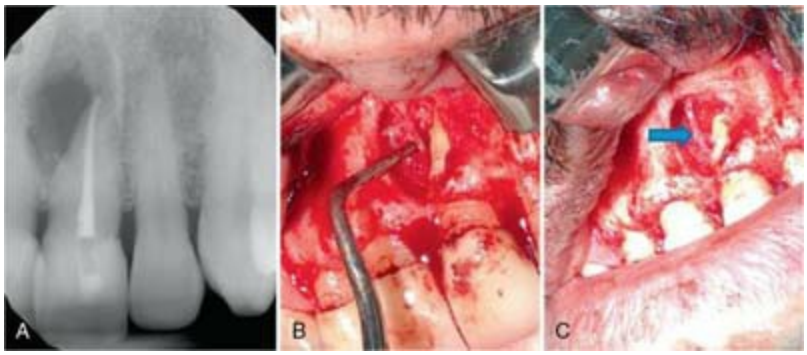

FIG. 11.8 **A,** Radiograph demonstrating periradicular radiolucency associated with previously treated tooth #9. **B–C,** After full-thickness flap reflection, the nasopalatine neurovascular bundle was identified

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(**B**), protected, and isolated (*arrow* in **C**) during curettage of cystic lesion.

Set of three illustrations are marked A through C as follows:

A) Radiograph shows a gray spot present to the left of apical foramen of a tooth.

B) Close-up of interior of mouth shows tip of bent instrument pointing at site of the extracted flap from gingiva.

C) Close-up of interior of mouth shows an arrow pointing at hollow space at site of extraction.

Source: (Courtesy Dr. Tim Rogers.)

Periradicular surgery on mandibular incisors often is more challenging than expected. The combination of lingual root inclination, a shallow vestibule, and a prominent mental protuberance all can increase the degree of difficulty, as can proximity to adjacent roots and the need for perpendicular root-end resection and preparation to include a possible missed lingual canal.

Cone-beam computed tomography

Radiographic examination is an essential component in all aspects of endodontic treatment from diagnosis and treatment planning to assessing outcome (see also Chapter 2). Information gained from conventional films and digital periapical radiographs is limited by the fact that the three-dimensional anatomy of the area is compressed into a two-dimensional image. As a result of superimposition, periapical radiographs reveal limited aspects of the three-dimensional anatomy. In addition, there may also be geometric distortion of the anatomic structures imaged. These problems may be overcome using small volume CBCT imaging techniques, which can produce three-dimensional images of individual teeth and the surrounding tissues. CBCT may be particularly useful in diagnosis and treatment planning for periradicular surgery (Fig. 11.9).[112,163,249,318] The term *cone-beam computed tomography* is often used interchangeably with *cone-beam volumetric tomography* (CBVT).

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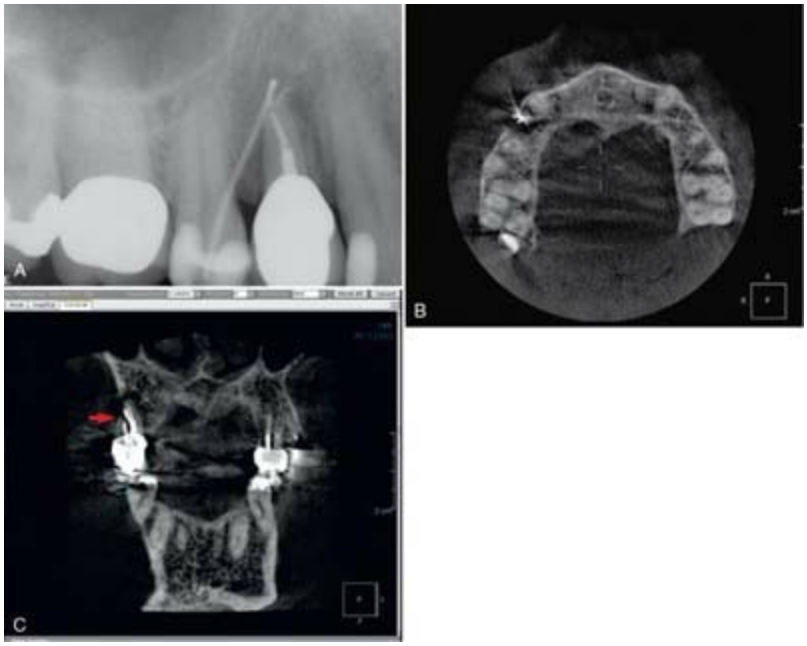

FIG. 11.9 **A,** Periapical radiograph with gutta-percha tracing the sinus tract to tooth #5. Periodontal probings were within normal limits. This tooth was initially referred for exploratory surgery to rule out root fracture. CBCT confirmed root fracture and the treatment plan was changed to extraction with possible bone graft and guided tissue regeneration for ridge augmentation prior to implant placement. **B,** Axial CBCT view demonstrating extent of lesion. **C,** Coronal CBCT view demonstrating root fracture *(red arrow)* and perforation of buccal cortical plate in area of root fracture but intact bone in the cervical area.

Set of three radiographs depict fracture in the root of a tooth obtained using different techniques.

Differences between computed tomography and cone-beam computed tomography imaging

The benefits of three-dimensional medical computed tomography (CT) imaging are already well established in certain dental specialties. Current CT scanners have a linear array of multiple detectors, allowing multiple slices to be taken simultaneously, resulting in faster scan times and often less radiation exposure to the patient.[507] The slices of data are then “stacked” and can be reformatted to obtain three-dimensional images. The high-radiation dose, cost, availability, poor resolution, and difficulty in interpretation have resulted in limited use of CT imaging in endodontics. These issues may be addressed by cone-beam innovations in CBCT technology.

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In 2000, the FDA approved the first CBCT unit for dental use in the United States. Cone-beam technology uses a cone-shaped beam of radiation to acquire a volume in a single 360-degree rotation, similar to panoramic radiography. The volume of acquired images by a CBCT is composed of voxels. Essentially, a voxel is a 3D pixel. Because the data are captured in a volume as opposed to slices, all the voxels are isotropic, which enables objects within the volume to be accurately measured in different directions. Unlike the CBCT voxel, a medical CT voxel is not a perfect cube, and measurements made in multiple planes are not accurate. In addition to increased accuracy and higher resolution, CBCT offers significant scan-time reduction, radiation dose reduction, and reduced cost for the patient.[472,595,606] CBCT systems can be classified into two categories: limited (dental or regional) CBCT or full (ortho or facial) CBCT. The field of view (FOV) of limited CBCT ranges in diameter from 40 to 100 mm, whereas the FOV of full CBCT ranges from 100 to 200 mm.[112] Another difference between the limited CBCT and full CBCT is that a voxel is generally smaller for the limited version (0.1 to 0.2 mm vs. 0.3 to 0.4 mm). Thus limited CBCT systems offer higher resolution and are better suited for endodontic applications. Limited volume CBCT scanners capture small volumes of data that can include just two or three individual teeth. The most important and clinically useful feature of CBCT technology is the highly sophisticated software that allows the huge volume of data collected to be reconstructed. Tomographic slices, as thin as one voxel, may be displayed in a number of ways. One option, for example, is for the images to be displayed in the three orthogonal planes—axial, sagittal and coronal—simultaneously. The axial and proximal (sagittal in the anterior, coronal in the posterior) views are of particular value because they are generally not seen with conventional periapical radiography. The ability to reduce or eliminate superimposition of the surrounding structures makes CBCT superior to conventional periapical radiography.[314]

Scan times are typically 10 to 40 seconds, although the actual exposure time is significantly less (2 to 5 seconds) as scans involve a number (up to 360) of separate, small, individual exposures rather than one continuous exposure. With medical CT scanners, the scanning and exposure times for the skull can be significantly longer. Most CBCT scanners are much smaller than

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medical CT scanners, taking up about the same space as a dental panoramic machine. They are also significantly less expensive than medical CT scanners. With the help of viewer software, the clinician is able to scroll through the entire volume and simultaneously view axial, coronal, and sagittal 2D sections that range from 0.125 to 2 mm thick. Comparing the radiation dose of different CBCT scanners with medical CT scanners may be confusing because different units of radiation dose are often used. There are three basic measurement units in radiation dosimetry: the radiation absorbed dose (D), the equivalent dose (H), and the effective dose (E). The *radiation absorbed dose* is defined as the measure of the amount of energy absorbed from the radiation beam per unit mass of tissue and is measured in joules per kilogram. The *equivalent dose* is defined as a measure that indicates the radiobiologic effectiveness of different types of radiation and thus provides a common unit. The *effective dose* is calculated by multiplying the equivalent dose by different tissue weighting factors, which converts all doses to an equivalent whole-body dose and allows doses from different investigations of different parts of the body to be compared. The unit remains as the Sievert (Sv) and can be used to estimate the damage from radiation to an exposed population. The reported effective doses from CBCT scanners vary, but it can be almost as low as those from panoramic dental x-ray units and can be considerably less than that from medical CT scanners.[398] The higher effective doses from particular models of CBCT scanners is in part due to the larger size of the FOV used as well as the type of image receptor employed. The small volume scanners capture information from a small region of the jaw, approximately the same area as a periapical radiograph. The effective dose has been reported to be in the same range as two to three standard periapical radiograph exposures, whereas the effective dose for a full mouth series of periapical radiographs has been reported to be similar to the effective dose of a large volume CBCT scan.[124,194] If multiple teeth in different quadrants require endodontic evaluation or treatment, a large volume CBCT scan with a large FOV may be more appropriate. If endodontic information is required from multiple teeth in one jaw, a large volume CBCT scan with the FOV only limited to the jaw of interest could be the investigation of choice. This has the advantage of reducing the effective dosage from the full large volume CBCT scan by up to 65%.[321]

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Potential applications of cone-beam computed tomography in the management of endodontic posttreatment disease

Clinical radiographic examinations are usually limited to two-dimensional views captured using radiographic film or digital sensors. Crucial information related to the true three-dimensional anatomy of teeth and adjacent structures is obscured. Even with paralleling techniques, distortion and superimposition of dental structures in periapical views is unavoidable. A major reported advantage of CBCT is the accuracy of measurements in all dimensions.[331] The ability to view thin sagittal, coronal, and axial slices effectively eliminates the problem of superimposition of anatomic structures. For example, roots of maxillary posterior teeth and surrounding tissues can be viewed without superimposition of the zygomatic buttress, alveolar bone, maxillary sinus, and other roots (Fig. 11.10). CBCT enables clinicians to detect changes in apical bone density at an earlier stage when compared to conventional periapical radiographs and therefore has the potential to detect previously undiagnosed periradicular pathoses.[163,314,371] CBCT may also prove to be a useful tool for noninvasive differentiation of periapical cysts and granulomas,[489] a previously unattainable goal using conventional radiographic imaging.

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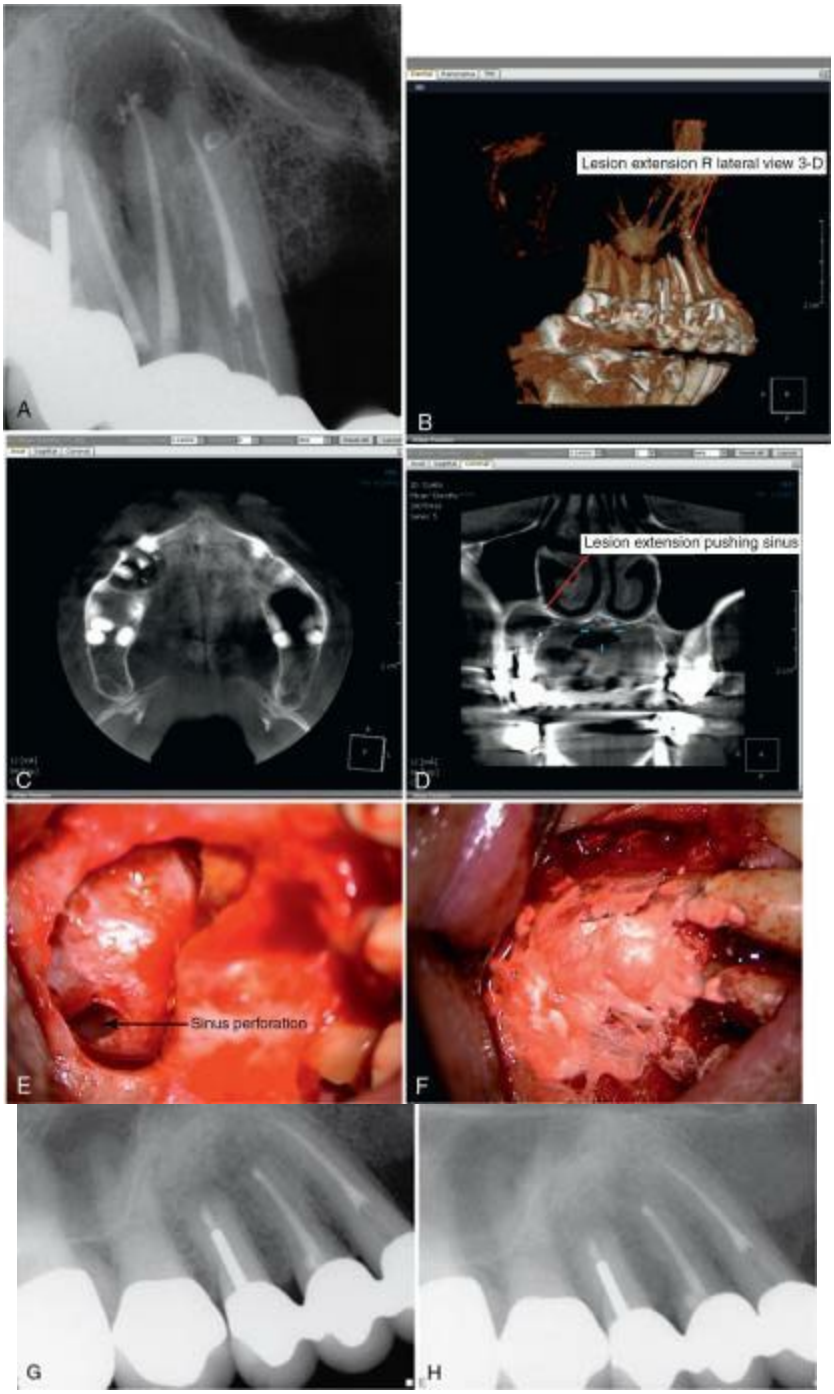

FIG. 11.10 **A,** Preoperative periapical radiograph showing large radiolucency associated with teeth #4, 5, and 6. **B,** CBCT reconstruction *(lateral view).* Rotation of the 3D volume on a computer monitor demonstrated that intact bone was present around the apex of tooth #6 and the lesion was associated only with #4 and 5 (this could not be detected with multiple angled periapical radiographs). **C,** Axial CBCT view demonstrating facial-palatal extent of the lesion. **D,** Coronal CBCT view showing displacement of the sinus membrane (a finding that suggests a sinus membrane perforation will be detected during

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surgery). **E,** Clinical image after surgical access removal of granulation tissue, and root-end resection (note sinus perforation [*arrow*]). **F,** Placement of bone graft and Capset. **G,** Immediate postoperative radiograph. **H,** Two-year follow-up radiograph demonstrating good healing.

Set of eight figures marked A through H depicts evaluation films and results of surgical treatment of periapical defect associated with tooth number 4 to 6.

Three-dimensional imaging allows for clear identification of the anatomic relationship of the root apices to important neighboring anatomic structures such as the mandibular canal, mental foramen, and maxillary sinus (Fig. 11.11). Velvart and colleagues reported that the relationship of the inferior alveolar canal to the root apices could be determined in every case when utilizing medical CT, but in less than 40% of cases when using conventional radiography.[556] It is likely that similar results could be achieved with CBCT using considerably less radiation. Rigolone and coworkers concluded that CBCT may play an important role in periapical microsurgery of palatal roots of maxillary first molars.[442] The distance between the cortical plate and the palatal root apex could be measured using CBCT and the presence of the maxillary sinus between the roots could be assessed. Additional information such as the thickness of the cortical plate, cancellous bone pattern, fenestrations, and inclination of the roots can be obtained prior to surgical entry.[371] Root morphology (shape, size, curvature, and number of canals) can be visualized in three dimensions. Unidentified (and untreated) canals in root-filled teeth can be routinely visualized in axial slices. CBCT has been used to determine the location and extent of invasive external root resorptive defects.[398]

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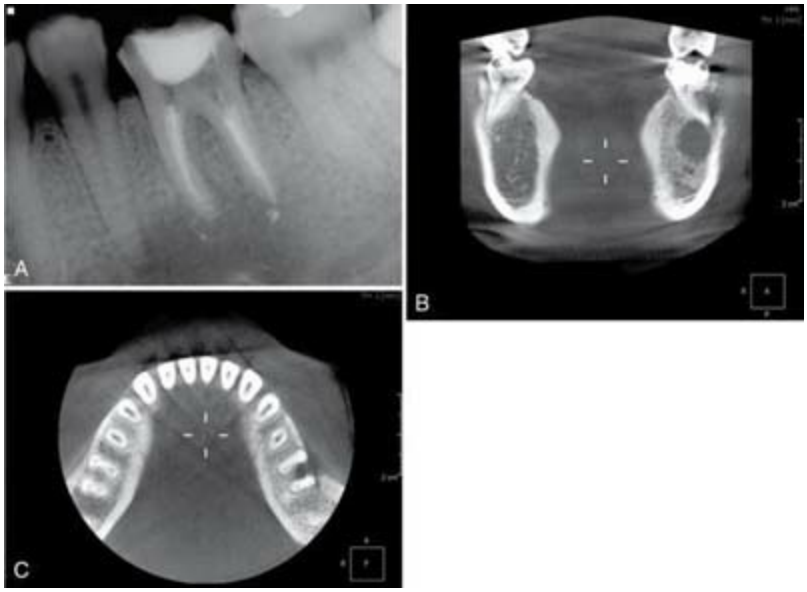

FIG. 11.11 **A,** Periapical radiograph demonstrating persistent periradicular pathosis following nonsurgical retreatment of tooth #19. The extension of the lesion in the vicinity of the mandibular canal and possible involvement of tooth #20 was uncertain based on interpretation of periapical and panoramic radiographs. Tooth #20 responded WNL to pulp vitality test. **B,** Coronal CBCT demonstrates that the periradicular lesion does not extend apical or lingual to the mandibular canal. **C,** Axial CBCT view demonstrating perforation of the buccal cortical plate and extension into, but not through, the furcation of #19 and presence of intact bone around #20.

Set of three radiographs marked A through C conducted to rule out extension of lesion from tooth 19 in the vicinity of mandibular canal to the adjacent tooth.

One of the most useful potential applications of CBCT may be the assessment of endodontic treatment outcomes, both nonsurgical and surgical. When compared to two-dimensional film and digital images, CBCT is dimensionally precise and can detect smaller changes in bone density.[331]

It is worth remembering that CBCT still uses ionizing radiation and is not without risk. It is essential that patient exposure is kept as low as reasonably practical and that justifiable selection criteria for CBCT use are developed. CBCT may be indicated when it is determined that the additional information obtained is likely to result in a more accurate diagnosis and enhanced patient safety.

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Patient preparation for surgery

Informed-consent issues specific to surgery

The general principles of informed consent discussed in Chapter 27 form the basis for informed consent for periradicular surgery. The patient must be thoroughly advised of the benefits, risks, and other treatment options and must be given an opportunity to ask questions. The main consent issues specific to surgical procedures are closely related to the anatomic considerations discussed in the previous section. That is, major neurovascular bundles may be traumatized, and a sinus exposure may occur. Paresthesia after mandibular posterior surgery is uncommon but should be discussed with the patient because this potential complication is a risk that some patients may be unwilling to assume. Postoperative swelling, bruising, bleeding, and infections are possible complications that typically are self-limiting or readily manageable. Although the incidence of serious complications related to surgical procedures is very low, patients should be advised of any risks unique to their situation. Prompt attention to any surgical complications and thorough follow-up are essential from a medical-legal standpoint.

Premedication: Nonsteroidal antiinflammatory drugs, antibiotics, chlorhexidine, and conscious sedation

Administration of an NSAID, either before or up to 30 minutes after surgery, enhances postoperative analgesia.[494] NSAIDs generally have proved more effective in the management of postoperative oral surgery pain than placebo or acetaminophen and codeine combinations.[9,41,143] The combination of preoperative administration of an NSAID and use of a long-acting local anesthetic may be particularly helpful for reducing postoperative pain.[144] Many types of NSAIDs are available, but ibuprofen remains the usual standard for comparison. Ibuprofen (400 mg) provides analgesia approximately equal to that obtained with morphine (10 mg) and significantly greater than that from codeine (60 mg), tramadol (100 mg), or acetaminophen (1000 mg).[346] The analgesic effectiveness of ibuprofen tends to level off at about the 400-mg level *(ceiling effect)*, although a slight increase in analgesic potential may be expected in doses up to 800 mg.

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The value of antibiotic prophylaxis before or after oral surgery is controversial, and the current best available evidence does not support the routine use of prophylactic antibiotics for periradicular surgery.[11] For most patients, the risks of indiscriminate antibiotic therapy are believed to be greater than the potential benefits.[526] The incidence of infection after oral surgery in healthy patients is very low. Peterson reported that only 1% of patients developed infections after third molar extractions.[355] A systematic review of the use of antibiotics to prevent complications after placement of dental implants found that evidence was lacking either to recommend or discourage the use of antibiotics for this purpose.[162] However, the use of prophylactic antibiotics for more invasive procedures, such as orthognathic surgery, has significantly reduced the risk of postoperative infection and complications.[146] Although routine use of prophylactic antibiotics for periradicular surgery is not currently recommended, clinical judgment is important in determining exceptions to the general rule. For example, immunocompromised patients may be good candidates for prophylactic antibiotic coverage. Certain categories of medically complex patients also may benefit from antibiotic coverage. Diabetic patients have shown impaired healing capacity after nonsurgical root canal treatment,[78,175] and a like pattern of delayed or impaired healing may emerge in studies of surgical outcomes. The global problems associated with overprescription of antibiotics are significant and should prompt due caution in the decision on whether to use these drugs prophylactically.

Chlorhexidine gluconate (0.12%) often is recommended as a mouth rinse to reduce the number of surface microorganisms in the surgical field, and its use may be continued during the postoperative healing stage.[7,281,545] Although no solid evidence supports this practice in periradicular surgery, the use of chlorhexidine follows from the general surgical principle of surface disinfection before incision and opening into a body cavity. In addition, chlorhexidine has proved to be a safe, effective adjunct in the treatment of periodontitis, and short-term use (i.e., several days) poses little or no risk. Chlorhexidine may be useful for reducing the risk of postoperative infection after oral surgery,[67,598] although the evidence in this area is conflicting. Postoperative use of chlorhexidine mouth rinse can reduce bacterial growth on sutures and wound margins,[389] but it may interfere with fibroblast

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reattachment to the root surface.[14] A useful empirical regimen is to have the patient rinse for 30 seconds twice a day beginning 1 or 2 days before surgery and continuing until the sutures are removed.

Conscious sedation, either by an orally administered sedative or by nitrous oxide/oxygen inhalation analgesia, may be useful for patients who are anxious about the surgical procedure or dental treatment in general. Benzodiazepines with a short half-life are particularly useful because they generally have a wide margin of safety, good absorption after oral administration, and limited residual sedative effects. When these drugs are used in sedative-hypnotic doses, the blood pressure, pulse, and respiration must be monitored, and states differ in their requirement for additional training or certification of staff members who do this monitoring. With oral conscious sedation, pulse oximetry should be used to monitor the pulse and blood oxygen saturation during the surgery.[84] As with all orally administered drugs, the dosage cannot be closely titrated; therefore the effect of the agent varies somewhat. A typical protocol is a single dose at bedtime the evening before the procedure and a second dose 1 hour before the start of surgery. The patient should not drive to or from the office and should have a responsible adult for assistance as needed. In appropriate doses, benzodiazepines and similar drugs may allow for a more relaxed patient and thus a less stressful surgical experience for both patient and surgeon.

Instruments and operatory setup

The development of microsurgical techniques and new materials has changed the typical surgical instrument tray dramatically. Instruments have been designed to take full advantage of the increased visibility obtained with dental operating microscopes, endoscopes, and orascopes. Better visualization of the surgical site would have limited value without microsurgical instruments, such as ultrasonic tips for root-end preparation and micromirrors for inspecting the root end. Fig. 11.12 shows a typical basic surgery tray arrangement. This setup is not a definitive guide to surgical armamentarium but rather an adequate, efficient starting point for most periradicular surgical procedures. Although the number of instruments can easily be doubled or even tripled, the ease of locating a specific instrument is

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inversely proportional to the number of instruments on a tray. Specialized instruments can be kept readily available in separate sterilized bags or trays and opened as needed. A skilled surgeon can use a wide variety of instruments (Figs. 11.12–11.24) to achieve excellent results.

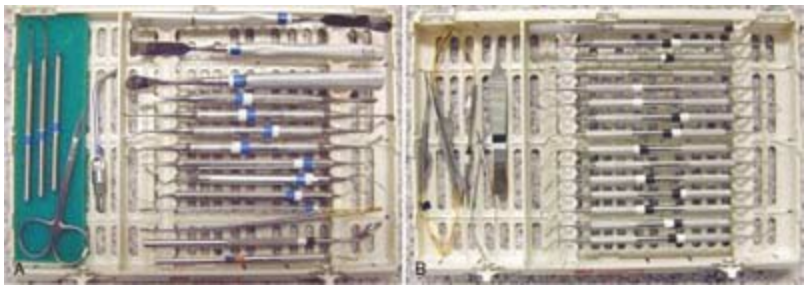

FIG. 11.12 **A,** Basic tray setup for initial surgical access. Surgical instruments shown are distributed by Hu-Friedy ([HF] Chicago), CK Dental Specialities ([CKDS] Orange, CA), EIE ([EIE]), and G. Hartzell & Son ([GHS]). *Left to right* (left section of tray): Small round micromirror (CKDS); medium oval micromirror (CKDS); handle for microscalpel (CKDS); scissors (S18 [HF]); surgical suction tip (GHS). *Top to bottom* (main section of tray): Carr #1 retractor (EIE); Carr #2 retractor (EIE); TRH-1 retractor (HF); periosteal elevator (HF); Ruddle R elevator (EIE); Ruddle L elevator (EIE); Jacquette curette (SJ 34/35 [HF]); spoon curette (CL 84 [HF]); scaler (7/8 [HF]); surgical forceps (TP 5061 [HF]); mouth mirror (HF); periodontal probe (HF). **B,** Instrument tray for root-end filling and suturing. *Left to right* (left section of tray): Two Castroviejo needle holders (Roydent Dental Products, Rochester Hills, MI); Castroviejo scissors (S31 [HF]); micro–tissue forceps (TP 5042 [HF]). *Top to bottom* (main section of tray): Cement spatula (HF); Feinstein super plugger (F1L); microexplorer (CX-1 [EIE]); endoexplorer (DG-16 [EIE]); right Super-EBA Placing & Plugging instrument (MRFR [HF]); left Super-EBA Placing & Plugging instrument (MRFL [HF]); small anterior microburnisher and plugger (HF); small left microburnisher and plugger (HF); small right microburnisher and plugger (HF); medium anterior microburnisher and plugger (HF); medium left microburnisher and plugger (HF); medium right microburnisher and plugger (HF); large anterior microburnisher and plugger (HF); large left microburnisher and plugger (HF); large right microburnisher and plugger (HF).

Close-up view of two surgical trays demonstrate a list of instruments used in the treatment.

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FIG. 11.13 Comparison of microsurgical scalpel *(top)* to #15C surgical blade. Microsurgical scalpels are particularly useful for the intrasulcular incision and for delicate dissection of the interproximal papillae.

A microsurgical scalpel on the top has triangular blade with the hypotenuse as its sharpest edge. A surgical blade at the bottom shows a small, curved cutting edge ideal for making short, precise incisions.

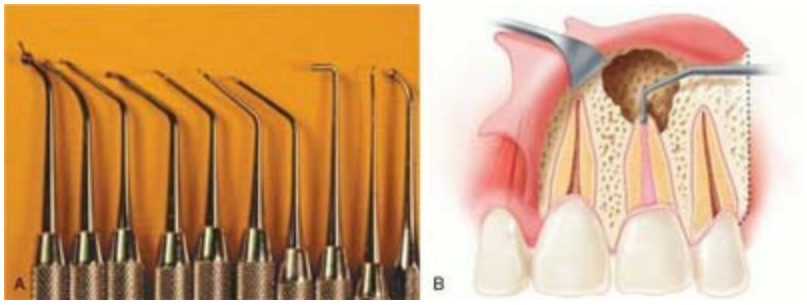

FIG. 11.14 **A,** Microcondensers in assorted shapes and sizes for root-end filling. **B,** The microcondenser should be selected to fit the root-end preparation.

A) A set of micro condensers varying in bent angle of tip, length and shape of the tip.

B) Diagram of interior of mouth shows a micro condenser entering the canal through the root, while flap above is held by a retractor.

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FIG. 11.15 Comparison of standard #5 mouth mirror *(top)* to diamond-coated micromirrors (CK Dental Specialties).

A mouth mirror depicted as a round mirror attached to a handle (left) is compared with three diamond coated micromirrors having tiny, round mirrors varying in size.

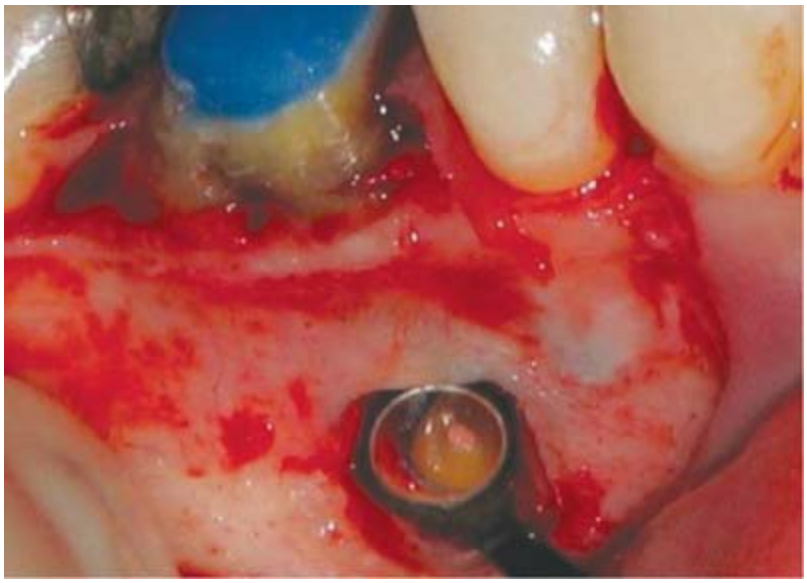

FIG. 11.16 Micromirror used to inspect resected mesial root of a mandibular first molar.

Close-up view of interior of mouth shows a diamond coated micromirror placed at the mesial root of the mandibular first molar.

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FIG. 11.17 Retractors used in periradicular surgery. *Top to bottom,* EHR-1, ER-2, and ER-1 (equivalent to Carr #2 and #1 retractors) (CK Dental Specialties).

Three types of retractors are shown as follows:

EHR-1 shows a curved tip; ER-2 shows a triangular top having a curved tip; and ER-1 shows rectangular flap holder having a curved edge.

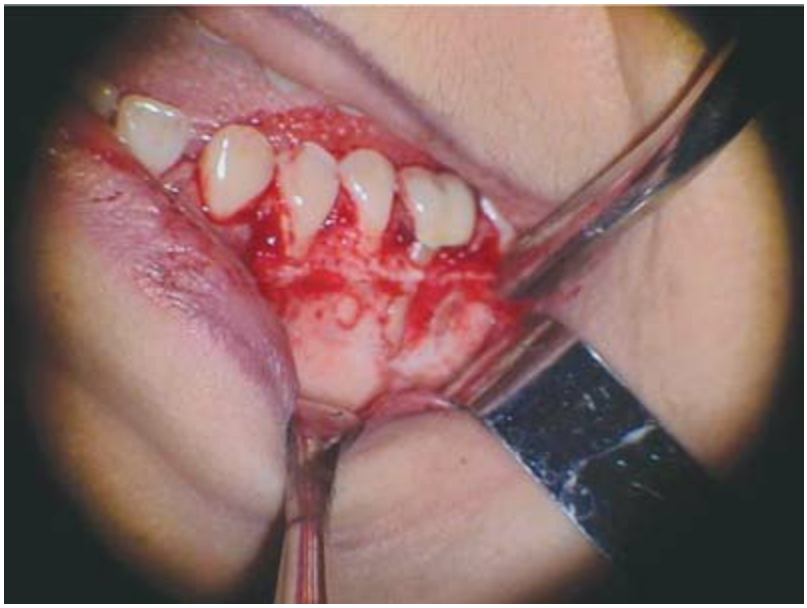

FIG. 11.18 Retractors positioned to expose the surgical site and protect adjacent soft tissues from injury. Care must be taken to rest the retractors only on bone, not on the reflected soft-tissue flap or on the neurovascular bundle as it exits the mental foramen.

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Close-up of interior of mouth shows three retractors placed near tooth, at edge of lip and at lower lip to hold flap of skin to expose gingiva and affected tooth.

FIG. 11.19 Teflon sleeve and plugger specially designed for placement of mineral trioxide aggregate (DENTSPLY Tulsa Dental Specialties).

Teflon sleeve and plugger is depicted as a Teflon bent shaped top attached to a plastic handle with tip covered with a small cylindrical piece of plastic.

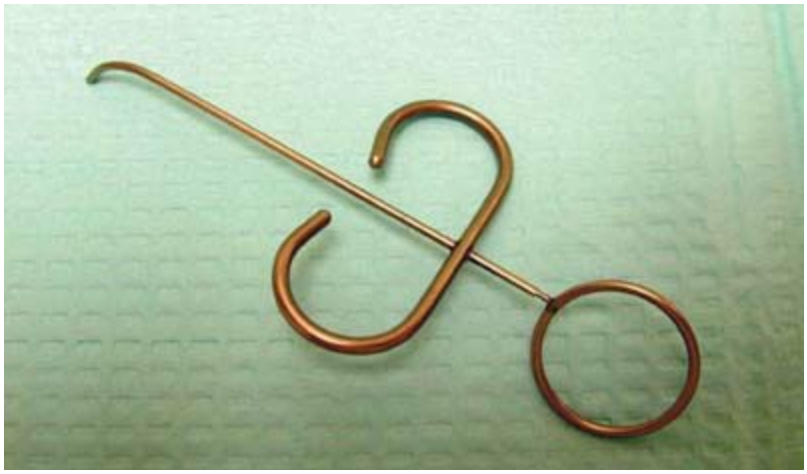

FIG. 11.20 Messing gun–type syringe (CK Dental Specialties) can be used for placement of various root-end filling materials.

The Messing-gun syringe is depicted as a narrow orifice having a plunger with a thumb ring and a curved barrel, with bevel containing orifice shown slightly

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curved.

FIG. 11.21 Another delivery system designed specifically for mineral trioxide aggregate placement (Roydent). Kit includes a variety of tips for use in different areas of the mouth and a single-use Teflon plunger.

Syringe is depicted as a narrow orifice having a plunger with a thumb ring and a straight barrel, with bevel containing orifice shown slightly curved. A needle is kept next to it.

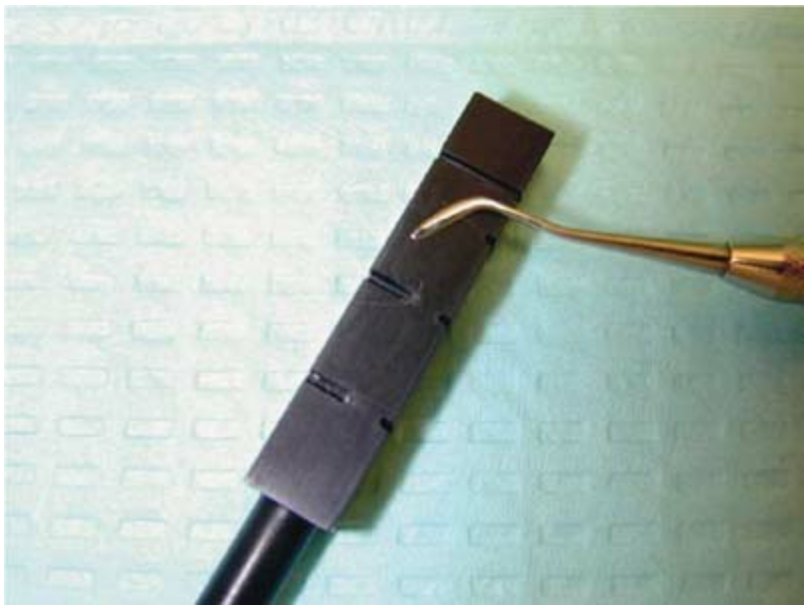

FIG. 11.22 Hard plastic block with notches of varying shapes and sizes (G. Hartzell & Son). MTA is mixed on a glass slab to the

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consistency of wet sand and then packed into a notch. The applicator instrument is used to transfer the preformed plug of MTA from the block to the root end.

A hard plastic block having a plate divided into three rectangular block with a tip of a syringe pointed towards a notch.

FIG. 11.23 Placement of a root-end filling.

An illustration of interior of mouth shows flap of gingiva held with a retractor while a tip of microcondensor points at the root of canal of the tooth.

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FIG. 11.24 Surgeon, assistant, and patient positioned for initiation of surgery. The patient should be given tinted goggles or some other form of eye protection before the procedure begins.

Two doctors look into an eyepiece of a device placed right above face of a patient on a stretcher wearing dark shades. On the doctor is shown adjusting knobs of the device.

Periradicular surgery can be performed without the benefit of enhanced magnification and illumination; however, those who use microscopes, endoscopes, and orascopes report dramatically improved visualization and control of the surgical site.[39,92,280,350] Studies reporting the highest surgical success rates and those cited previously in this chapter all involved some form of enhanced magnification and illumination as part of the standard operating protocol. A systematic review and meta-analysis found that the outcome for endodontic surgery performed with high magnification and illumination (microscope or endoscope) was significantly higher than surgery performed with loupes only or no magnification.[482]

Local anesthesia for surgery

Local anesthesia for surgical root canal procedures differs from that for nonsurgical root canal treatment, primarily in the need for localized hemostasis in addition to profound local anesthesia. In fact, use of a local anesthetic with a vasoconstrictor may be the single most important local

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measure to help control hemorrhage and provide a clear surgical field. Otherwise, the same regional block and local infiltration techniques used for nonsurgical treatment are used for surgical root canal procedures. Infiltration of the surgical site with a local anesthetic containing 1:50,000 epinephrine is the technique of choice to obtain vasoconstriction and hemostasis.[280] The local anesthetic is first slowly deposited in the buccal root apex area of the alveolar mucosa at the surgical site and extended two or three teeth on either side of the site. Usually palatal or lingual infiltration is also required, although this requires a much smaller amount of local anesthetic than the primary buccal infiltration. After the injections for anesthesia, the surgeon should wait at least 10 minutes before making the first incision.

Long-acting local anesthetics (e.g., 0.5% bupivacaine with 1:200,000 epinephrine) have been shown to reduce postoperative pain and analgesic use after surgical removal of impacted third molars.[125,204] However, use of a local anesthetic with 1:200,000 epinephrine may result in greater blood loss during surgery.[234,493] To maximize postoperative analgesia and minimize intraoperative bleeding, a local anesthetic can be used with higher epinephrine concentrations (1:100,000 or 1:50,000) for the primary surgical anesthesia and supplemented with one cartridge of long-acting local anesthetic immediately after surgery. Long-lasting local anesthetics are particularly beneficial in mandibular surgery but much less so for surgery in the maxillary arch.

Every effort must be made to ensure profound local anesthesia before surgery begins. Usually a minimum of 10 to 20 minutes is required from the time of injection to the start of surgery to ensure both profound local anesthesia and adequate vasoconstriction for hemostasis. The patient should be asked about the usual signs of soft-tissue anesthesia, and a sharp explorer can be used to test the surgical area for sensation. Even when the surgeon pays careful attention to local anesthetic technique, a patient sometimes has inadequate anesthesia or loss of anesthesia during the surgical procedure. Providing supplemental infiltration anesthesia is difficult after a full-thickness flap has been reflected. A supplemental block injection may be useful for mandibular teeth and maxillary posterior teeth. In the maxillary anterior area, a palatal approach to the anterior middle superior nerve may be helpful. The key to this approach is slow injection of approximately 1 mL of

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local anesthetic in the area of the first and second maxillary premolars midway between the gingival crest and the palatal midline. An intraosseous injection also may be used to regain lost anesthesia, but even when it is effective, the area of local anesthesia often is smaller than desired for a surgical procedure. As a last resort, the procedure can be terminated short of completion and the patient can be rescheduled for surgery under sedation or general anesthesia.

Surgical access

The goals of periradicular surgery are to access the affected area, remove the diseased tissue, evaluate the root circumference and root canal system, and place a biocompatible seal in the form of a root-end filling that can stimulate regeneration of the periodontium. The formation of new cementum on the surgically exposed root surface and on the root-end filling material is essential to regeneration of the periodontium.

Successful endodontic surgery requires the surgeon to use several conceptual elements in planning the procedure. A vision of the immediate postoperative surgical end point (i.e., replacement of the reflected tissues) is essential for designing each phase of the surgery. Visualization of a three-dimensional image of the surgical procedure allows the surgeon to anticipate and prepare for unusual circumstances. In surgical root canal treatment, once a procedure is started, it must be completed uninterrupted within a limited time; this is a paramount difference between nonsurgical and surgical root canal treatment. For this reason, it is absolutely essential that the surgeon plan the procedure thoroughly and include alternative plans of action that anticipate unusual findings during the process of discovery.

Several general principles are important for designing the access to a diseased region: (1) the surgeon must have a thorough knowledge of the anatomic structures in relation to each other, including tooth anatomy; (2) the surgeon must be able to visualize the three-dimensional nature of the structures in the soft and hard tissue (this reduces unnecessary tissue damage); (3) the trauma of the surgical procedure itself must be minimized, which includes the preservation of the tooth and supporting structures; and (4) the tissue and instruments must be manipulated within a limited space with the aim of removing diseased tissues and retaining healthy tissues.

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Soft-tissue access

In designing the soft-tissue access window to the diseased tissue, the surgeon must take into consideration various anatomic features, such as frenum-muscle attachments, the width of attached gingiva, papillary height and width, bone eminence, and crown margins. The supraperiosteal blood vessels of the attached gingiva extend from the alveolar mucosa and run parallel to the long axis of the teeth, lying in the reticular layer superficial to the periosteum.[172] A vertical, rather than an angled, releasing incision severs fewer vessels,[325] reducing the possibility of hemorrhage. Also, the blood supply to the tissue coronal to the incision is not compromised,[477] which prevents localized ischemia and sloughing of these tissues. Ultimately, the result is less bleeding during the procedure and enhanced healing. For these reasons, an angled releasing incision is contraindicated in periradicular surgery.

Vertical incision

The general principles for placement of a vertical relieving incision are as follows:

1. The incision should be made parallel to the supraperiosteal vessels in the attached gingiva and submucosa (Fig. 11.25).
2. No cuts should be made across frenum and muscle attachments.
3. Frenum and muscle attachments should not be located in the reflected tissue, if possible.
4. The incision should be placed directly over healthy bone.
5. The incision should not be placed superior to a bony eminence.
6. The dental papilla should be included or excluded but not dissected.
7. The incision should extend from the depth of the vestibular sulcus to the midpoint between the dental papilla and the horizontal aspect of the buccal gingival sulcus.

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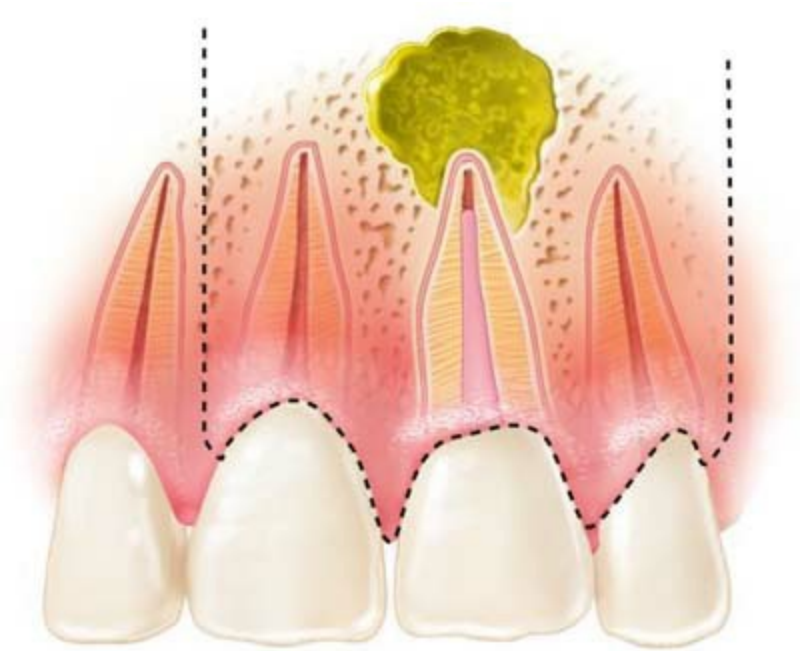

FIG. 11.25 Intrasulcular incision with two vertical releasing incisions (rectangular flap).

An illustration of interior of mouth shows a lesion on the apical foramen of tooth at the center, with a dashed rectangular flap drawn parallel to supraperiosteal vessels covering the junction of the submucosa and the attached gingiva.

Horizontal incision

Three types of horizontal incisions can be used to gain access to a surgical site in hard tissue:

- *An intrasulcular incision that includes the dental papilla.* This incision extends from the gingival sulcus through the PDL fibers and terminates at the crestal bone of the alveolar bone proper. The incision then passes in a buccolingual direction adjacent to each tooth of the dental papilla and includes the midcol region of each dental papilla. Thus the entire dental papilla is completely mobilized.
- *An intrasulcular incision that excludes the dental papilla (papillary-based incision,* Fig. 11.26). This technique consists of a shallow first incision at the base of the papilla and a second incision directed to the crestal bone.

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- *An incision made in the attached gingiva (a submarginal or Ochsenbein-Luebke flap).*[322] With this technique, at least 2 mm of attached gingiva must be retained to prevent mucogingival degeneration.[296] Consequently, the incision must be placed at least 2 mm from the depth of the gingival sulcus. Extensive periodontal probing should be done to establish the depth of the gingival sulcus before the incision is made. The average width of the attached gingiva is 2.1 to 5.1 mm in the maxilla and 1.8 to 3.8 mm in the mandible.[10,199,341,563] It is widest over the central and lateral incisors, narrows over the canine and the first premolar, and then widens over the second premolar and the first molar. These variances were similar for both the maxilla and mandible.[517] Overall, this incision technique has a narrow margin of safety. It generally is recommended for use in the maxilla, especially where the aesthetics of existing crown margins are a concern.

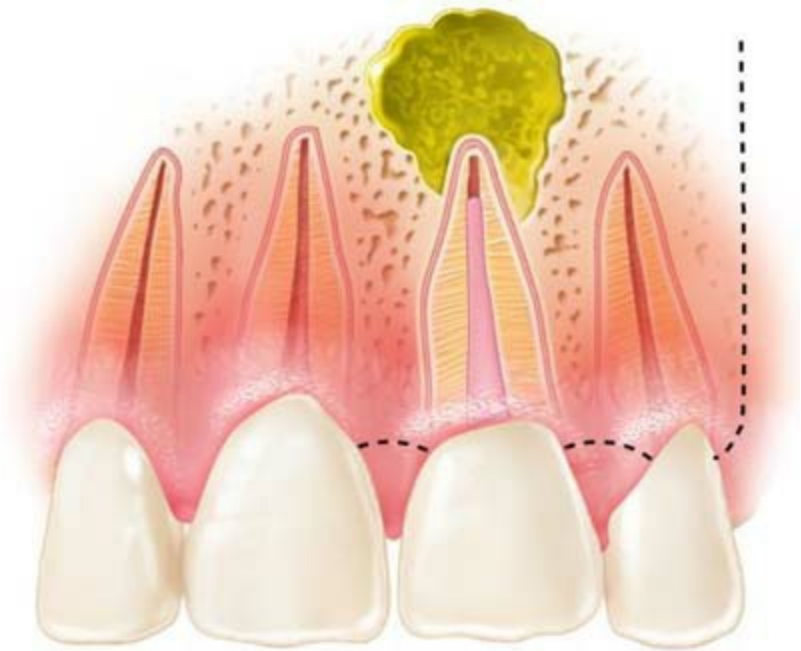

FIG. 11.26 Papillary-based incision with one vertical releasing incision.

An illustration of interior of mouth shows a lesion on the apical foramen of tooth at the center, with first incision at the base of papilla and second incision directed to the crestal bone.

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Studies have compared the incision techniques that include and exclude the dental papilla in patients with healthy marginal periodontal conditions.[554,555,557] These researchers found that the papilla base incision resulted in rapid, recession-free healing. In contrast, complete mobilization of the papilla led to a marked loss of papilla height. The authors suggested that use of the papilla base incision in aesthetically sensitive regions could help prevent papilla recession and surgical cleft, or double papilla.

Flap design

Combinations of vertical and horizontal incisions are used to achieve various flap designs (Figs. 11.27 and 11.28). The full mucoperiosteal and limited mucoperiosteal are the two major categories of flap design used during periradicular surgery, the main differentiating feature being the position of the horizontal incision. In each case the entire body of soft tissue is reflected as one unit and includes the alveolar mucosa, the gingival tissues, and the periosteum. The number and position of the vertical relaxing incisions therefore governs the major variation in design:

1. Full mucoperiosteal (intrasulcular incision including the dental papilla or papillary based) (see Figs. 11.27 and 11.28):
    a. Triangular: one vertical relieving incision
    b. Rectangular: two vertical relieving incisions
    c. Trapezoidal: two angled vertical relieving incisions
    d. Horizontal: no vertical relieving incision
2. Limited mucoperiosteal (Figs. 11.29 and 11.30):
    a. Curve submarginal (semilunar)
    b. Freeform rectilinear submarginal (Ochsenbein-Luebke)

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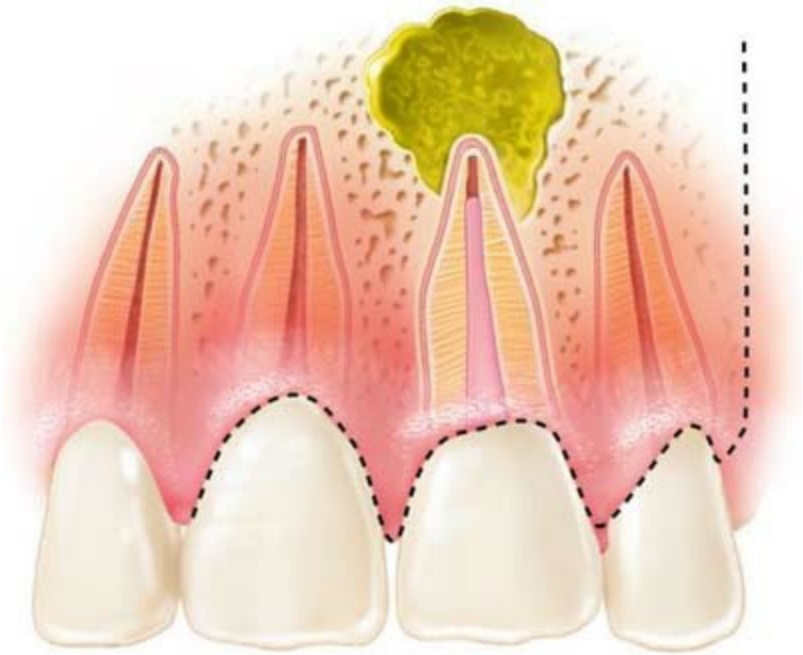

FIG. 11.27 Intrasulcular incision with one vertical releasing incision (triangular flap).

An illustration of interior of mouth shows a lesion on the apical foramen of tooth at the center, with first incision at the base of papilla including gingival sulcus and second incision directed to the crestal bone.

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FIG. 11.28 Reflection of the triangular flap to expose the root-end area.

An illustration shows triangular flap of gingiva held by a retractor to expose the root end area of the affected tooth at the center.

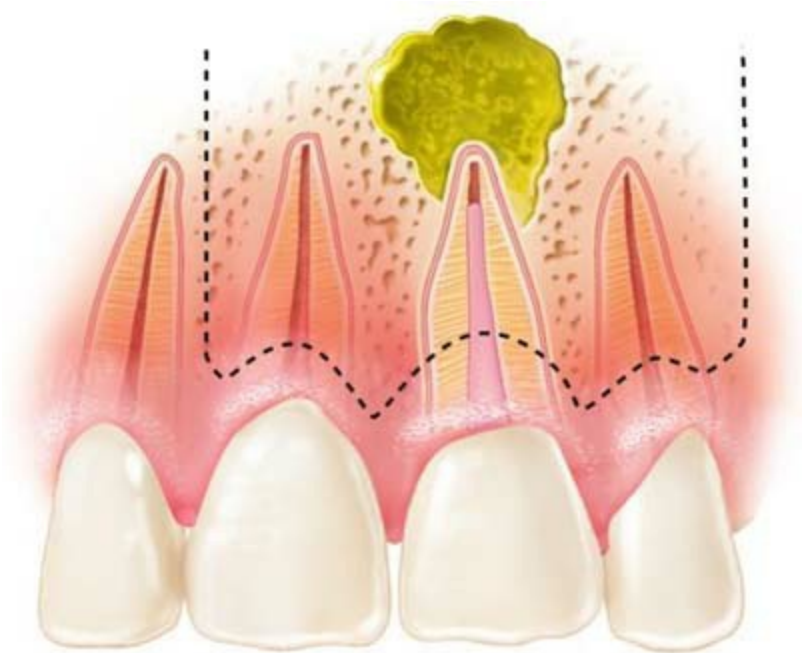

FIG. 11.29 Submarginal (Ochsenbein-Luebke) flap.

An illustration of interior of mouth shows a lesion on the apical foramen of tooth at the center, with a dashed rectangular flap drawn parallel to supraperiosteal vessels covering the junction of the submucosa and the attached gingiva. The incision at base is placed at 2 millimeters from the depth of the gingival sulcus.

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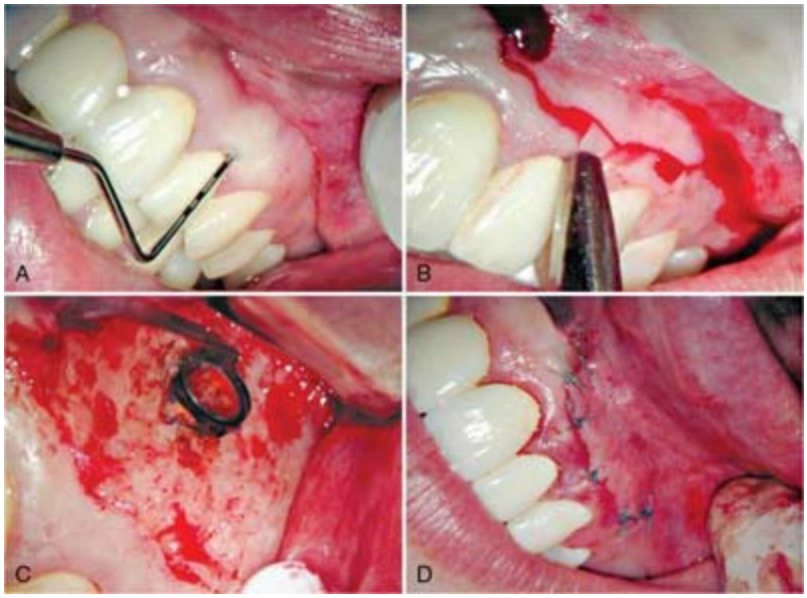

FIG. 11.30 Clinical case involving a submarginal incision and flap. **A,** Periodontal probing of the entire area was performed before this type of flap was selected and the incision started. A submarginal incision often is used in aesthetic anterior areas of the mouth where postoperative gingival recession might expose the crown margins. **B,** The incision should be at least 2 mm apical to the depth of the sulcus. **C,** With the flap reflected, osteotomy and root-end resection were performed. Methylene blue dye was placed to mark the outline of the root end and to help identify cracks or fractures before root-end cavity preparation and filling. **D,** The flap was repositioned and sutured with 5-0 Tevdek.

Set of four photographs marked A to D depict steps involved in the a surgical procedure including a submarginal incision and flap, which includes periodontal probing, submarginal incision, flap reflection, and flap repositioned and sutures.

Source: (Courtesy Dr. Martin Rogers.)

Tissue reflection

Elevation and reflection of the entire mucoperiosteal complex, maintaining the microvasculature in the body of the tissue flap, increases hemostatic control during surgery. Tissue reflection should begin from the vertical releasing incision at the junction of the submucosa and the attached gingiva (Figs. 11.31 and 11.32). By initiating the reflection process at this point, damage to the delicate supracrestal root-attached fibers is avoided. Using a flap reflection technique that reduces reflective tissue forces in the

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intrasulcular incisional wound and avoids curettage of the root surface conserves the root-attached tissues and helps prevent apical down-growth of epithelium and loss of soft-tissue attachment.[222] Force should be applied such that the periosteum and superficial tissues are reflected as a complete unit. Using a gentle rocking motion, the surgeon initially should reflect the tissue in a horizontal direction.[213] The underlying bone of the cortical plate is irregular, and it is critical to avoid damaging the fragile tissues during elevation.

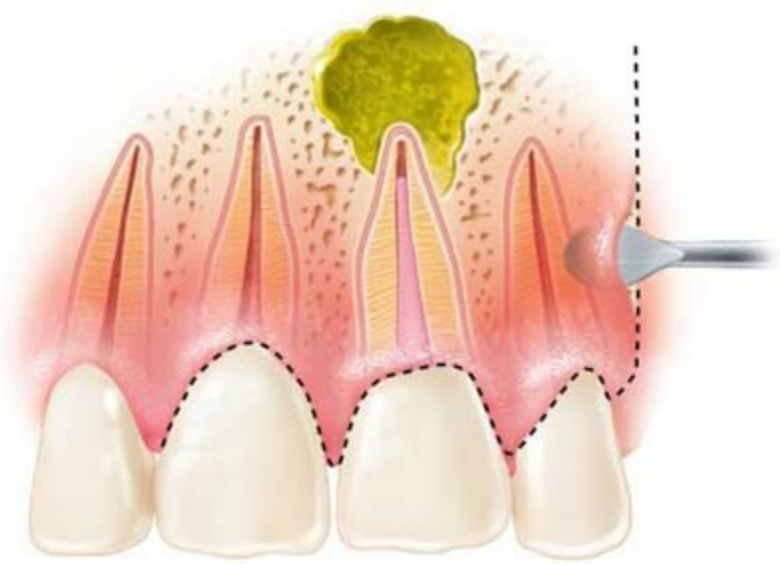

FIG. 11.31 Elevator placed in the vertical incision for the first step in undermining flap reflection.

An illustration of maxillary jaw shows a lesion present at the apical foramen of second tooth from the right, with an elevator placed in the vertical incision.

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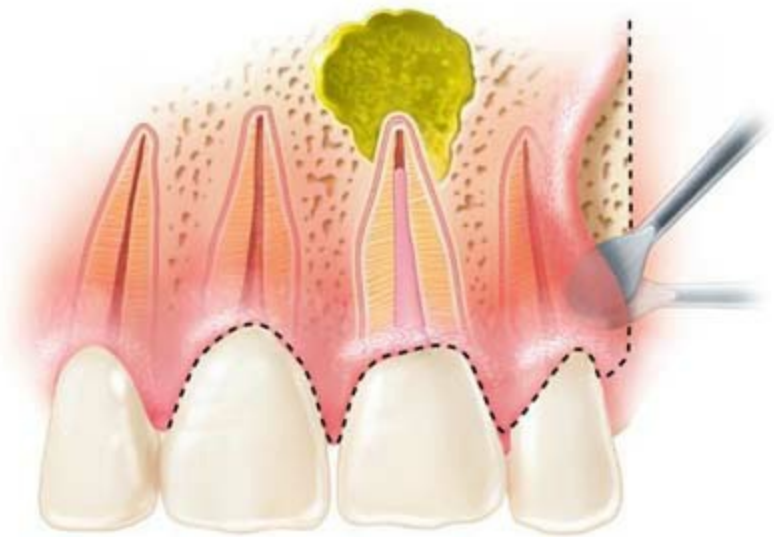

FIG. 11.32 Continuation of reflection of full-thickness flap.

An illustration of maxillary jaw shows a lesion present at the apical foramen of second tooth from the right, with a retractor placed above an elevator placed in the vertical incision.

The operator should take great care not to slip during the tissue reflection process by using an appropriate instrument stabilized with adequate finger support. Slipping can result not only in a puncture of the immediate overlying tissue but also in damage to the surrounding structures.

As space permits, the elevator should be directed coronally, undermining the attached gingiva. As the interdental papilla is approached, a narrower instrument may be required to undermine and gently elevate the tissue in this region, to avoid crushing the delicate free gingival tissues. This process should be continued gradually until the osseous tissues overlying the diseased tooth structure have been adequately exposed. Generally, elevation of the flap 0.75 cm apical to the estimated apex of the root should allow adequate space to perform the surgical procedure.

No single instrument is essential for the flap elevation procedure, because every instrument has both advantages and disadvantages. Surgeons should familiarize themselves with the various instruments available.

Tissue retraction

After the tissue is reflected, it must be retracted to provide adequate access for bone removal and root-end procedures. The main goals of tissue

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retraction are to provide a clear view of the bony surgical site and to prevent further soft-tissue trauma. Accidental crushing of the soft tissues leads to more postoperative swelling and ecchymoses.[222,223]

The general principles of retraction are as follows: (1) retractors should rest on solid cortical bone; (2) firm but light pressure should be used; (3) tearing, puncturing, and crushing of the soft tissue should be avoided; (4) sterile physiologic saline should be used periodically to maintain hydration of the reflected tissue; and (5) the retractor should be large enough to protect the retracted soft tissue during surgical treatment (e.g., prevent it from becoming entangled in the bone bur). No one retractor suffices for all surgical procedures, therefore the surgeon should have a selection of retractors available for the various situations that arise during surgery. If difficulty is encountered in stabilizing the retractor, a small groove can be cut into the cortical plate to support it.

Hard-tissue access

Two biologic principles govern the removal of bone for hard-tissue access to diseased root ends: (1) healthy hard tissue must be preserved, and (2) heat generation during the process must be minimized.

Temperature increases above normal body temperature in osseous tissues are detrimental. Heating osseous tissue to 117° to 122°F (47° to 50°C) for 1 minute significantly reduces bone formation and is associated with irreversible cellular damage and fatty cell infiltration.[161,338] Two critical factors determine the degree of injury: how high the temperature increases and how long it remains elevated. As the temperature rises above 104°F (40°C), blood flow initially increases. It stagnates at 198°F (46°C) applied for 2 minutes. Heating osseous tissue to 133°F (56°C) deactivates alkaline phosphatase.[422,423] Studies using animal bone have shown that at temperatures above 109°F (42.5°C), for every 1°C elevation in temperature, the exposure time for the same biologic effect decreases by a factor of approximately 2.[158-161] Temperatures above 117°F (47°C) maintained for 1 minute produce effects similar to those at 118°F (48°C) applied for 30 seconds. This correlation means that the decisive exposure time declines quickly as the temperature increases. Temperatures above 127°F (53°C) applied for less than 1 second can adversely affect osteogenesis.[158-161]

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Several factors determine the amount of heat generated during bone removal, including the shape and composition of the bur, the rotational speed, the use of coolant, and the pressure applied during cutting.

The round bur has the best shape for removing osseous tissue, and it should be used with a gentle brushstroke action.[519] This type of bur also readily allows access of coolant to the actual cutting surfaces. Studies comparing the heat generated with round and fissure burs found more favorable results with the round burs.[85,111,216,338,360] Cutting with round burs produced a wound site with less inflammation, which is more favorable for rapid wound healing. Although fissure-type burs cut efficiently on the sides, the tip of the bur is very inefficient because it allows no coolant access. The net result is increased inflammation and a reduced healing response.

Use of a diamond bur to remove osseous tissue is inefficient and slows ultimate wound healing. Because of its larger surface area, more of a diamond bur is in contact with the bone tissue. As a result, less coolant reaches the cutting surface, and the bur has a greater tendency to become clogged with residual bone fragments. The net effect is greater heat generation, increased inflammation, and reduced healing.[85,586]

Use of a coolant during bone cutting is essential. If an appropriate irrigant is not used, temperatures can exceed those known to impair bone healing[274]; histologically, healing can be delayed up to 3 weeks.[169] It also is critical that the coolant reach the cutting surface. Temperatures can rise above 212°F (100°C) when excess pressure is applied during cutting; this burrows the bur into the bone, where little or no irrigant can reach the cutting tip,[519] hence the recommendation for a gentle brush-stroke technique.[213] Favorable results are obtained with these provided the surgeon follows the basic tenet of minimizing heat generation: using a round fluted bur with coolant and a brushstroke technique. A high-speed handpiece that exhausts air from the base rather than the cutting end is recommended to reduce the risk of air embolism (Fig. 11.33).

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FIG. 11.33 Surgical handpiece with 45-degree angle head and rear air exhaust (Impact Air 45).

A surgical instrument shows a 45-degree head containing a hook attached to a handpiece.

Periradicular curettage and biopsy

Most periradicular lesions originate in the pulp and can be classified histopathologically as granulomas or cysts.[53,58,292,295,297,359,367,382,488] Histologically, such lesions consist mainly of granulation tissue associated with angiogenesis, fibroblasts, connective tissue fibers, and inflammatory cells. Foreign material, cholesterol clefts,[365,370] and stimulated strands of epithelium also may be present. The stimulated epithelium can form into a stratified, squamous epithelium–lined cystic cavity.[366] These periradicular lesions (granulomas and cysts) are inflammatory lesions that develop in response to irritation caused by intraradicular and extraradicular microorganisms associated with the root canal system[363,368] or by foreign materials forced into the periradicular tissues.[600]

An important aspect of periradicular surgery is the removal of diseased tissue associated with the root apex. Because a large portion of this tissue is reactionary, the focus of surgical root canal treatment is removal of the irritant or diseased tissues. Histologically, an inflammatory periradicular lesion is similar to healing granulation tissue. If the irritant can be readily

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identified and successfully eliminated, it is not always necessary to completely curette all the inflamed periradicular tissues during surgery.[306] This is especially true when complete removal might result in injury to neural or vascular tissues. In addition to removing diseased tissue, periradicular curettage provides visibility and accessibility to facilitate treatment of the apical root canal system or the removal of foreign materials in the periradicular tissues.

The need for a histopathologic assessment of all tissues removed from the body cannot be overstated. Although only a small percentage of periradicular lesions are associated with pathoses other than a periradicular cyst or granuloma, all lesions must be diagnosed definitively because of the potential gravity of the few rare diseases associated with periradicular lesions.[12,121,185,383,409]

The technical aspect of removing soft tissue from the bony crypt varies among surgeons and clinical settings. Various bone and periodontal curettes are available for this purpose, and no one instrument suffices for all cases. Regardless of the instrument selected, the basic principles are the same. A sharp instrument is always preferable to a blunt instrument. The soft-tissue lesion first should be peeled away from the osseous crypt, starting at the lateral borders. This can be accomplished efficiently by using the curette with the concave surface facing the internal wall of the osseous crypt. Once the soft-tissue lesion has been separated from the osseous crypt to the point where the crypt changes its convexity, the curette can be used in a scraping manner to remove the remainder of the lesion from the medial wall of the osseous defect.

Localized hemostasis

Localized hemostasis during periradicular surgery is essential to successful management of the resected root end. Appropriate hemostasis during surgery minimizes surgical time, surgical blood loss, and postoperative hemorrhage and swelling.[213] The hemostatic agents used during endodontic surgery are intended to control bleeding from small blood vessels or capillaries. Localized hemorrhage control not only enhances the visibility and assessment of the root structure but also ensures the appropriate environment for

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placement of the current root-end filling materials and minimizes root-end filling contamination.

Many hemostatic agents have been advocated for use during surgery, and the action of these agents, their ability to control bleeding, and their effect on healing vary considerably. They generally aid coagulation by inducing rapid development of an occlusive clot, either by exerting a physical tamponade action or by enhancing the clotting mechanism and vasoconstriction (or both). No one local hemostatic agent is ideal; each has disadvantages. Therefore further investigation is indicated to find the ideal local hemostatic agent.

Preoperative considerations

A thorough review of the patient's body systems and medical history increases the likelihood of detecting an undiagnosed condition that might affect hemostasis during periradicular surgery. Review of the patient's medications, both prescribed and OTC drugs, is essential. Many OTC drugs can affect the clotting mechanism. The patient's vital signs (i.e., blood pressure, heart rate, and respiratory rate) should be assessed. Vital signs also can be used to monitor anxious patients. An increase in blood pressure and heart rate above a patient's known normal values indicates increased stress or poorly controlled hypertension. Easing the patient's anxiety before surgery reduces the possible hemostatic-potentiating effect of elevated cardiac output during surgery.[156] Anxiety and stress can be alleviated with planning, sedation, and profound local anesthesia.

Local hemostatic agents

Collagen-based materials

Various collagen-based hemostatic agents are available for use as local hemostatic agents. The principal differences are in the microstructure and density of the collagen. Collagen can act as a mild allergen, but the problem of allergenization and unwanted tissue reaction does not occur when highly purified animal collagen is used.[49] The mechanisms by which collagen products help achieve hemostasis involves stimulation of platelet adhesion, platelet aggregation and release reaction,[270,271] activation of factor XII

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(Hageman factor),[335,336] and mechanical tamponade by the structure that forms at the collagen-blood wound interface. Collagen shows minimal interference in the wound healing process, with a limited foreign body reaction.[215] It does not increase the incidence of infection and only slightly delays early bone repair.[246] Osseous regeneration in the presence of collagen typically proceeds uneventfully, without a foreign body reaction.[168]

Collagen-based materials can be difficult to apply to the bony crypt because they adhere to wet surfaces, particularly instruments and gloves.[466] Several collagen-based products are commercially available. They include CollaCote (Integra Life Sciences, Plainsboro, NJ) (Fig. 11.34, *A*), CollaStat (American Medical Products Corp, Eatontown, NJ), Hemocollagene (Septodont, United Kingdom) and Instat (Ethicon, Somerville, NJ). These materials act in essentially similar ways, and the surgical area undergoes a similar healing pattern.[485,504] Overall, studies of wound healing with collagen-based hemostatic agents have shown favorable results.

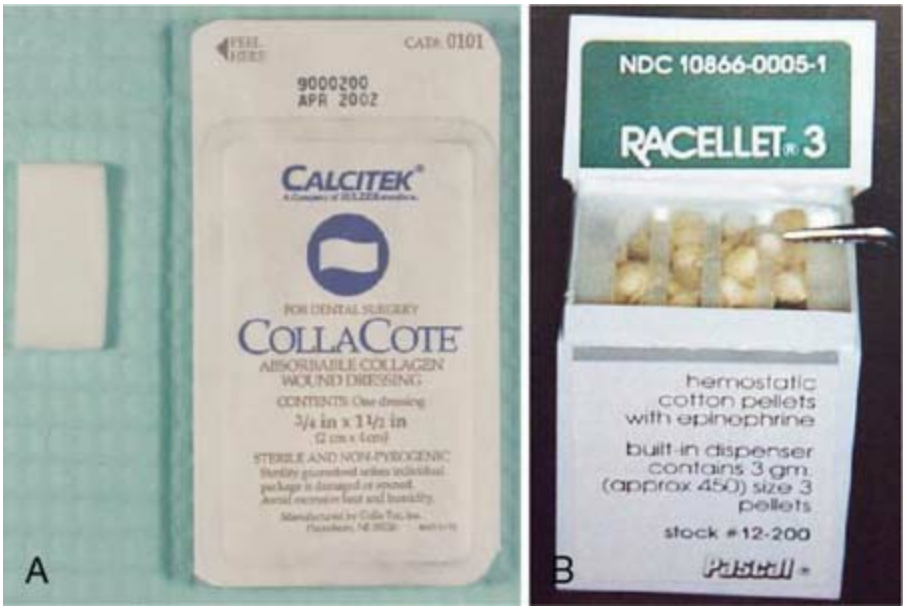

FIG. 11.34 **A,** Absorbable collagen (CollaCote) is a convenient, biocompatible packing material for localized hemostasis. **B,** Cotton pellets impregnated with racemic epinephrine (Racellet) also may be used for localized hemostasis.

A) A package of Calcitex by Colla Cote.

B) An opened package of Racellet-3 reading text: Hemostatic cotton pellets

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with epinephrine built-in dispenser contains 3 grams (approx. 450) size 3 pellets.

Surgicel

Surgicel (Ethicon) is a chemically sterilized material prepared through oxidation of regenerated alpha cellulose (oxycellulose). The basic element of Surgicel is polyanhydroglucuronic acid, which is spun into threads and then woven into gauze. Surgicel has a pH of 3. If the material is maintained in the wound for up to 120 days, a pH this low could slow healing.[408] It is primarily a physical hemostatic agent, which acts as a barrier to blood and then becomes a sticky mass that serves as an artificial coagulum. It does not enhance the clotting cascade through adhesion or aggregation of platelets. Surgicel is retained in the surgical wound,[373] and healing is retarded, with little evidence of resorption of the material at 120 days.[57] Use of Surgicel in extraction sockets resulted in greater postoperative pain compared with a control in a split mouth–designed study.[408]

Gelfoam

Gelfoam (Pharmacia, Peapack, NJ) is a gelatin-based sponge that is water insoluble and biologically resorbable. It stimulates the intrinsic clotting pathway by promoting platelet disintegration and the subsequent release of thromboplastin and thrombin.[164] The initial reaction to Gelfoam in the surgical site is a decrease in the rate of healing. Extraction sockets containing Gelfoam showed a greater inflammatory cell infiltrate, marked reduction in bone ingrowth, and a foreign body reaction at 8 days.[74] However, these effects were transitory and did not impair long-term bone healing.[392]

Bone wax

Historically, bone wax has been advocated for controlling both hemostasis and debris in the bony crypt during periradicular surgery.[480] It is a nonabsorbable product composed of 88% beeswax and 12% isopropyl palmitate. Healing with bone wax is best described as poor. The bony crypt typically contains fibrous connective tissue and has no bony or hematopoietic tissue. Bone wax retards bone healing and predisposes the surgical site to

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infection[117,378] by producing a chronic inflammatory foreign body reaction[35] and impairing the clearance of bacteria.[263] The use of bone wax can no longer be recommended because it impairs healing and several good alternatives are available.[589]

Ferric sulfate

Ferric sulfate (Cut-Trol, Ichthys Enterprises, Mobile, AL), a necrotizing agent with an extremely low pH, is one of the few products investigated for use in periradicular surgery. In studies using a rabbit model, Lemon and colleagues[301] and Jeansonne and colleagues[256] reported hemostatic control for 5 minutes and near normal healing with only a mild foreign body reaction, provided the surgical wound was adequately curetted and irrigated with saline. Failure to remove ferric sulfate from the surgical wound site resulted in severely impaired healing, a foreign body-type reaction, and, in some cases, abscess formation. The possibility of acute inflammation and necrosis of the surrounding soft tissue with careless use of this solution should not be underestimated.[298] A similar product, Monsel solution (ferric subsulfate), has been used to control local hemostasis in dermatologic procedures. However, the popularity of this solution has declined because application to wound sites has resulted in tissue necrosis for up to 2 weeks,[130] differences in the degree of epidermal maturation, and tattoo formation.[536]

Calcium sulfate

Calcium sulfate has been used as a substitute bone graft material to fill bone defects since the late 1800s. The presence of calcium sulfate in an osseous wound does not inhibit bone formation.[574] It is gradually removed from the site of implantation regardless of whether new bone has formed.[110] Use of calcium sulfate during periradicular surgery does not significantly affect healing, and deposition of cementum and osseous healing proceed normally.[27] As a hemostatic agent, calcium sulfate acts as a physical barrier. The material is placed in the bony crypt, allowed to set, and then partly carved away to allow access to the root end.[283] The remaining material lines the crypt walls, preventing bleeding. When the root-end filling has been placed and all extraneous root-end filling material removed, the residual calcium sulfate can be removed or left in situ.

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Epinephrine pellets

Epinephrine, a sympathomimetic-amine vasoconstrictor, is frequently used to control hemorrhage during oral surgery.[82,283] All granulation tissue should be removed from the root apex area prior to placement of the epinephrine pellet to assure direct contact with bone.[282] Vasoconstrictive amines exert their effects by binding to and interacting with adrenergic receptors in various body tissues. When epinephrine is bound to $alpha_1$- and $alpha_2$-adrenergic receptors, a powerful vasoconstricting effect results. Racemic epinephrine cotton pellets (Racellet #3; Pascal Co., Bellevue, WA) contain an average of 0.55 mg of racemic epinephrine hydrochloride per pellet, half of which is the pharmacologically active L-form. Racellets (see Fig. 11.31, *B*) provide good localized hemostasis in periradicular surgery.[558]

Two concerns arise with the use of Racellets in the surgical site: the cardiovascular impact of the additional epinephrine and the retention of cotton fibers in the wound, resulting in impaired wound healing.[213,265] A study by Vickers and coworkers[558] examined the cardiovascular effects of epinephrine pellets and concluded that no evidence existed of cardiovascular changes (blood pressure and pulse) compared with saline-saturated pellet controls. These authors hypothesized that the vasoconstrictive effect on the capillaries is localized and immediate and that little or no systemic uptake of epinephrine occurred.

Although the cardiovascular effect appears to be of little concern, retention of cotton fibers in the surgical site could result in inflammation and impaired wound healing. Therefore due diligence on the part of the surgeon is paramount when epinephrine pellets are used. Each pellet applied during surgery must be accounted for, and the bony crypt should be lightly curetted to remove any embedded cotton fibers before the wound is closed. The retention of cotton fibers in the crypt can be eliminated by substituting CollaCote saturated with 10 drops of 2.25% racepinephrine inhalation solution.[567] As previously noted, CollaCote is biocompatible and does not interfere with wound healing.

Cautery/electrosurgery

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Cautery stops the flow of blood through coagulation of blood and tissue protein, leaving an eschar that the body attempts to slough.[537] The effect of cautery in the bony crypt during periradicular surgery has not been studied to date. However, the effect of electrosurgery on alveolar bone has been studied in periodontal surgery. Tissue destruction was greater in areas exposed to electrosurgery and healing was delayed compared with surgical sites not exposed to electrosurgery. Twelve hours after surgery, a more extensive inflammatory reaction and greater destruction of periosteum were noted with electrosurgery.[36] At 24 hours, many empty lacunae were observed in the bone associated with electrosurgery, and this necrosis was even more extensive by 48 hours. At 96 hours the electrosurgical connective tissue wounds were still lined by coagulum, whereas the scalpel wound was beginning to repair.[381] The detrimental effect of applying heat to bone is proportional to both temperature and the duration of application.

Management of the root end

Management of the resected root end during periradicular surgery is critical to the overall success of a case. The aim of surgery should be to create an environment conducive to regeneration of the periodontium—that is, healing and regeneration of the alveolar bone, PDL, and cementum overlying the root end and root-end filling material. The key to regeneration is the presence of appropriate inducible cell types, growth factors, and specific substances necessary for mineralization. Failure to create an environment conducive to this process results in tissue repair rather than regeneration and possibly less-than-ideal healing.

Determination of the need for root-end resection and filling

The basis for periradicular surgery is twofold. The first objective is to remove the etiologic factor; the second is to prevent recontamination of the periradicular tissues once the etiologic agent has been removed.

Etiologic factors typically can be categorized as intraradicular or extraradicular bacteria,[254,287,367,491,540,541,559,580] intraradicular or extraradicular chemical substances, or extraradicular physical

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factors.[370,496,497] The etiology cannot always be determined with complete certainty; frequently, a number of factors are involved.[98] However, most cases involve some form of bacterial participation (e.g., bacteria within apical ramifications). The only definitive means of eradicating such an irritant is physical removal through root-end resection. The rationale for root-end resection in such cases is to establish access to and remove the diseased tissues. This ensures that the optimum environment for wound healing is established.

As mentioned, the second objective of periradicular surgery is to prevent recontamination of the tissues after removal of the etiologic agent. It follows, then, that if the remainder of the canal system cannot be verified to be irritant free, then a root-end filling should be placed to seal any remaining irritants within the canal system, thus preventing recontamination of the periradicular tissues.

Root-end resection

Two main principles dictate the extent of the root-end resection. First, the cause (or causes) of an ongoing disease process must be removed; this includes removal of the diseased tissue and, when indicated, reduction of an apically fenestrated root. Second, adequate room must be provided for inspection and management of the root end.

The anatomy of each tooth root is complex. The surgeon must understand the anatomy of the apical third of the root to determine the extent of a root-end resection. Approximately 75% of teeth have canal aberrations (e.g., accessory or lateral canals) in the apical 3 mm of the tooth.[131,481] An apical resection of approximately 3 mm should include most accessory and lateral canals and thus eliminate most residual microorganisms and irritants (Fig. 11.35). When roots with more than one main canal are resected, isthmus tissue may be present, and the preparation should be modified to include the isthmus area (Fig. 11.36).

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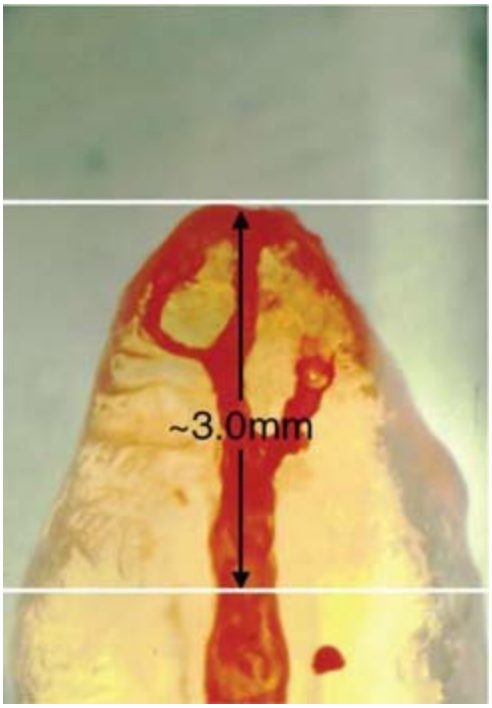

FIG. 11.35 Cleared section of a typical single-canal root that was injected with dye to demonstrate apical accessory canals. Most of the apical ramifications can be eliminated with a 3-mm resection.

A cleared section of tooth shows red dye in the canal branching out at the apical region with resection marked with a double headed arrow labeled as approximately 3 millimeters.

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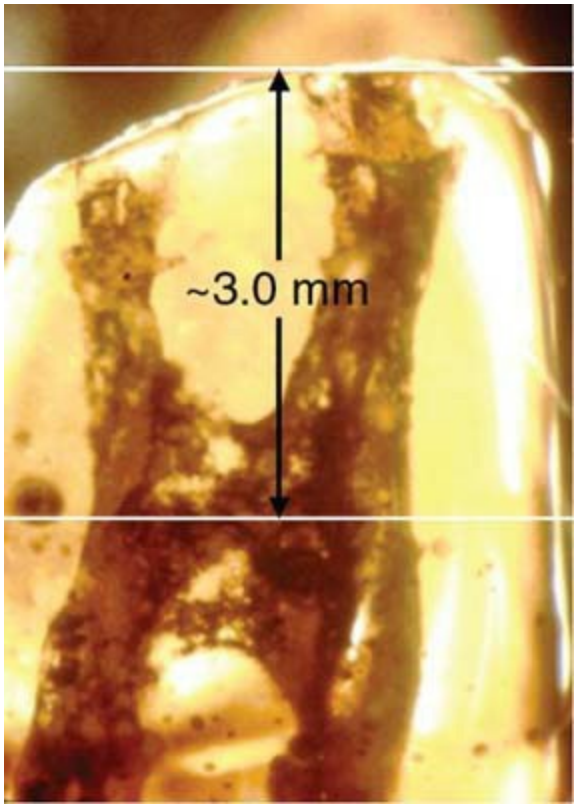

FIG. 11.36 Cleared section of a mesiobuccal (MB) root in a maxillary first molar. Root resection at the recommended 3-mm level exposes isthmus tissue connecting the MB-1 and MB-2 canals.

A cleared section of tooth shows a double headed arrow labeled as approximately 3 millimeters covering isthmus tissue linked to mesiobuccal cannal 1 and 2.

If the root apex is close to the buccal cortical plate, apical fenestration can occur, leading to persistent symptoms, especially tenderness to palpation over the root apex.[72] Reduction of an apically fenestrated root apex below the level of the surrounding cortical bone allows remodeling of the bone over the tooth structure. The buccal root of the maxillary first premolar often is closest to the buccal cortical plate.

The surgeon's convenience as a rationale for root-end resection depends on the individual case and the surgeon's abilities. The basic principle of the dental surgeon's convenience should be modified by the desire to minimize the trauma of the surgical procedure itself, including the preservation of tooth and supporting structures. Access to and visibility of the periradicular root structures historically has determined the extent of root-end resection. The

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surgeon must be able to inspect the resected root end, prepare a root-end cavity, and place a root-end filling. Enhanced visualization equipment (e.g., microscopes, endoscopes, and orascopes) have reduced the need to resect large amounts of the root to gain adequate visualization and access.[91,94,228,280,440,441,448,486] In some cases part of the root must be resected to gain access to the entire soft-tissue lesion, an additional palatally positioned root (e.g., maxillary premolars), or foreign material in the periradicular tissues.

A major consideration in determining the extent of root-end resection is the presence of anatomic structures such as the mental foramen or mandibular canal.[355,410-412] The surgeon should position the resection of the root to avoid possible damage to these structures.

Angle of resection

Enhanced magnification and illumination techniques have eliminated the need to create a beveled root surface in most cases.[94,441] From a biologic perspective, the most appropriate angle of root-end resection is perpendicular to the long axis of the tooth (Figs. 11.37 and 11.38). The rationale for a perpendicular root-end resection is based on several anatomic parameters. First, a perpendicular resection approximately 3 mm from the anatomic apex is more likely to include all the apical ramifications in that region of the tooth.[339] Second, as the angle of resection increases, the number of dentinal tubules that communicate with the periradicular region and the root canal system increases significantly; therefore the probability that irritants from within the canal system will gain access to the healing tissues also rises as the resection angle increases.[181,523] Third, extending the root-end cavity preparation beyond the coronal extent of the root surface is simpler if the root-end resection is perpendicular to the long axis of the tooth.[195] Finally, with a perpendicular root-end resection, the stress forces exerted in the apical region are more evenly distributed; this may reduce the propagation of apical fractures and provide a better environment for apical healing.[470]

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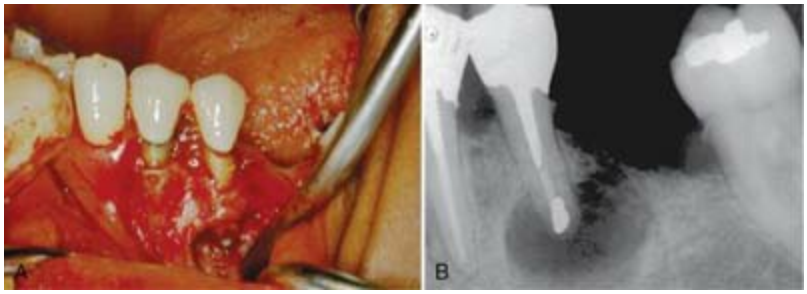

FIG. 11.37 Periradicular surgery circa 1990. **A,** The root end was prepared with a 45-degree bevel and rotary bur microhandpiece. Amalgam was a commonly used root-end filling material at this time. **B,** Immediate postoperative radiograph of a mandibular second premolar with amalgam root-end filling. Although many teeth treated this way healed successfully, newer materials and techniques described in this chapter are currently recommended.

A) Close-up of interior of mouth shows a 45-degree bevel and micro hand piece inserted at the root end of the tooth in the gingiva.

B) Radiograph shows a gray patch at the root of treated tooth with amalgam filling at root end depicted as a radiopaque spot.

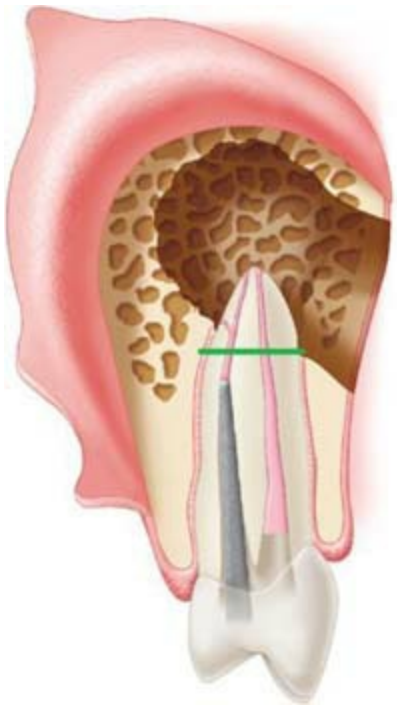

FIG. 11.38 Perpendicular or near perpendicular root-end resection *(green line)* can be achieved with the use of microsurgical instruments and enhanced magnification and illumination.

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An illustration of a tooth shows a triangular flap exposing the root of tooth with a horizontal green line drawn in the apical region.

Root-end surface preparation

As with all phases of endodontic surgery, the goal is to produce a resected root end with optimum conditions for the growth of cementum and subsequent regeneration of the PDL across the resected root end. Two important aspects of this process are the surface topography of the resected root end and chemical treatment of the resected root end. Healthy cementum on the root end is required for successful regeneration of periodontal tissues.[22] A number of substances found in cementum stimulate the migration, growth, and attachment of periodontal fibroblasts. Cementum extracts also activate fibroblast, protein, and collagen synthesis, which is necessary to reestablish a functional PDL.[52,471,562]

Resected root-end surface topography

The conventional axiom for surface preparation of the resected root end has been to produce a smooth, flat root surface without sharp edges or spurs of root structure that might serve as irritants during the healing process. However, little information exists on whether smooth root ends heal differently or more quickly than rough root ends after root-end resection. A study investigating the effect of the surface topography of resected root ends on human PDL fibroblast attachment found no significant difference in fibroblast attachment to the root ends prepared with various instruments.[584] However, with a smooth resected root-end surface, the surgeon is better able to detect surface cracks and anatomic variations.[357] Given that human PDL fibroblast attachment to a smooth surface was not impaired, it would seem appropriate to produce as smooth a surface as possible to facilitate inspection of the resected root end. A smooth root end therefore should be considered advantageous.

Different types of burs tend to produce different patterns on the resected root surface.[212] Several studies have compared the root-end surface after resection.[357,377,585] Generally, crosscut fissure burs in both high-speed and low-speed handpieces produced the roughest and most irregular surfaces.

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Morgan and Marshall[357] compared the surface topography of root-end resection with a #57 straight fissure bur (Midwest Dental Products, Des Plains, IL), a Lindeman bone bur (Brasseler USA, Savannah, GA), and the Multi-Purpose bur (Dentsply Maillefer, Ballaigues, Switzerland), then finished with a either a multifluted carbide finishing bur (Brasseler USA) or an ultrafine diamond finishing bur (Brasseler USA). The Multi-Purpose bur produced the smoothest, most uniplanar resected root-end surface with the least amount of shattering. Regardless of the type of bur used, smearing and shredding of the gutta-percha across the root face occurred only when the handpiece was moved across the root face in reverse direction in relation to the bur's direction of rotation.[585] Burs that produce a smooth surface also tend to cut with less vibration and chatter, resulting in greater patient comfort.

Root-end conditioning

Root surface conditioning removes the smear layer and provides a surface conducive to mechanical adhesion and cellular mechanisms for growth and attachment. It exposes the collagenous matrix of dentin and retains biologically active substances, such as growth factors, in the dentin proper. Experimental studies have shown that demineralized dentin can induce the development of bonelike mineralized tissue.[42,43,241,550,597] Some contend that root surface conditioning produces a biocompatible surface conducive to periodontal cell colonization without compromising the vitality of the adjacent periodontium.

Three solutions have been advocated for root surface modification: citric acid, tetracycline, and ethylenediamine tetra-acetic acid (EDTA). All three solutions have enhanced fibroblast attachment to the root surface in vitro. However, citric acid is the only solution tested in an endodontic surgical application.

Citric acid traditionally has been the solution of choice. Periodontists have used an aqueous solution of citric acid (pH 1) for 2 to 3 minutes to etch diseased root surfaces to facilitate formation, new attachment, and cementogenesis.[434-438] Craig and Harrison[114] examined the effect of citric acid demineralization of resected root ends on periradicular healing. They found that 1- or 2-minute applications of 50% citric acid (pH 1) resulted in demineralized root ends and earlier complete healing than in the

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nondemineralized root ends. However, the periodontal literature has questioned the benefit of etching dentin surfaces with low pH agents. At a low pH, adjacent vital periodontal tissues may be compromised. Also, extended application (3 minutes) has been shown to discourage alveolar bone growth.[60,62]

EDTA, a solution with a neutral pH that endodontists have used as a canal irrigant, has been shown to be equally effective at exposing collagen fibers on dentin surfaces.[64] Unlike the lower pH solution, EDTA does not adversely affect the surrounding tissues.[63]

A series of studies that examined the effect of EDTA and citric and phosphoric acids in a periodontal application showed that application of 15% to 24% EDTA for approximately 2 minutes produces the optimum root surface.[59,61,64] These researchers concluded that EDTA, at neutral pH, was able to selectively remove mineral from a dentin surface, exposing a collagenous matrix. Citric and phosphoric acids, which have a low pH, appeared not only to remove the mineral component but also to denature the collagenous matrix.

Tetracycline has been shown to remove the dentin smear layer, leaving clean, open tubules, with application times as short as 30 seconds.[327] A histologic evaluation of new attachment in periodontally diseased human roots treated with tetracycline hydrochloride showed a trend for greater connective tissue attachment after tetracycline treatment of roots.[13] Studies comparing the effect of a 3-minute application of either EDTA (pH 7.3) or tetracycline HCl (pH 1.8) showed no significant difference in the treated tooth surfaces.[37] However, EDTA has been shown to be more favorable to human PDL cell attachment.[601]

Although the root surface conditioning effects of citric acid, EDTA, and tetracycline are well documented in the periodontal literature, this treatment has not translated into significant gains in periodontal attachment in periodontally diseased teeth.[330] Currently, only citric acid has been assessed as a root-end conditioning agent. In an animal model, citric acid was shown to enhance periradicular healing. Nonetheless, its effect and the effect of other root-end conditioning agents on the outcome of human periradicular surgery have not been established. Based on periodontal research, it would appear that if a root surface conditioning agent were to be used during

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periradicular surgery, EDTA might be the most appropriate solution. However, the manufacturer (personnel communication, Dr. Torabinejad) has advised against the use of EDTA when MTA is used as a root-end filling material, because it may interfere with the hard tissue–producing effect of MTA.

Root-end cavity preparation

The root-end cavity preparation is a crucial step in the establishment of an apical seal. The goal is to make a cavity in the resected root end that is dimensionally sufficient for placement of a root-end filling material and at the same time avoid unnecessary damage to the root-end structures. The ideal preparation is a class I cavity prepared along the long axis of the tooth to a depth of at least 3 mm (Figs. 11.39 and 11.40). The surgical procedure is most likely to be successful if the remaining canal system has been thoroughly cleaned and shaped to eliminate microorganisms and irritants.[178,449] Traditionally, a microhandpiece with a rotating bur has been used for this purpose. However, with the advent of ultrasonic tips designed specifically for this purpose (Fig. 11.41), root-end preparations now are most often performed with the ultrasonic technique.[91] Clinical evidence is emerging to support the benefit of an ultrasonic root-end preparation when compared to traditional bur preparation, especially in molar surgery.[132]

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FIG. 11.39 Diagram of a perpendicular root-end preparation and 3-mm deep cavity preparation along the long axis of the root.

An illustration of a tooth shows a triangular flap held with a retractor exposing the root of tooth with root removed and replaced with a hollow cavity.

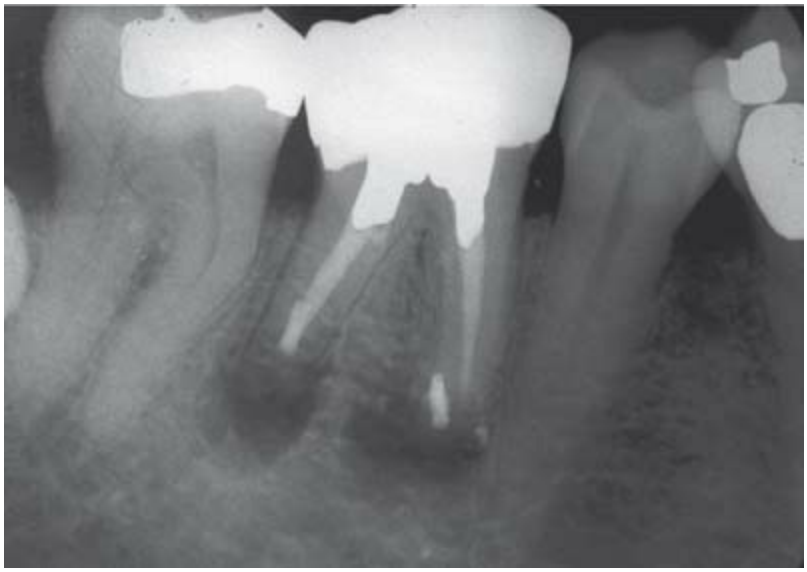

FIG. 11.40 Error in root-end cavity preparation: ultrasonic preparation did not follow the long axis of the mesial root and therefore did not allow for proper sealing of this root. Healing is unlikely.

Radiograph shows a teeth with black spots at the apical foramen.

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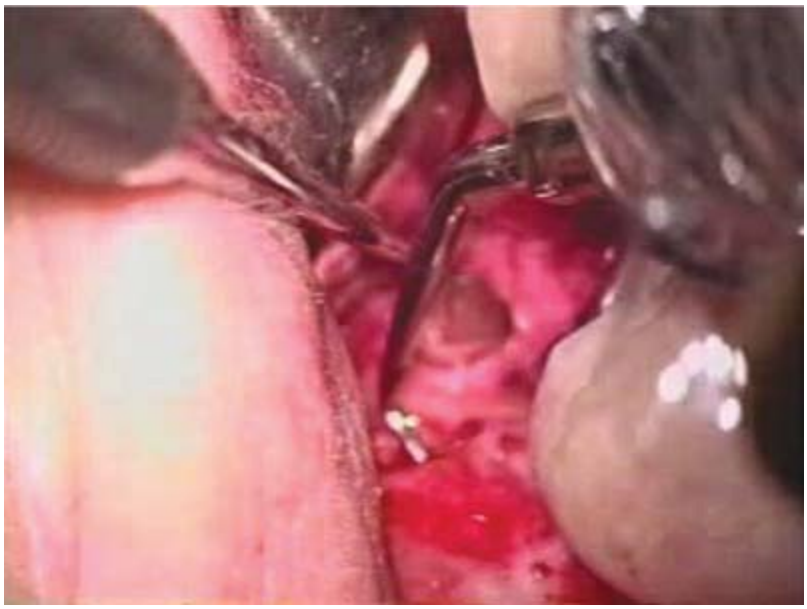

FIG. 11.41 Ultrasonic tip in use. Preparation along the long axis of the root is possible using tips designed for each area of the mouth. In this case, the tip is properly positioned for root-end cavity preparation of a maxillary first premolar but is dangerously close to the lip because of improper retraction. Heat generated by an ultrasonic tip can cause a thermal burn, which may result in scar tissue formation.

Close-up view shows tip placed very close to lip near the maxillary first premolar.

Ultrasonic root-end preparation techniques have several advantages over the microhandpiece method. Less osseous tissue must be removed to gain proper access to the resected root end. Also, the surgeon is better able to produce a more conservative preparation that follows the long axis of the tooth and remains centered in the canal. The risk of root-end perforation is reduced, partly because of enhanced manipulation of the instrument. In addition, ultrasonic root-end techniques produce a more consistent, deeper cavity preparation that requires less beveling of the root.[95,304,347,592] Ultrasonic apical preparation generates significantly less smear layer compared with burs alone[205]; root-end preparation with a bur produces a heavy smear layer at all levels of preparation.[214]

The major concern with ultrasonic root-end preparation is the potential for

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creating root fractures as a result of the ultrasonic vibration (Fig. 11.42).

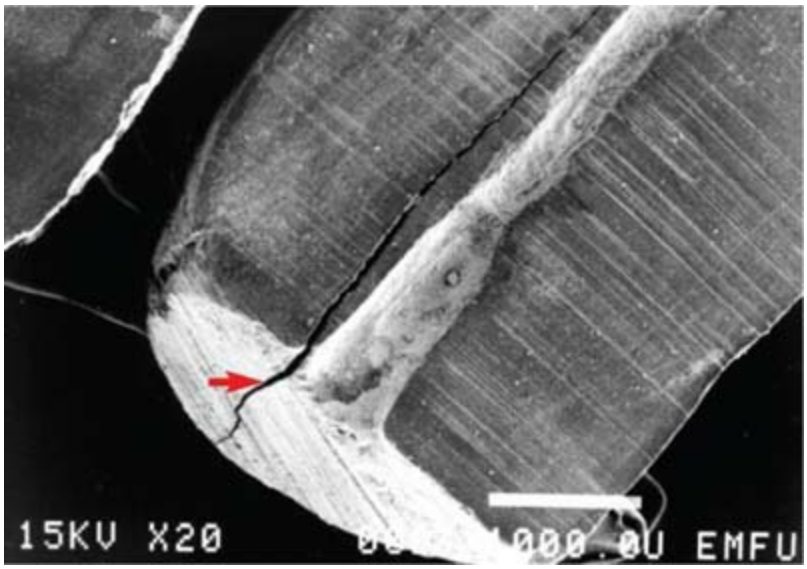

FIG. 11.42 Scanning electron microscope image of a root end prepared in vitro with an ultrasonic device at high-power setting. A distinct fracture line can be seen *(red arrow).* Root-end preparation with ultrasonic devices should be done at low power and with water coolant.

Microscopic image of apical region of a tooth shows a red arrow pointing at a vertical black line depicting root fracture.

Ultrasonic root-end preparation and apical fractures

Several studies have investigated the fracture-inducing potential of ultrasonic root-end preparation techniques. This is possibly the most controversial question that arises in relation to the use of ultrasonic root-end preparation tips. Three types of root-end fractures have been described: intracanal fractures (originating from the root canal system and extending into the dentin), extracanal fractures (originating on the root surface and extending into the dentin), and communicating fractures (extending from the root surface to the root canal system).[425] This classification system is not universally used in studies of root-end cavity preparation; therefore it is difficult to establish the significance, if any, of one type of fracture over another. When a strain gauge was used to measure root deformation, ultrasonic root-end preparation was shown to produce significantly greater

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strain, on average, than that generated by a microhandpiece. However, this did not translate into an increase in cracks observed on the resected surface of roots after root-end cavity preparation.[48,303,375,425] Several other in vitro studies that used different models to assess root-end fractures conversely concluded that ultrasonic root-end preparation does induce apical fractures.[2,184,299,354,469] Thus the degree to which apical fractures are induced during ultrasonic root-end cavity preparation is difficult to determine from in vitro studies.

On the other hand, in an in vivo study[358] and cadaver-based studies[86,207] designed to replicate the clinical scenario, root fractures were not attributed to ultrasonic use. In these studies, ultrasonic root-end preparation did not induce a significant number of root-end fractures. Several explanations may account for the differences observed in these studies. The surrounding tissues in cadaver and clinical subjects may disperse the ultrasonic energy away from the root tip. Thermal energy produced during ultrasonic preparation may have been controlled more adequately in some studies than in others. The power setting used on the ultrasonic unit may have been in the low range; a low power setting has been shown to produce fewer fractures in an in vitro setting[176,299] and is therefore recommended for clinical use.

Significance of ultrasonic tip design

Different types of ultrasonic tips are available for root-end preparation (Figs. 11.43 and 11.44), including tips of varying lengths and diameters constructed of stainless steel. These tips are left uncoated or are coated with diamond or zirconium nitride. Tips with a curvature of 70 degrees or greater are more susceptible to fracture under continuous loading, and fracture typically occurs at the bend.[573] Coating of ultrasonic tips undoubtedly improves the cutting efficiency compared with uncoated stainless steel tips; this translates into significantly less time required to prepare a root-end cavity.[200,405] In the coated tips, the diamond coating appears to be the most aggressive and requires the least amount of time to produce a root-end cavity preparation.[253] Furthermore, the type of tip (i.e., stainless steel, diamond coated, or zirconium nitride coated) appears to have little effect on the number or types of fractures that can be induced in the root end during root-end preparation.[76,200,253,375,425] The cavity walls of root-end preparations formed

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by stainless steel tips typically have cleaner canal walls than those formed by coated root-end preparation tips. Stainless steel tips appear to produce less superficial debris and smear layer. Coated root-end preparation instruments typically produce a heavily abraded, debris-covered cavity wall surface.[76,610] However, overall the quality of the coated-tip preparation has been suggested to be superior.[405]

FIG. 11.43 Ultrasonic tips (Obtura Spartan) are available in a wide variety of configurations for use in different areas of the mouth. Most new tips have a special coating (zirconium nitride or diamond) that improves cutting efficiency.

Close-up of an ultrasonic tip with a 90-degree bent tip kept on a platform.

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FIG. 11.44 Ultrasonic tip with diamond coating and irrigation port (DENTSPLY Tulsa Dental Specialties).

Close-up of ultrasonic top with a drop-shaped hollow space in handle as irrigation port.

Temperature changes induced by ultrasonic instruments

The importance of heat generation and temperature changes has already been discussed. All ultrasonic surgical tips should have an irrigation port. Use of an ultrasonic instrument in the periradicular tissues without adequate irrigation results in an extreme temperature increase in the tissues, although this specific effect has not been demonstrated during root-end preparation. Scaling without irrigation can increase the temperature in dentin as much as 95°F (35°C) above the baseline temperature[290]; such an increase can injure pulpal and periodontal tissues.[380]

Bonded root-end fillings

Root-end cavity preparation for bonded root-end filling materials requires a change in the standard root-end cavity preparation technique. A shallow, scalloped preparation of the entire root surface should be made using a round or oval bur; the preparation should be at least 1 mm at the deepest concavity.[26,453] An ultrasonic preparation can be made into the root canal system, but this may not be necessary. The root-end filling material is placed

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in a dome fashion and bonded to the entirety of the resected root end (Fig. 11.45).

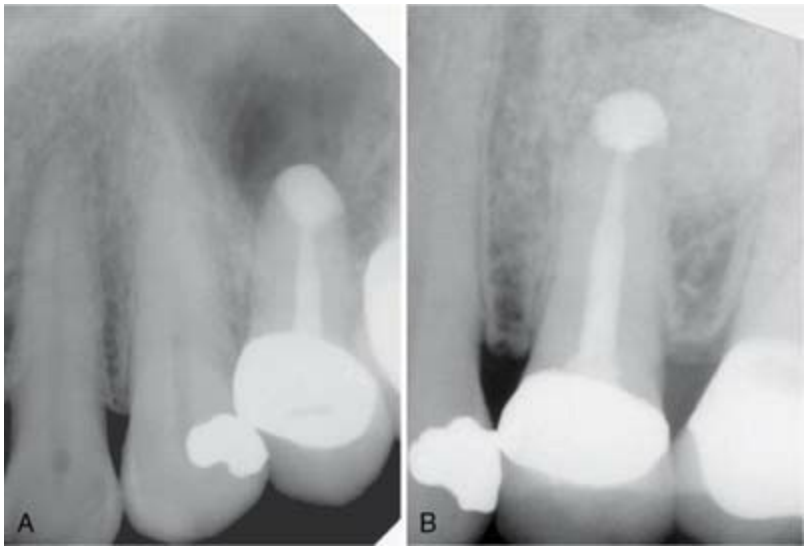

FIG. 11.45 **A,** Immediate postoperative radiograph of a bonded root-end filling in a maxillary first premolar. **B,** Follow-up radiograph at 20 months showing good periradicular healing.

Set of two radiographs are marked A and B as follows:

A) Radiograph shows radiopaque bud attached to the crown of the tooth with a gray spot present at the root of tooth.

B) Radiograph shows disappearance of gray spot at the apical root of the tooth with root end filling in place depicted as a radiopaque region.

Root-end filling materials

The ideal root-end filling material seals the contents of the root canal system within the canal, preventing egress of any bacteria, bacterial by-products, or toxic material into the surrounding periradicular tissues. The material should be nonresorbable, biocompatible, and dimensionally stable over time. It should be able to induce regeneration of the PDL complex, specifically cementogenesis over the root-end filling itself. Finally, the handling properties and working time should be such that the endodontic surgeon can place a root-end filling with sufficient ease.

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Many materials have been used as root-end fillings, including gutta-percha, polycarboxylate cements, silver cones, amalgam, Cavit (3M ESPE, St. Paul, MN) zinc phosphate cement, gold foil, and titanium screws. However, this section focuses on root-end filling materials discussed in the literature within the past 10 years that are in common clinical use. These materials are zinc oxide–eugenol (ZOE) cements (IRM and Super-EBA), glass ionomer cement, Diaket, composite resins (Retroplast), resin–glass ionomer hybrids (Geristore), and mineral trioxide aggregate (ProRoot-MTA), and bioceramics.

Zinc oxide–eugenol cements

Zinc oxide powder and eugenol liquid can be mixed to form a paste that is compacted into a cavity preparation. Use of this material dates back to the 1870s. Eugenol is released from ZOE mixtures, although this declines exponentially with time and is directly proportional to the liquid-powder ratio.[243] When ZOE comes in contact with water, it undergoes surface hydrolysis, producing zinc hydroxide and eugenol. This reaction continues until all the ZOE in contact with the free water is converted to zinc hydroxide.[243-245] Eugenol can have a number of effects on mammalian cells, depending on the concentration and length of exposure. These effects include cell respiration depression, macrophage and fibroblast cytotoxicity, depressed vasoconstrictor response, inhibition of PG, and suppressing or enhancing effects on the immune response.[139,349,561] Other materials have been added to the basic ZOE mixture in an effort to increase the strength and radiopacity and reduce the solubility of the final material. Commercially available ZOE materials include intermediate restorative material (IRM; Dentsply Caulk, Milford, DE) and Super-EBA (Bosworth Company, Skokie, IL).

Intermediate restorative material

IRM consists of a powder containing greater than 75% zinc oxide and approximately 20% polymethacrylate mixed in equal parts with a liquid that contains greater than 99% eugenol and less than 1% acetic acid. IRM seals better than amalgam and is not affected by the liquid-powder ratio or root-end conditioning agents.[116,407] IRM appears to be tolerated in the periradicular tissue, but it has no dental hard-tissue regenerative capacity. The response is similar to that seen with other ZOE-based materials.[221,329,417-420] In vitro,

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IRM prevents adherence of enamel matrix proteins.[463]

Super-EBA

Super-EBA consists of a powder containing 60% zinc oxide, 34% aluminum oxide, and 6% natural resins. It is mixed in equal parts with a liquid that contains 37.5% eugenol and 62.5% o-methoxybenzoic acid. Super-EBA is available in two forms, fast set and regular set. Other than the setting time, the properties of the two forms appear to be the same.[593] Super-EBA has radiopacity[484] and sealing effects similar to those of IRM and is less leaky than amalgam.[232,388] The leakage pattern of Super-EBA does not appear to be affected by root-end conditioning or finishing techniques.[174,469] When Super-EBA and IRM were finished with a carbide finishing bur in a high-speed handpiece, marginal adaptation was better than with ball burnishing, which was equal to burnishing with a moistened cotton pellet.[170] The environment of the periradicular wound may affect the long-term stability of Super-EBA, which has been shown to disintegrate over time in an acid pH environment.[30]

Biologically, Super-EBA is well tolerated in the periradicular tissues when used as a root-end filling. However, it has no capacity to regenerate cementum. Bone healing has been demonstrated at 12 weeks, with some fibrous tissue persisting. Super-EBA root-end fillings show a basophilic-stained line adjacent to the filling material, which may indicate hard-tissue formation.[404,418,419,542] Collagen fibers appear to grow into the cracks of the material,[396] but the significance of this is unknown. Super-EBA has limited antibacterial effect.[101] The cytotoxicity of Super-EBA is similar to that of amalgam and IRM.[102,605] The incidence of persistent disease after endodontic surgery in which Super-EBA was used as a root-end filling material ranges from 4% to 20%. In comparative studies in which amalgam was also used as a root-end filling material, the use of Super-EBA always resulted in less persistent disease. The follow-up period for these studies ranged from 0.5 to 4.6 years.[147,326,397,448,518,565]

Glass-ionomer cements

Glass-ionomer cement (GIC) consists of aqueous polymeric acids, such as polyacrylic acid, plus basic glass powders, such as calcium aluminosilicate.

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GIC sets by a neutralization reaction of aluminosilicate, which is chelated with carboxylate groups to cross-link the polyacids; a substantial amount of the glass remains unreacted and acts as reinforcing filler. GICs can be either light or chemically cured. Silver has been incorporated into GIC to improve the physical properties, including compressive and tensile strength and creep resistance. Both forms of GIC have been suggested as an alternative root-end filling material.[44,415,416]

The seal and marginal adaptation of light-cured GIC are superior to those with chemical-cured GIC. The seal achieved with GIC generally is better than that with amalgam and similar to that with IRM.[105,106,252,444,520,591] Long-term surface changes can occur in silver-GIC that may affect the stability of silver-GIC in the periradicular tissues.[55] GICs are susceptible to attack by moisture during the initial setting period, resulting in increased solubility and decreased bond strength.[188,508,596] Contamination with moisture and blood adversely affected the outcome when GIC was used as a root-end filling material; this occurred significantly more often in unsuccessful cases.[602] The cytotoxicity of chemical- and light-cured GIC does not differ significantly from that of Super-EBA or amalgam.[102,394] The tissue response to GIC is considerably more favorable than to amalgam and similar to that with ZOE-based materials.[100,104,133,416] In a comparative clinical study using amalgam or GIC for root-end filling, healing was evaluated clinically and radiographically after 1 and 5 years.[261,602] No difference was seen in healing capacity between the two materials. The overall success rate in both groups was 90% at 1 year and 85% at 5 years. This study showed that the 5-year follow-up result can be predicted in more than 95% of the cases at the 1-year follow-up. These authors concluded that GIC is a valid alternative to amalgam for use as an apical sealant after root-end resection and that it provides equally good long-term clinical results.[261,602]

Diaket

Diaket (ESPE GmbH, Seefeld, Germany) a polyvinyl resin initially intended for use as a root canal sealer, has been advocated for use as a root-end filling material.[587] It is a powder consisting of approximately 98% zinc oxide and 2% bismuth phosphate mixed with a liquid consisting of 2.2-dihydroxy-5.5

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dichlorodiphenylmethane, propionylacetophenone, triethanolamine, caproic acid copolymers of vinyl acetate, and vinyl chloride vinyl isobutylether. Leakage studies comparing Diaket to other commonly used root-end filling materials have shown it to have a superior sealing ability.[190,264,313,571] Its sealing ability has not been directly compared to that of MTA. When Diaket was used as a root canal sealer, biocompatibility studies showed that it was cytotoxic in cell culture[276] and generated long-term chronic inflammation in osseous[502] and subcutaneous tissues.[393] However, when mixed at the thicker consistency advocated for use as a root-end filling material, Diaket has shown good biocompatibility with osseous tissues.[379,587] Histologically, a unique tissue barrier has been observed to form across the Diaket at the resected root end, the nature of which is unknown. This tissue resembles an osteoid or cementoid type of matrix with a close approximation of periodontal tissue fibers, suggesting a regenerative response to the root-end filling material.[587] In animal studies Diaket has shown a better healing response than resected gutta-percha in uninfected teeth[590] and a healing response similar to that with MTA. However, no cementum formation was evident.[433] This material is no longer available in the United States.

Composite resins and resin-ionomer hybrids

Composite resin materials have some desirable properties and may be considered for use as root-end filling materials. Generally, when assessed for sealability, composite resins perform well in vitro studies. Composite resins also tend to leak less than amalgam, Super-EBA, IRM, and GICs.[126,344,345,520] However, blood contamination during the bonding process reduces bond strength and increases leakage.[353,560] Marginal adaptation varies, depending on the conditions and the bonding agents.[17] Certain components of composite resins and dentin-bonding agents can have a cytotoxic effect on cells; this effect varies, depending on the agent and its concentration.[80,217,428,429,514] Studies have shown that once the composite resin sets, cells can grow on its surface.[328,362,402,604] The healing response of the periradicular tissues to composite resins in general appears to be very diverse, ranging from poor to good[25,542]; this may depend on the type of material used. Two composite resin–based materials, Retroplast (Retroplast

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Trading, Rørvig, Denmark) and Geristore (Den-Mat, Santa Maria, CA), have been advocated for use as root-end filling materials.

Retroplast

Retroplast is a dentin-bonding composite resin system developed in 1984 specifically for use as a root-end filling material. The formulation was changed in 1990, when the silver was replaced with ytterbium trifluoride and ferric oxide. Retroplast is a two-paste system that forms a dual-cure composite resin when mixed. Paste A is composed of Bis-GMA/TEGDMA 1:1, benzoyl peroxide N,N-di-(2-hyydroxyethyl)-p-toluidine, and butylated hydroxytoluene (BHT). This is mixed in equal parts with paste B, which is composed of resin ytterbium trifluoride aerosil ferric oxide. A Gluma-based dentin bonding agent is used to adhere the material to the root-end surface. The working time is 1½ to 2 minutes, and the radiopacity (due to the ytterbium trifluoride content) is equivalent to 6 mm of aluminum.

Only limited information is available on the physical and chemical properties of Retroplast, although a number of human clinical studies have been published.[22,25,361, 451-455,457-460] In all cases the material appeared to be well tolerated and promoted a good healing response. There is evidence that Retroplast promotes hard-tissue formation at the root apex, and some have suggested that this is a form of cementum. In a limited number of case reports, Retroplast root-end fillings have demonstrated regeneration of the periodontium with a cementum layer over the root-end restoration.[22,453,454] The healing response in these cases showed deposition of minimal cementum and the insertion of new Sharpey's fibers. The PDL fibers also entered the newly formed adjacent alveolar bone, indicating that tissue regeneration, including cementogenesis, may occur on composite material, consequently forming a biologic closure of the root canal.[25] In an investigation of 388 cases comparing root-end fillings of Retroplast or amalgam, radiographic healing after 1 year was as follows: with Retroplast, 74% showed complete healing, 4% showed fibrous healing, 15% were uncertain, and 7% were failures; with amalgam, 59% showed complete healing, 3% showed fibrous healing, 30% were uncertain, and 8% were failures.[453] Complete healing occurred significantly more often after root-end filling with Retroplast. The number of immediate postoperative complications did not differ significantly

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between the composite and the amalgam groups. A more recent clinical study of 351 cases reported a complete healing rate of 80% to 89%.[459] A 10-year follow-up of 34 of these cases showed complete healing in 33 of the cases.[458]

Resin-ionomer suspension (Geristore) and compomer (Dyract)

The resin-ionomer suspension and compomer group of materials attempts to combine the various properties of composite resins and glass ionomers. Geristore and Dyract (Dentsply, Tulsa, OK) have been investigated for use as root-end filling materials, although the available published literature on both is limited. These two materials require light activation and resin-dentin bonding agents to attach to the tooth.

Geristore has been recommended both as a root-end filling material[93] and for use in restoring subgingival surface defects such as root surface caries, external root resorption lesions, iatrogenic root perforations, and subgingival oblique fractured roots. Clinical evaluation of Geristore as a restorative material for root caries and cervical erosions showed it to be an acceptable material.[183,372,475,548] When it is used for surgical repair of root perforations and as an adjunct to GTR, the results have been favorable in isolated case reports.[3,4,47,440,487] Geristore's dual-curing paste/paste formulation is a hydrophilic Bis-GMA with long-term fluoride release. Light activation for 40 seconds cures the material to approximately 4 mm. However, the top layer is harder until the material achieves uniform hardness at 1 day after activation.[512] In vitro leakage assessment of Geristore and Dyract indicates that the materials leak less than root-end fillings made of IRM, amalgam, or Super-EBA.[65,209] Geristore has a leakage pattern similar to that of MTA.[474] An acid pH significantly reduces dye leakage of Geristore.[446] These materials are less sensitive to moisture than conventional GIC; however, dry environments produce stronger bonds.[99] The effect of blood contamination during the bonding phase in a clinical scenario is unknown. Geristore appears to have the potential to allow regeneration of the periradicular tissue. In one study, PDL and gingival fibroblasts attached to Geristore, and the attachment improved with time and cell proliferation.[87] Studies investigating epithelial and connective tissue adherence to Geristore found clinical and histologic evidence of cellular attachment when the material was placed in subgingival cavities.[150,151,475] However, the healing response in the periradicular region

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is best described as unpredictable. In a study in dogs, 10 of the 18 root end–filled teeth developed abscesses. The author attributed this to the technical difficulty of placing Geristore root-end filling. However, a small number of specimens developed cementum on the root-end fillings. The cemental covering was never greater than 25% of the root-end filling surface, which was considerably less than the amount of cementum developed on both white and gray MTA.[315]

Mineral trioxide aggregate

MTA (ProRoot MTA; DENTSPLY Tulsa Dental Specialties), a material developed specifically as a root-end filling,[529] has undergone numerous in vitro and in vivo investigations comparing its various properties to Super-EBA, IRM, and amalgam. In vitro sealing ability and biocompatibility studies comparing root-end filling materials have shown MTA to be superior to other commonly used materials.[277,300,527,530,531,533-535] When various in vitro leakage models were used, MTA prevented leakage as well as composite resin and GIC.[6,129,171,591] However, the setting and subsequent leakage of MTA are not affected by the presence of blood.[527] Torabinejad and colleagues[535] developed the original product (gray MTA). The main constituents of this material are calcium silicate ($CaSiO_4$), bismuth oxide (Bi_2O_3), calcium carbonate ($CaCO_3$), calcium sulfate ($CaSO_4$), and calcium aluminate ($CaAl_2O_4$). Hydration of the powder produces a colloidal gel that solidifies into a hard structure consisting of discrete crystals in an amorphous matrix. The crystals are composed of calcium oxide, and the amorphous region is composed of 33% calcium, 49% phosphate, 2% carbon, 3% chloride, and 6% silica.[529] In a study comparing the setting time, compressive strength, radiopacity, and solubility of MTA to those of amalgam, Super-EBA, and IRM, MTA was found to be less radiopaque than amalgam but more radiopaque than Super-EBA and IRM.[529] MTA had the longest setting time (2 hours, 45 minutes) and the lowest compressive strength at 24 hours after mixing (40 MPa), although compressive strength increased to 67 MPa at 21 days after mixing. The solubility of MTA after setting was similar to that of amalgam and Super-EBA. Initially MTA has a pH of 10.2, which rises to 12.5 3 hours after mixing.[529] The pH has been reported to be approximately

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9.5 at 168 hours after mixing.[152] MTA is less cytotoxic than amalgam, Super-EBA, or IRM root-end fillings.[531] Endodontic surgery studies in dogs and monkeys have reported less periradicular inflammation and cementum deposition immediately adjacent to the root-end filling material.[27,173,236,528,532] Holland and colleagues[236,237] theorized that the tricalcium oxide in MTA reacts with tissue fluids to form calcium hydroxide, resulting in hard-tissue formation.

The importance of the presence of cementum-like tissue adjacent to MTA cannot be understated. Cementum deposition is essential to regeneration of the periodontal apparatus.[310] Augmentation of new cementum across the root end and root-end restoration is essential for ideal healing of the periodontium. A layer would also enhance the integrity of the apical barrier, making it more resistant to penetration by microorganisms; in effect, establishing a biologic barrier.[22] This is seen most frequently in sections where MTA was used as the filling material. MTA appears to be able to induce cementoblastic cells to produce hard tissue. Cementogenesis in the presence of MTA has been evaluated by assessment of the expression of osteocalcin (OCN), cell growth, and the morphology of cementoblast-like cells.[522] Scanning electron microscope (SEM) analysis indicated that cementoblasts could attach to and grow on MTA. In addition, strong expression of the OCN gene was seen after application of MTA. MTA can also increase the production of both proinflammatory and antiinflammatory cytokines from osteoblasts. The clinical significance of this reaction is not known. The effect of MTA on periradicular tissues is probably partly due to these reactions.

In a human outcomes assessment study comparing ProRoot MTA to IRM, the rate of persistent disease with MTA was 16% at 12 months and 8% at 24 months.[103] The rate of persistent disease with IRM was 24% at 12 months and 13% at 24 months. These authors concluded that the use of MTA as a root-end filling material resulted in a high success rate that was not significantly better than that obtained with IRM. A prospective clinical trial using MTA as a root-end filling material along with current microsurgical techniques reported 89% clinical success, with follow-up time ranging from 4 to 72 months.[468]

A variation of the original formula of gray MTA has been introduced. This material, which is a cream white color, is often called white MTA. The

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chemical composition of white MTA is very similar to that of the original. White and gray ProRoot-MTA materials differ by less than 6% in any one component. Both are fine powders with a mean particle size of approximately 10 μm (the range in particle size is approximately 0.1 to 100 μm). The radiopacity of both materials is equivalent to approximately 3.04 mm of aluminum.[45] When white MTA was implanted in the subcutaneous connective tissue of rats, the results were similar to those reported for gray MTA.[238] One study compared the tissue reaction evoked by the two materials when used as root-end fillings in canines.[315] The only statistically significant difference observed was for the presence of macrophages or multinucleated giant cells adjacent to material. Gray MTA had more samples with mild to moderate infiltration of macrophages or multinucleated giant cells, and white MTA had more samples with no macrophages and/or multinucleated giant cells adjacent to the material. All other parameters assessed were essentially the same.

Bioceramics

Bioceramics are a relatively new and potentially promising addition to the group of materials available for root-end filling. In vitro testing of EndoSequence Root Repair Material (ERRM; Brasseler USA) demonstrates biocompatibility and antimicrobial activity that is similar to MTA.[107,122,317,323] ERRM is composed of calcium silicates, monobasic calcium phosphate, and zirconium oxide.[107] The material is hydrophilic, radiopaque, and has high pH. ERRM is available as a putty and a syringable paste. Because this is a relatively new material, long-term clinical studies are not yet available.

Overview of root-end filling materials

Many different materials have been advocated for use as root-end filling materials, and each has specific advantages and disadvantages. However, from the biologic perspective of regeneration of the periradicular tissues, MTA, followed by Retroplast, appears to have a clear advantage over the other available materials. Bioceramic materials may join this group but require more clinical testing. Retroplast and other composite resin–based

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filling materials require meticulous hemostasis and a dry surgical field for optimum results. The most commonly cited disadvantage of MTA is its handling properties. Even when properly prepared, MTA is more difficult to place in the root-end cavity than most other materials. Several devices have been modified or developed specifically for use with MTA (Fig. 11.46; also see Figs. 11.19–11.23). Typical clinical cases showing the surgical procedures described in the previous sections are presented in Figs. 11.47–11.49.

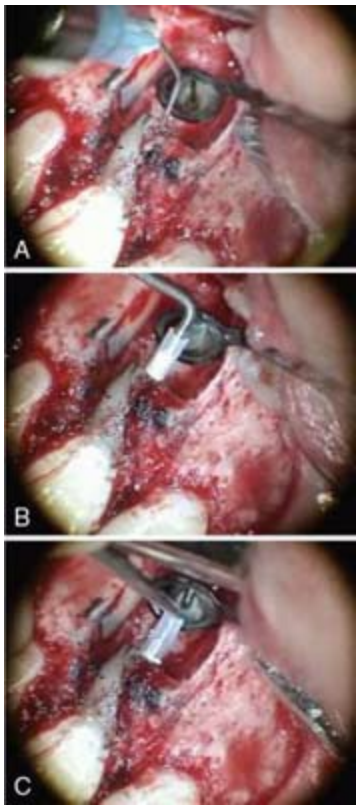

FIG. 11.46 **A,** Stropko syringe used to dry a root-end preparation before placement of the root-end filling material. **B,** Clinical use of an MTA delivery system (Dentsply Tulsa Dental). The device is loaded with MTA and placed over the root-end preparation. **C,** Pressing the plugger into the sleeve delivers the filling material to the root-end cavity preparation. The filling material then is compacted with microcondensers, and additional filling material is placed as needed.

Set of three photographs showing interior of mouth, marked A through C depict use of devices in MTA.

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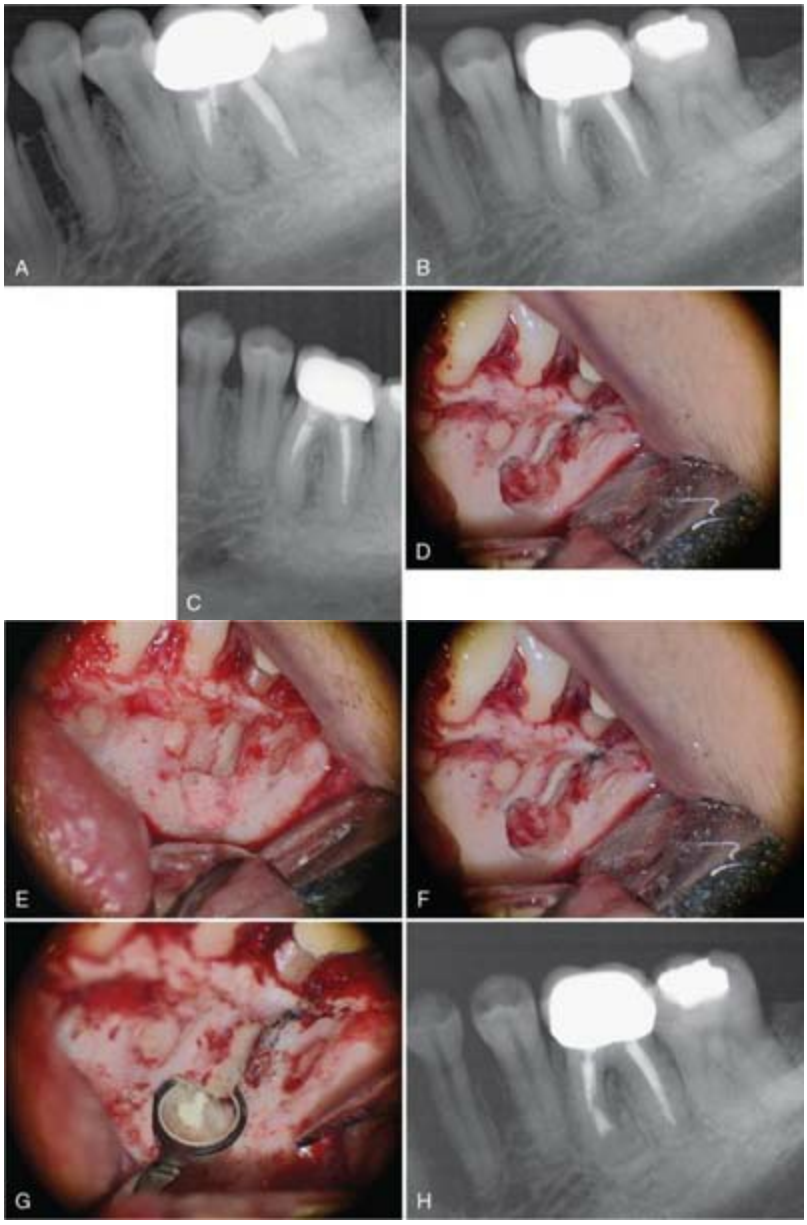

FIG. 11.47 **A,** Preoperative radiograph (mesial angle) of a left mandibular first molar. Periradicular disease and symptoms had persisted after nonsurgical retreatment by an endodontic resident. The mesial canals were completely obstructed at the midroot level. The preoperative evaluation included three periapical radiographs, two different horizontal angulations, and one vertically positioned radiograph. **B,** Straight view preoperative radiograph. **C,** Vertical periapical view preoperative radiograph. **D,** Osteotomy and root resection perpendicular to the long axis of the root were performed; a partial bony dehiscence over the mesial root can be seen. **E,** Racellets were packed into the bony crypt to establish hemostasis. **F,** The Racellets were removed (one usually is left in the deepest part of the crypt during root-end preparation and filling), and the root end was

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beveled perpendicular to the long axis of the root. Methylene blue dye can be useful for identifying the root outline and locating any cracks. Ultrasonic root-end preparation was completed to a depth of 3 mm, connecting the MB and ML canals. **G,** Mineral trioxide aggregate root-end filling was placed and inspected. The bony crypt was then gently curetted to initiate bleeding and to remove any remnants of hemostatic materials. The flap was repositioned, and a radiograph was taken. **H,** An immediate postoperative radiograph confirmed the depth and density of the root-end filling and the absence of any foreign objects. Note that calcium sulfate and bone grafting material was placed in the osseous defect and over the root because of the large buccal dehiscence, although this is not routinely required in these cases.

Set of radiographs and photographs marked A to H depict surgical treatment of left mandibular first molar for periradicular disease.

Source: (Courtesy Dr. Vince Penesis.)

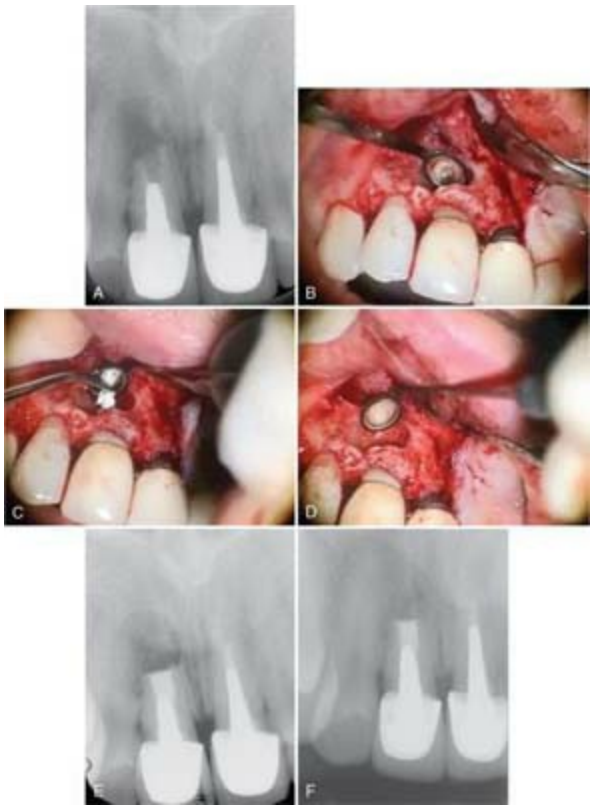

FIG. 11.48 **A,** Preoperative radiograph of a maxillary left central incisor showing evidence of previous surgery but no apparent root-end filling. Because of the short root length and inadequate band of keratinized gingiva, an intrasulcular incision and full-thickness triangular mucoperiosteal flap design were selected. **B,** The root was minimally

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resected, and the root-end cavity was prepared ultrasonically. **C,** Mineral trioxide aggregate was placed and condensed into the root-end cavity preparation. **D,** The root-end filling was inspected before the flap was repositioned and sutured. **E,** Immediate postoperative radiograph. **F,** The 6-month follow-up radiograph showed good initial periradicular healing.

Set of radiographs and photographs marked A to H depict surgical treatment of maxillary left central incisor as follows:

A) Radiograph shows a radiolupency in the apical region of tooth on the left depicting short root length.

B) Close-up of interior of mouth shows diamond coated micromirror placed in the cavity at the root of affected tooth, with flap held by retractor.

C) Close-up of interior of mouth shows a dot of MTA being filled in the hollow cavity.

D) A micromirror is held near the cavity.

E) Radiograph shows a blur spot at the root of affected tooth.

F) Radiograph shows filling extending the canal of till the root of the tooth.

Source: (Courtesy Dr. Shawn Velez.)

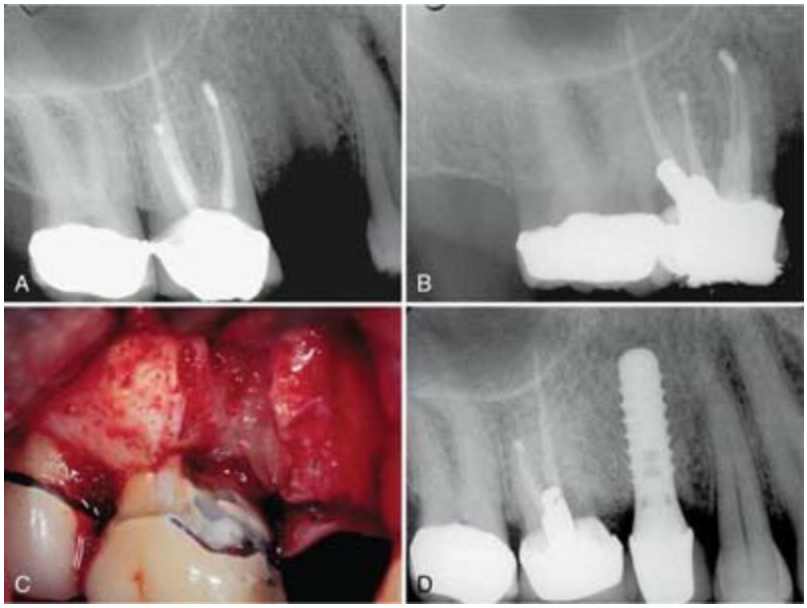

FIG. 11.49 **A,** A maxillary right first molar that was sensitive to percussion and tender to palpation over the MB root. Periodontal

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probing revealed a deep, narrow bony defect on the facial aspect of the MB root. The presence of a vertical root fracture was confirmed visually using magnification and methylene blue dye. The maxillary right second premolar had been recently extracted because of a vertical root fracture. **B,** A bonded amalgam core buildup was placed in the DB and P canals, and Geristore was placed in the MB canal system. **C,** The MB root was resected, and methylene blue dye was used to help define the extent of the fracture. **D,** At the 3-year follow-up visit, a new crown had been fabricated for the molar, and the premolar had been replaced with an implant.

Set of three radiographs and a photograph marked A through depict steps involved in surgical treatment of maxillary right first molar.

Closure of the surgical site and selection of suture material

Closure of the surgical site

The surgical site should be closed only after careful visual and radiographic inspection of the area. Before suturing, a radiograph should be taken with the flap held loosely in place to detect any foreign objects in the crypt or adhering to the flap. This image is also important for confirming the depth and density of the root-end filling. The osteotomy site then is gently curetted and irrigated with sterile saline or water to remove any remnants of hemostatic agents and packing materials. Some bleeding is encouraged at this point, because the blood clot forms the initial scaffold for subsequent healing and repair. If indicated, grafting materials or barriers may be placed at this time. Slight undermining of the unreflected soft tissue adjacent to the flap facilitates the placement of sutures. The flap is then repositioned and gently compressed with a piece of chilled, sterile moist cotton gauze to express excess blood and tissue fluids.

For the common flap designs discussed in this chapter, the corners are first identified and sutured in place with a single interrupted suture. Interrupted sutures are initially passed through the free portion of the flap approximately 2 to 3 mm from the edge and then connected to the attached tissue. The suture is secured with a simple surgeon’s knot, which is positioned away from the incision line. The center of the flap then is located and sutured with

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either an interrupted or a sling suture. A continuous locking suture technique may be used to close a submarginal (Ochsenbein-Luebke) flap.[288] The primary advantage of a continuous suture technique is the ease of suture removal compared with multiple interrupted sutures. The disadvantages are possible difficulty with precise control of tension in each area, and the fact that the entire suture may loosen if one suture pulls through the flap. A sling suture is commonly used for the central tooth in the surgical site to close a full-thickness intrasulcular (rectangular or triangular) flap. The tension on this type of suture can be varied slightly to allow some control of the apicocoronal positioning of the flap. Interrupted sutures then are placed as needed.

When suturing is complete, chilled, sterile moist cotton gauze is again placed over the flap and pressure is applied for 5 minutes. Pressure to the area provides stability for the initial fibrin stage of clot formation and reduces the possibility of excessive postoperative bleeding and hematoma formation under the flap. The iced gauze also supports hemostasis. Final inspection of the area should confirm that all soft-tissue margins have been closely approximated and bleeding has been controlled. An additional injection of long-acting local anesthetic may be administered at this time, although care must be taken not to inject it directly under the newly repositioned flap. The patient is given a cold compress and instructed to hold it on the face in the surgical area, on for 20 minutes and then off for 20 minutes, for the rest of the day. The patient is also given verbal and written postoperative instructions, including after-hours contact information (Fig. 11.50). The patient should sit in an upright position for approximately 15 minutes and the surgical site should be inspected one more time before the patient is discharged.

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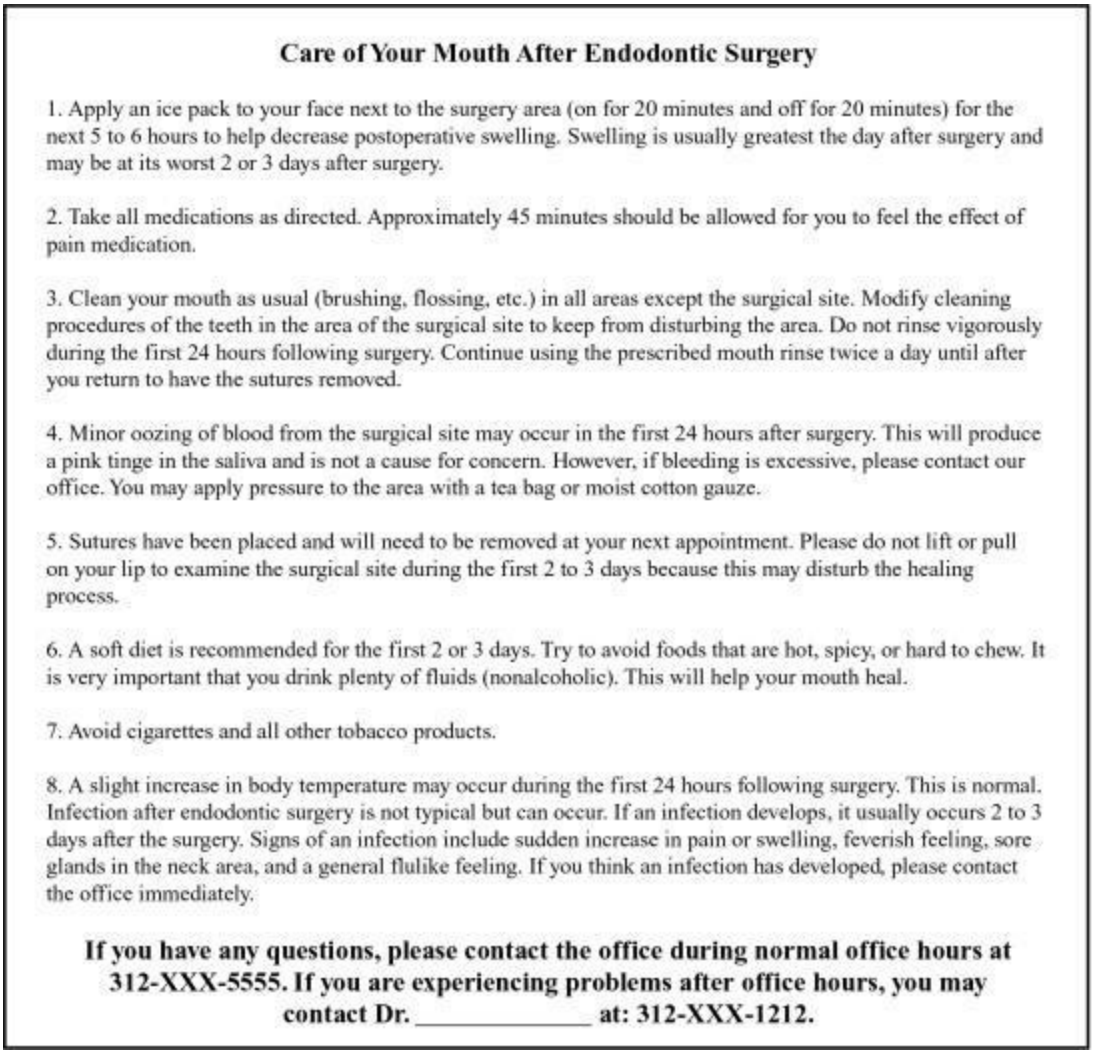

Care of Your Mouth After Endodontic Surgery

1. Apply an ice pack to your face next to the surgery area (on for 20 minutes and off for 20 minutes) for the next 5 to 6 hours to help decrease postoperative swelling. Swelling is usually greatest the day after surgery and may be at its worst 2 or 3 days after surgery.

2. Take all medications as directed. Approximately 45 minutes should be allowed for you to feel the effect of pain medication.

3. Clean your mouth as usual (brushing, flossing, etc.) in all areas except the surgical site. Modify cleaning procedures of the teeth in the area of the surgical site to keep from disturbing the area. Do not rinse vigorously during the first 24 hours following surgery. Continue using the prescribed mouth rinse twice a day until after you return to have the sutures removed.

4. Minor oozing of blood from the surgical site may occur in the first 24 hours after surgery. This will produce a pink tinge in the saliva and is not a cause for concern. However, if bleeding is excessive, please contact our office. You may apply pressure to the area with a tea bag or moist cotton gauze.

5. Sutures have been placed and will need to be removed at your next appointment. Please do not lift or pull on your lip to examine the surgical site during the first 2 to 3 days because this may disturb the healing process.

6. A soft diet is recommended for the first 2 or 3 days. Try to avoid foods that are hot, spicy, or hard to chew. It is very important that you drink plenty of fluids (nonalcoholic). This will help your mouth heal.

7. Avoid cigarettes and all other tobacco products.

8. A slight increase in body temperature may occur during the first 24 hours following surgery. This is normal. Infection after endodontic surgery is not typical but can occur. If an infection develops, it usually occurs 2 to 3 days after the surgery. Signs of an infection include sudden increase in pain or swelling, feverish feeling, sore glands in the neck area, and a general flulike feeling. If you think an infection has developed, please contact the office immediately.

If you have any questions, please contact the office during normal office hours at 312-XXX-5555. If you are experiencing problems after office hours, you may contact Dr. _____ at: 312-XXX-1212.

FIG. 11.50 Example of postoperative instructions. Written instructions provide an essential reference for the patient, because verbal instructions often are difficult to remember after surgery. The instructions may be modified as needed; it is important to provide instructions that the patient can understand. For example, the readability of these instructions is at approximately the eighth-grade level using the Flesch-Kincaid Grade Level scale.

A specimen titled, Care of Your Mouth After Endodontic Surgery, reads eight points as follows:

1. Apply an ice pack to your face next to the surgery area (on for 20 minutes and off for 20 minutes) for the next 5 to 6 hours to help decrease postoperative swelling. Swelling is usually greatest the day after surgery and may be at its worst 2 or 3 days after surgery.

2. Take all medications as directed. Approximately 45 minutes should be allowed for you to feel the effect of pain medication.

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3. Clean your mouth as usual (brushing, flossing, etc.) in all areas except the surgical site. Modify cleaning procedures of the teeth in the area of the surgical site to keep from disturbing the area. Do not rinse vigorously during the first 24 hours following surgery. Continue using the prescribed mouth rinse twice a day until after you return to have the sutures removed.

4. Minor oozing of blood from the surgical site may occur in the first 24 hours after surgery. This will produce a pink tinge in the saliva and is not a cause for concern. However, if bleeding is excessive, please contact our office. You may apply pressure to the area with a tea bag or moist cotton gauze.

5. Sutures have been placed and will need to be removed at your next appointment. Please do not lift or pull on your lip to examine the surgical site during the first 2 to 3 days because this may disturb the healing process.

6. A soft diet is recommended for the first 2 or 3 days. Try to avoid foods that are hot, spicy, or hard to chew. It is very important that you drink plenty of fluids (nonalcoholic). This will help your mouth heal.

7. Avoid cigarettes and all other tobacco products.

8. A slight increase in body temperature may occur during the first 24 hours following surgery. This is normal. Infection after endodontic surgery is not typical but can occur. If an infection develops, it usually occurs 2 to 3 days after the surgery. Signs of an infection include sudden increase in pain or swelling, feverish feeling, sore glands in the neck area, and a general flulike feeling. If you think an infection has developed, please contact the office immediately.

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Selection of the suture material

The properties of an ideal suture material for periradicular surgery include pliability for ease of handling and knot tying, a smooth surface that discourages bacterial growth and wicking of oral fluids, and a reasonable cost. Suture material in size 5-0 is most commonly used, although some clinicians prefer slightly larger (4-0) or smaller (6-0) suture. Sutures smaller than 6-0 tend to cut through the relatively fragile oral tissues when tied with the tension required to approximate the wound margins. Silk suture material has commonly been used in dental surgery for decades and is both inexpensive and easy to handle. However, silk tends to support bacterial growth and allows for a wicking effect around the sutures. For these reasons, other materials are preferable to silk.[92]

Resorbable suture materials (plain gut and chromic gut) are not routinely used for periradicular surgery, although this material may be indicated if the patient will be unavailable for the regular suture removal appointment (48 to 96 hours after surgery) or if the suture will be used in areas of the mouth where access is difficult. The primary problem with resorbable suture materials is the variable rate of resorption—that is, sutures may weaken and dissolve too soon or, more commonly, may remain in the incision area for longer than desired. Gut suture materials are packed in isopropyl alcohol. The handling properties of gut sutures can be improved by immersion in sterile water for 3 to 5 minutes before use.[430]

Suture materials with a smooth Teflon or polybutilate coating (e.g., Tevdec and Ethibond, respectively) are particularly well suited for use in periradicular surgery. Synthetic monofilament suture materials (e.g., Supramid and Monocryl) are also commonly used. These materials are easy to handle and do not promote bacterial growth or wicking of oral fluids to the same extent as silk. Gortex (expanded PTEE-Teflon) sutures have many desirable properties but are more expensive than the previously mentioned materials.

Tissue adhesives such as cyanoacrylate and fibrin glues may hold promise for wound closure after periradicular surgery.[113,196,403,599] Although the currently available research is insufficient to recommend these adhesives as a routine replacement for more traditional suture materials, future applications

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in periradicular surgery are possible.

Guided tissue regeneration and endodontic surgery

The amount and location of bone adjacent to the root structures affect the prognosis of periradicular surgery. Kim and Kratchman[282] propose a six-category classification system to assist in predicting surgical prognosis and determining the need for bone grafting and barrier techniques. Class A (no lesion), class B (small periapical lesion), and class C (large periapical lesion without periodontal communication) all represent situations that are favorable for healing without supplemental grafting or barriers. Class D (similar to class C with independent periodontal pocketing), class E (endodontic-periodontal communication to the apex), and class F (apical lesion with complete loss of buccal bone) represent situations with a more guarded prognosis and usually require concurrent use of bone grafting and barrier techniques. Figs. 11.51–11.63 are examples of cases that required GTR.

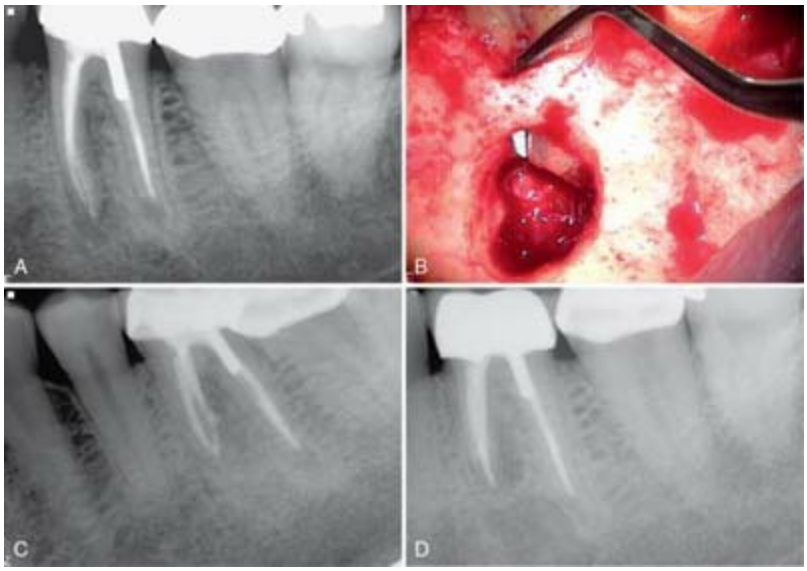

FIG. 11.51 **A,** Preoperative periapical radiograph of tooth #19. **B,** Clinical image demonstrating periodontal defect along the facial aspect of the mesial root. **C,** Immediate postsurgical radiograph. The mesial root end was prepared with ultrasonics and filled with MTA. A mixture of DFDBA and Capse was placed for guided tissue regeneration. **D,** One-year follow-up radiograph demonstrating good healing.

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Periodontal probings were WNL.

A) Radiograph of tooth number 19 shows radiolucent spots in the mesial root.

B) Close-up of interior of mouth shows a hollow cavity present at the facial aspect of mesial root.

C) Radiograph show hollow line present left root of the tooth.

D) Radiograph shows filling depicted as fully grown left root depicted as blur white line in the root of the tooth.

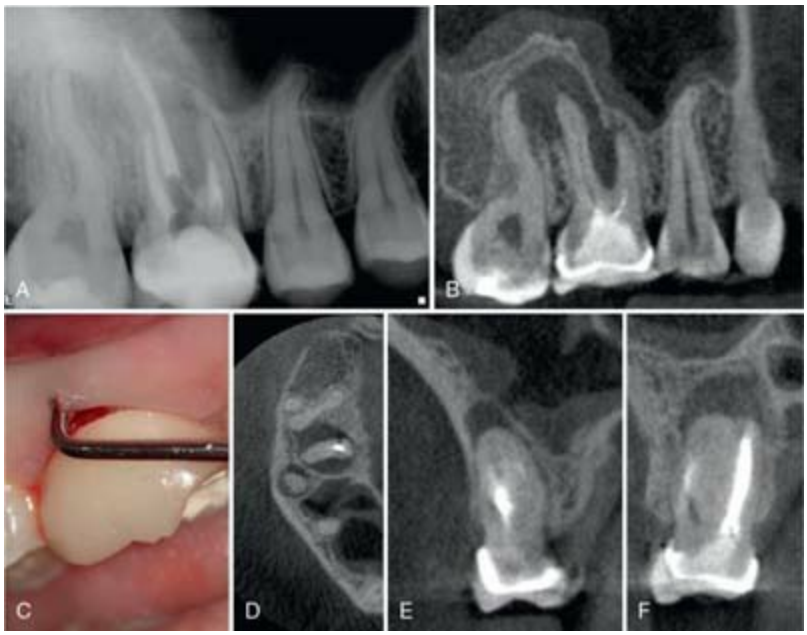

FIG. 11.52 (Case 1): **A,** Periapical radiograph of tooth #3. Retreatment RCT was attempted, but MB1 and MB2 were blocked. **B,** Sagittal view demonstrating the extent of the periapical lesion. **C,** Clinical view of periodontal probing to the apex. **D,** Axial view of the apical one third demonstrating the close proximity of the periapical defect to the MB root of tooth #2 (#2 responded WNL to pulp vitality testing). **E** and **F,** Coronal views of the MB and DB/P roots, respectively.

A)Radiograph shows two black spots on the MB1 and MB2 of maxillary third tooth.

B) Radiograph shows a black spot as the peripheral lesion.

C) Close-up of tooth shows probe inserted in gingiva right above the tooth.

D) Radiograph shows axial view of apical one third depicted as black dot.

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E) Radiograph of coronal view of a tooth shows radiolucent spot in mesobucaal root.

F) Radiograph of coronal view of a tooth shows radiopaque line in the distobuccal root.

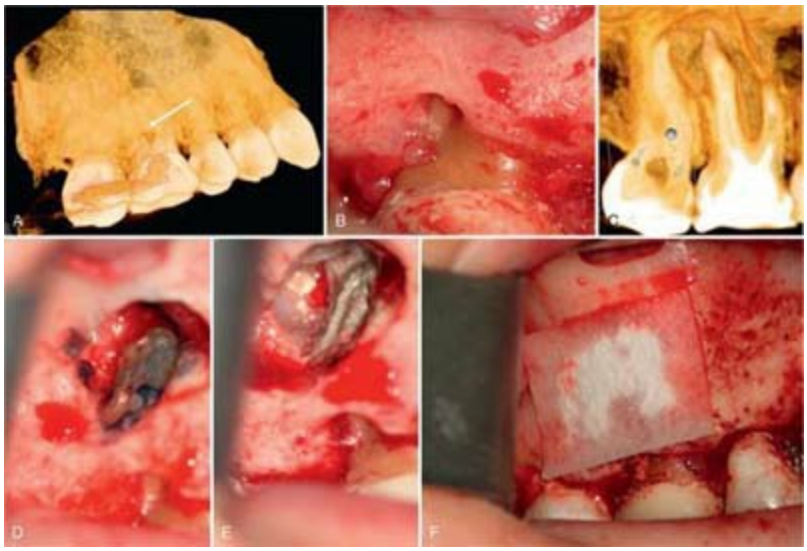

FIG. 11.53 (Case 1, continued): **A,** A 3D reconstruction of the tooth #3 area demonstrating the furcation periodontal defect. **B,** After flap reflection, clinical view demonstrating the periodontal defect. **C,** A 3D reconstruction with the buccal plate cropped to visualize the extent of the periradicular defect. **D** and **E,** DB and P root resection, root-end preparation, root-end filling with mineral trioxide aggregate. **F,** Periradicular defect grafted with Puros allograft (Zimmer Dental, Carlsbad, CA) and CopiOs pericardium membrane (Zimmer Dental).

Set of six photographs marked A through F shows surgical treatment of periodontal defect in third tooth.

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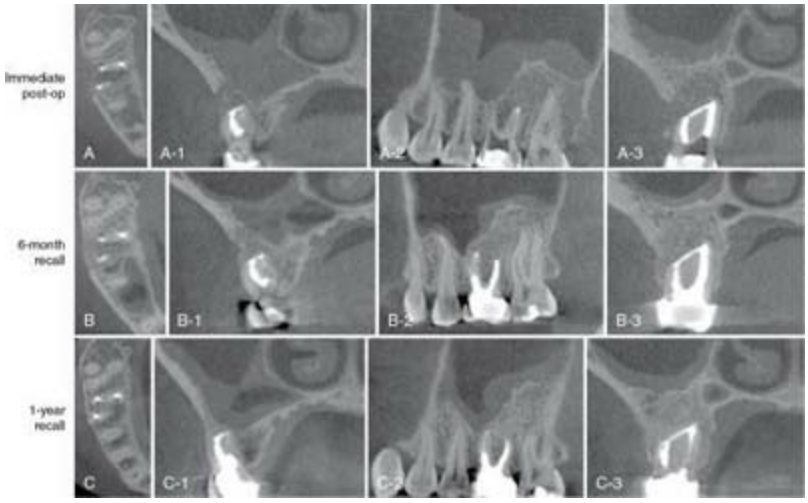

FIG. 11.54 (Case 1, continued): **A,** Axial view of immediate postoperative CBCT scan, A-1 coronal view of MB root, A-2 sagittal view and A-3 coronal view of DB and palatal roots. **B,** A 6-month recall, axial view, B-1 coronal view of MB root, B-2 sagittal view, and B-3 coronal view of DB and palatal roots. **C,** A 1-year recall, axial view, C-1 coronal view of MB root, C-2 sagittal view, and C-3 coronal view of DB and palatal roots.

Set of three radiographs present postoperative results immediate post-op, six-month recall, and 1-year recall.

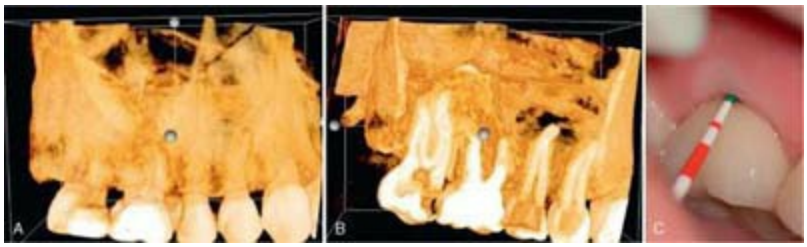

FIG. 11.55 (Case 1, 1-year recall): **A** and **B,** A 3D reconstruction of 1-year recall. **C,** Clinical picture showing resolution of the periodontal defect.

A) 3D reconstruction of five maxillary teeth shows a white tinge on the second teeth from left.

B) 3D reconstruction of four teeth shows a gray dot placed on the palatal root of the second tooth from left.

C) Close-up view of a tooth shows tip of a probe inserted in the gingiva right above the tooth with skin of gingival above tooth turned white.

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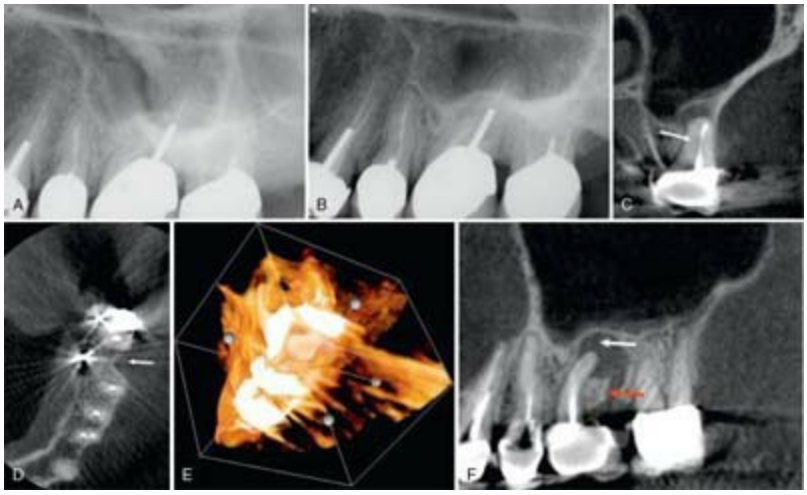

FIG. 11.56 (Case 2, tooth #14): **A** and **B,** Periapical radiographs of tooth #14, mesial and distal angles, respectively. **C,** Coronal view of the MB root demonstrating a missed MB2 canal *(arrow).* **D,** Axial view showing a previous DB root amputation site *(arrow)* that was not obvious in the periapical radiograph. **E,** A 3D reconstruction demonstrating a crestal defect. **F,** Sagittal view demonstrating the crestal defect *(red arrow)* communicating with the periapical lesion *(white arrow),* elevation of the floor of the maxillary sinus but no evidence of sinus perforation.

Set of six radiographs are marked A to F as follows:

A) Radiograph shows the mesial view of four teeth with root of second tooth from left shown radiolucent.

B) Radiograph shows the distal view of four teeth with root of second tooth from left shown as a thin line.

C) Radiograph shows coronal view of tooth with a white arrow pointing at the missing MB2 canal.

D) Radiograph shows an arrow pointing at hollow space between distobuccal root.

E) 3D reconstruction image shows a tooth inside a cube shows four dots marked in different planes.

F) Radiograph shows a red arrow pointing the gray spot of lesion present at the root while a red arrow points at a translucent area to the right of canal of tooth depicting elevation of floor of maxillary sinus.

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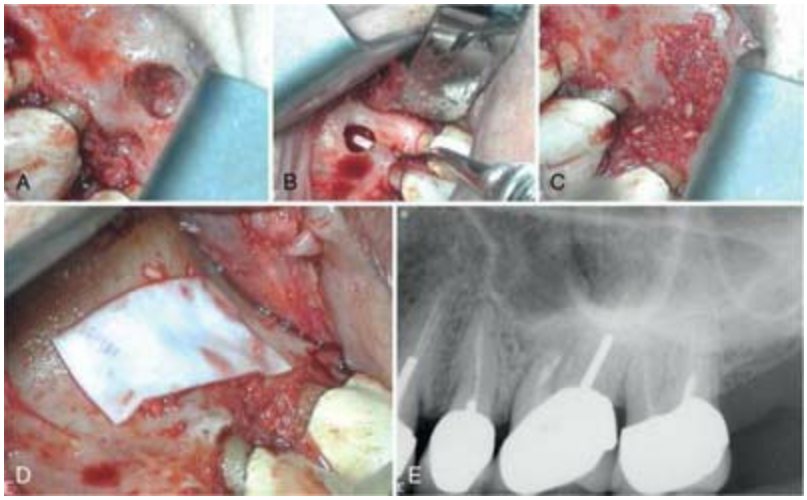

FIG. 11.57 (Case 2, continued): **A,** Clinical view after flap reflection demonstrating both crestal and periapical lesions. **B,** Communication between both defects. **C,** Both defects grafted with EnCore Combination Allograft (Osteogenics Biomedical, Lubbock, TX). **D,** CopiOs pericardium membrane (Zimmer Dental, Carlsbad, CA). **E,** Immediate postoperative radiograph.

A) Close-up of interior of mouth shows a cavity present at the root of the tooth.

B) Close-up of interior of mouth shows a retractor holding the flap with tip of probe inserted in the cavity.

C) Close-up shows cavity filled with grafts.

D) A pericardium membrane is placed above the surgical site.

E) Post operative radiograph shows radiolucent mesobuccal canal 2.

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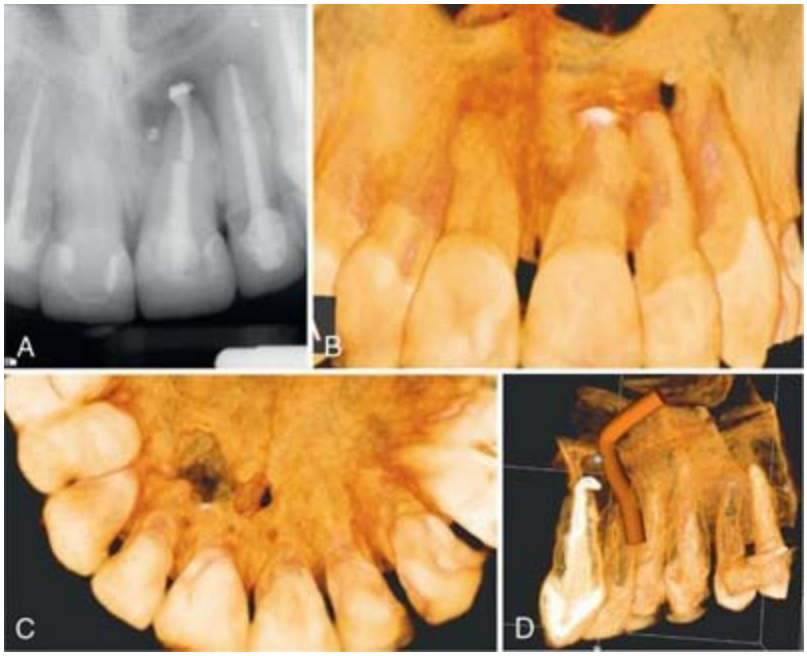

FIG. 11.58 (Case 3): **A,** Periapical radiograph of maxillary anterior region demonstrating a periapical lesion associated with tooth #9. **B,** CBCT reconstruction demonstrating the intact buccal cortical plate. **C,** Palatal view of the 3D reconstruction demonstrating perforation of the palatal plate. **D,** A 3D reconstruction showing the nasopalatine bundle.

Set of radiograph and 3D construction images marked A through D depict results of surgical treatment of periapical lesion on ninth tooth.

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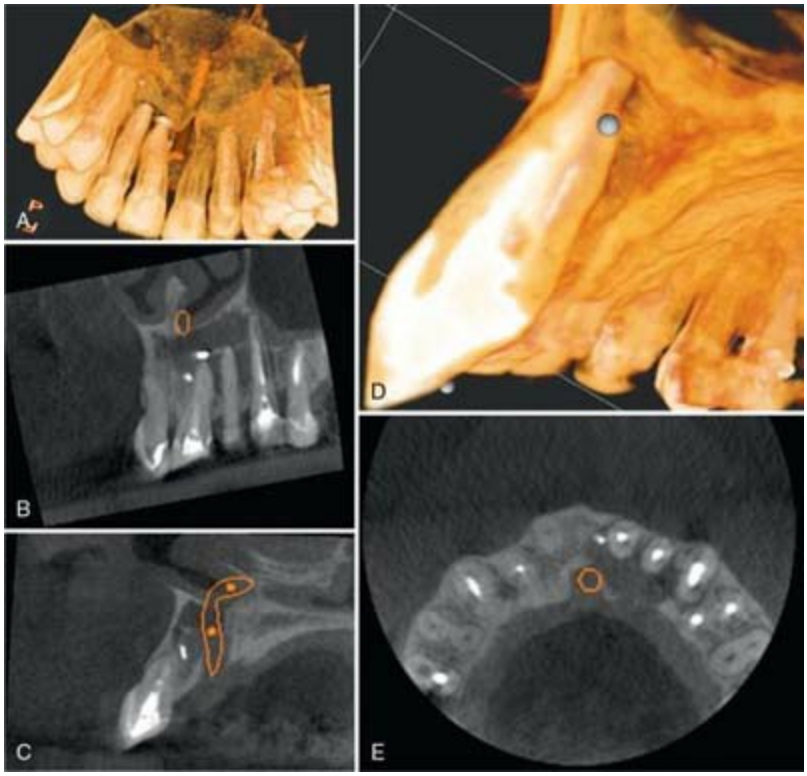

FIG. 11.59 (Case 3, continued): **A,** Palatal view CBCT reconstruction showing the exit of the nasopalatine neurovascular bundle from the incisive canal. **B,** Sagittal view demonstrating the periapical radiolucency involving teeth #9, 10, and 11. **C,** Coronal view the exit of the nasopalatine bundle from incisive canal. **D,** A 3D reconstruction demonstrating the periapical lesion extension to tooth #11. **E,** Axial view demonstrating the extension of the lesion, palatal plate perforation, and relation of the nasopalatine bundle to the periapical lesion.

Set of 3D constructed images and radiographs marked A through E depict continuation of surgical treatment of periapical lesion on ninth tooth.

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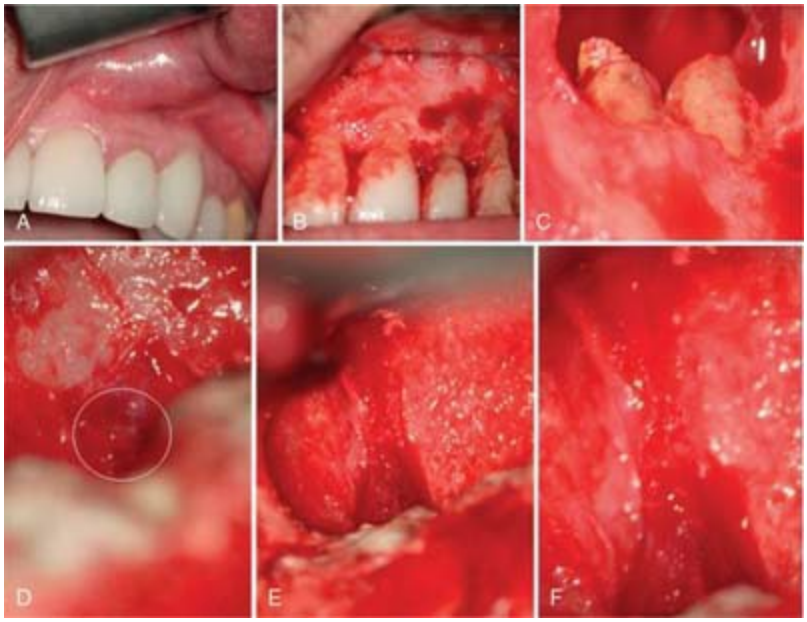

FIG. 11.60 (Case 3, continued): **A** and **B,** Clinical pictures before and after flap reflection showing the intact buccal plate. **C,** Periapical defect after degranulation showing the apices of teeth #9 and 10 prior to resection. **D,** Palatal bone perforation with palatal mucosa evident *(circle).* **E** and **F,** Clinical pictures of the nasopalatine bundle intact after degranulation.

Set of six photographs marked A through F are depicted as follows:

A) Close-up of interior of mouth shows gingiva exposed with flap held by a retractor.

B) Close-up of interior of mouth shows flap closed with sutures.

C) Close-up of interior of mouth shows tip of tooth exposed before resection.

D) Close-up of interior of mouth shows a circle marked on the palatal bone perforation.

E) Magnified of close-up of interior of mouth shows nasopalatine bundle intact.

F) Magnified view of intact nasopalatine bundle.

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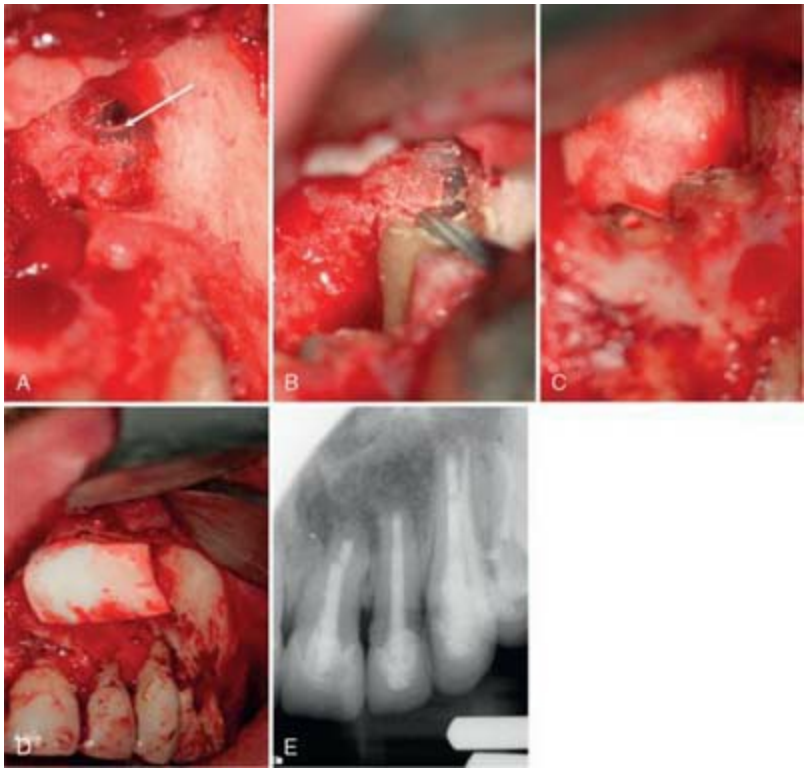

FIG. 11.61 (Case 3, continued): **A** and **B,** Lateral wall of the maxillary sinus distal to tooth #11. **C,** CopiOs membrane placed palatally to cover the palatal mucosa. **D,** CopiOs membrane covering the Puros allografting material. **E,** Through-and-through defect immediate postoperative radiograph.

A) Close-up view of interior of mouth shows an arrow pointing at hollow space in maxillary sinus distal to teeth number 11.

B) Close-up view of interior of mouth shows lateral wall of maxillary sinus distal to teeth number 11.

C) Close-up view of interior of mouth shows graft of CopiOs membrane placed palatally to cover the palatal mucosa.

D) Close-up view of interior of mouth shows CopiOs membrane placed over allograft.

E) Radiograph of four teeth shows radiolucent spot at the apical region of tooth.

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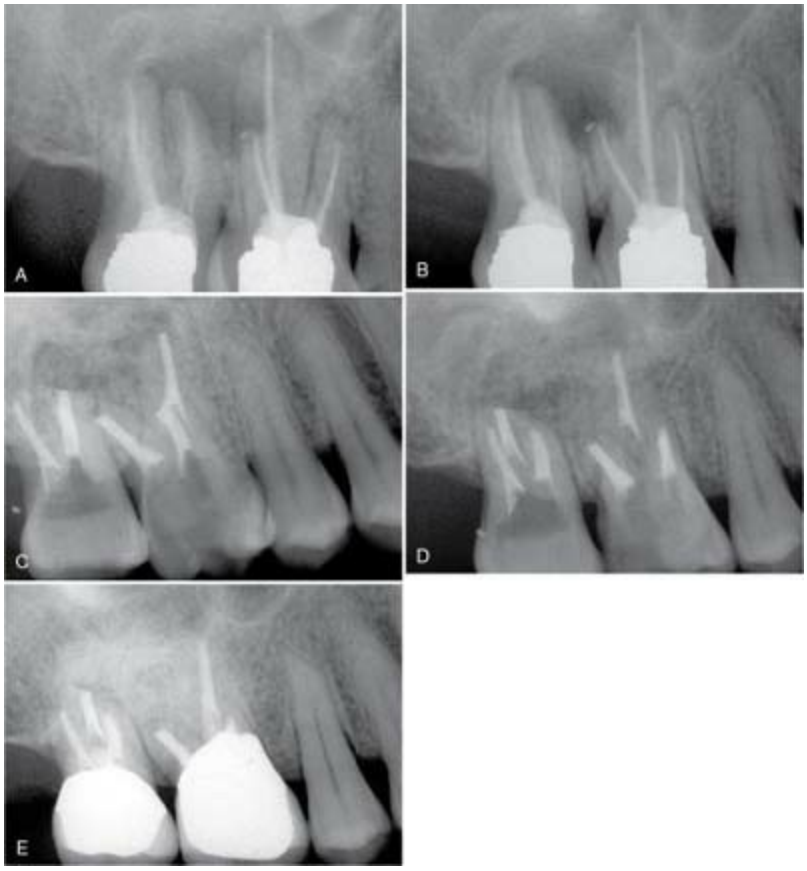

FIG. 11.62 **A,** Preoperative angled radiograph of maxillary right first and second molars. Both teeth had been treated previously, and the patient has reported a history of pain in the area for the past 5 years. The treatment plan included nonsurgical retreatment followed by periradicular surgery with bone grafting and guided tissue regeneration (GTR). **B,** Preoperative straight-on radiograph of the maxillary right first and second molars. **C,** Immediate postoperative radiograph showing root-end resections and fillings. The root ends were prepared with ultrasonics, conditioned with 17% ethylenediaminetetraacetic acid (EDTA), filled with Diaket, and smoothed with a superfine diamond finishing bur. The crypt was packed with BioOss xenograft material, and a Guidor resorbable membrane was placed. **D,** Immediate postoperative radiograph (straight on view). **E,** A 4-year follow-up radiograph. The patient was asymptomatic, and all objective findings were within normal limits. The teeth were restored with porcelain fused to metal crowns.

Set of five radiographs marked A through E depict surgical treatment results of maxillary right first and second molars.

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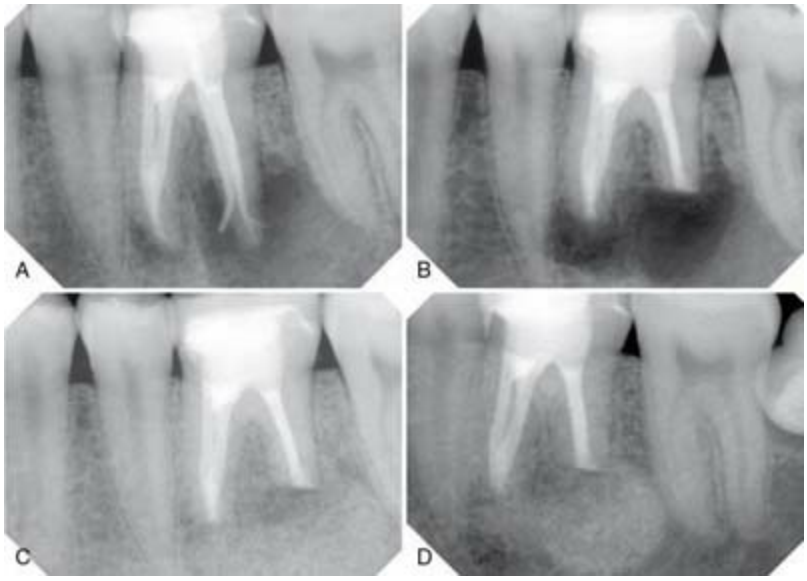

FIG. 11.63 **A,** Preoperative radiograph of a mandibular left first molar. Gutta-percha was inserted into the buccal sulcus and traced to the apex of the distal root. Nonsurgical root canal treatment had been performed 12 months earlier. **B,** Root-end resection and MTA root-end fillings (M and D roots). **C,** Immediate postoperative radiograph. BioOss xenograft material was placed. **D,** The 19-month follow-up radiograph showed good periradicular healing.

Set of four radiographs are marked A through D as follows:

A) Radiograph shows mandibular left first molar shows a gray spot at the apex of distal root.

B) Radiograph shows two black spots, with one each placed at the apex of root.

C) Radiograph shows elimination of spots from apex, with a mild spot on the apex of root.

D) Radiograph shows elimination of spots from apex of root.

An apicomarginal defect[141] or a localized bony defect distinguished by a total deficiency of alveolar bone over the entire root length has a significant adverse effect on the outcome, reducing the rate of complete healing by approximately 20% or more when compared to teeth with an isolated endodontic-only lesion.[233,279,499] The presence of a periradicular lesion 15 mm or greater in diameter has also been linked to a poorer prognosis.[233] Advanced periodontitis with deep pocket formation has been associated with chronic periradicular inflammation after endodontic surgery and subsequent

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failure of the root-end surgery.[450] The cause of failure has been identified as in-growth of nonosteogenic tissues into the periradicular surgical site and down-growth of epithelial tissue along the root surface. Successful treatment may depend more on controlling epithelial proliferation than root-end management. GTR techniques have been advocated for use in such cases.[206,547]

The basic principle of guided tissue and bone regeneration is that different types of cells repopulate a wound at different rates during healing. The soft-tissue cells are considerably more motile than the hard-tissue cells, therefore they tend to migrate into the wound more quickly during healing. A barrier interposed between the gingival tissue and the exposed root surfaces and supporting alveolar bone prevents colonization of the exposed root surface by gingival cells. This encourages selective repopulation of the root surface by PDL cells. The use of an absorbable barrier theoretically would allow PDL cells and other cells with osteogenic potential to repopulate the defect, resulting in new connective tissue attachment and bone formation. Dahlin and coworkers[119,120] demonstrated that in monkeys, a significant increase in osseous healing occurs when membranes are used in through-and-through bone defects in periradicular surgery of the lateral maxillary incisors. The use of resorbable GTR membranes in endodontic surgery with buccal apicomarginal type defects also has been shown to enhance regeneration of the periodontium and surrounding bone in dogs.[149] This type of matrix barrier promoted greater amounts of connective tissue and alveolar bone and minimized the formation of junctional epithelium.

Several case reports have discussed the use of GTR techniques in conjunction with endodontic surgery.[5,32,81,123,154,273,337,414,421,432,515,544,549,607] These studies largely have reported favorable outcomes in cases involving large periradicular lesions, through-and-through bone defects, and repair of a surgical perforation or loss of the buccal cortical plate adjacent to the root.

Pecora and colleagues[400] compared the healing of 20 large periradicular defects (>10 mm diameter) with and without the use of nonresorbable membrane. They reported that at 12 months after surgery, the sites in which membranes had been used had healed more quickly and that the quality and quantity of the regenerated bone was superior. One study evaluated

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periradicular and periodontal healing in cases involving apicomarginal defects when GTR (Bio-Oss and Bio-Gide membrane; Osteohealth Co., Shirley, NY) was performed in conjunction with periradicular surgery. At 12 months after surgery, 86% were considered healed both clinically and radiographically. It was concluded that GTR should be considered as an adjunct to periradicular surgery in cases of apicomarginal defects.[142] However, use of a resorbable membrane when a standard apical osteotomy is performed and the buccal bone over the remainder of the root is intact has no beneficial effect on healing.[186]

Several different types of membranes are available. They can be grouped into two broad categories, nonresorbable and resorbable (Table 11.1). Resorbable membranes are generally better suited for endodontic uses because a second surgical procedure is not required to remove the membrane.

Table 11.1

Examples of Membrane Materials

Composition	Trade Name/Manufacturer
NONRESORBABLE	
Polytetrafluoroethylene	Gortex (WL Gore & Associates Inc, Flagstaff, AZ) TefGen FD (Lifecore Biomedical, Chaska, MN) Bicon Barrier Membrane (Bicon, Boston, MA) Cytoflex (Unicare Biomedical, Laguna Hills, CA)
RESORBABLE	
Laminar bone	Lambone (Pacific Coast Tissues Bank, Los Angeles, CA)
Polylactic acid	Guidor* This product was used extensively in early research with very favorable results (Guidor USA) Atrisorb (CollaGenex Pharmaceuticals, Newtown, PA)
Polyglactic acid	Vicyl Mesh (Ethicon, Somerville, NJ)
Polylactic acid, polyglycolic acid, and trimethylene carbonate	Resolut (WL Gore & Associates Inc, Flagstaff, AZ)

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Collagen	Biomend (Zimmer Dental, Carlsbad, CA) Bio-Guide (Osteohealth, Shirley, NY) Bicon Resorbable Collagen Membrane (Bicon, Boston, MA)

*No longer available.

Membranes frequently require support so that the membrane does not collapse into the defect itself. Support for the membrane may be provided by using either a titanium-tented membrane or a graft material. Graft materials have two main functions: to act as a mechanical substructure that supports the membrane and the overlying soft tissues and to serve as a biologic component that enhances bone formation. Bone graft materials (Table 11.2) can be categorized as osteoconductive or osteoinductive. An osteoconductive material provides a framework into which bone can grow. The pore size of the material is similar to that of normal bone, and the material eventually is absorbed and remodeled. An osteoinductive material stimulates the production of new bone cells such that healing occurs more quickly. The BMP family has been investigated extensively for use in this role. A combination of osteoconductive and osteoinductive materials also can be used for bone grafts.

Table 11.2

Examples of Bone Graft Materials

Graft Type	Description	Product/Manufacturer or Source
Autogenous graft	Obtained from patient's own body	Ramus, chin, iliac crest
Allograft	Demineralized freeze-dried human bone (DFDBA)	Osteofil (Regeneration Technologies, Alachua, FL) Grafton (Osteotech, Eatontown, NJ) Dynagraft (GenSci, Toronto, Ontario, Canada) Opteform (Exactech, Gainesville, FL) Puros (Zimmer Dental, Carlsbad CA)

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		MTF DeMin Bone (DENTSPLY Friadent CeraMed, Lakewood, CO)
Xenograft	Inorganic bovine/porcine bone particles	BioOss (Osteohealth, Shirley, NY) OsteoGraf (DENTSPLY Friadent CeraMed, Lakewood, CO)
Ceramic/synthetic grafts	Calcium sulfate, calcium phosphate/hydroxyapatite, bioactive glass	CapSet (Lifecore Biomedical, Chaska, MN) OsteoSet (Wright Medical Technology, Arlington, TN) HTR (Bioplant HTR, Kerr Corporation, West Collins, CA) Biogran (3i, Palm Beach Gardens, FL) Norian SRS (Synthes, West Chester, PA) NovaBone-C/M (NovaBone Products, LLC, Sales and Manufacturing, Alachua, FL) PerioGlas (NovaBone Products, LLC, Sales and Manufacturing, Alachua, FL)
Bioactive proteins	Bone morphogenic proteins (BMPs)	Experimental
Combination graft	Allograft, xenograft, or ceramic/synthetic grafts plus bioactive protein	PepGen P15 (DENTSPLY Friadent CeraMed, Lakewood, CO)

The use of GTR techniques raises several additional issues that should be discussed with the patient before surgery. These include the cost of the additional material, the origin of the material (synthetic, animal, or human), the need to manage the wound for a longer period, and potential postoperative complications related specifically to these techniques and materials. Discussion of the composition of the materials to be used is very important, because some patients may have concerns based on religious or ethical grounds. The surgeon must discuss all the ramifications of using these materials with the patient before beginning the procedure, because it is not

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always possible to predict before surgery when grafting materials may be needed.

If GTR techniques are to be used during periradicular surgery, a resorbable membrane should be chosen and a protocol should be followed (see Figs. 11.62 and 11.63):

1. The membrane is extended to cover 2 to 3 mm of bone peripheral to the margins of the crypt; it should be supported with a bone substitute graft material so that it does not collapse into the crypt or onto underlying tooth structures.
2. Tissue closure techniques should ensure total tissue coverage of the membrane. The traditional postoperative compression is eliminated, because this would collapse the membrane onto the underlying structures.
3. Smoking is contraindicated with GTR techniques because it consistently has been shown to affect the outcome adversely.[73,324,445,525,538,539]

Ridge preservation

With the growing use of dental implants for the replacement of missing teeth, clinicians should be aware of ridge preservation strategies, even if they do not place implants.[308] As an example, ridge preservation should be considered when a tooth is determined to have a vertical root fracture during an exploratory surgical procedure and is extracted. In this situation, there is often a complete absence of the buccal bony plate, and simple extraction of the tooth would predispose the patient to a loss of ridge height and width, thereby complicating future implant placement. GTR with graft and barrier placement (as previously described) at the time of extraction may be indicated to create a more favorable site for future implant placement.[31,251,342,608] An atraumatic extraction technique is desirable, as one of the goals is to preserve the maximum amount of existing bone. Periotomes are particularly useful for this type of bone-preserving extraction technique.

Intentional replantation

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Intentional replantation may be an option when surgical access is limited or presents unacceptable risks. Mandibular second molars are a common example for this technique because of the typically thick overlying buccal bone, shallow vestibular depth, and proximity of the root apices to the mandibular canal (Fig. 11.64). However, any tooth that can be atraumatically removed in one piece is a potential candidate for intentional replantation. Contraindications include teeth with flared or moderately curved roots and the presence of periodontal disease. Vertical root fracture has often been considered a contraindication,[401] although some investigators have demonstrated moderate success using a dentin-bonded resin and intentional replantation for the treatment of teeth with root fractures.[226,268,506] The prognosis was generally better for incisors and for teeth with fractures less than two thirds of the root length. Clinical success after 1 year was about 89% and decreased to 59% at 5 years.[226]

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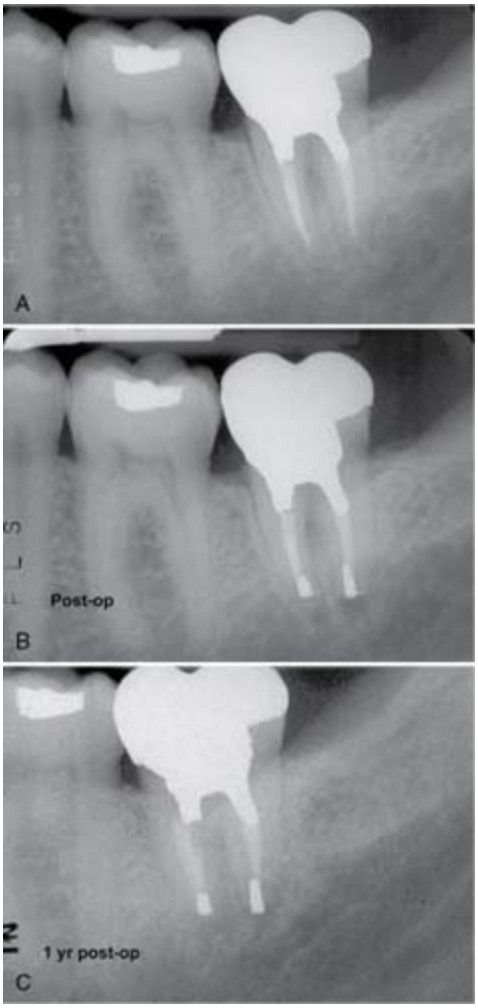

FIG. 11.64 Intentional replantation. **A,** Preoperative radiograph of a mandibular left second molar. The tooth was persistently sensitive to percussion and biting after nonsurgical retreatment. **B,** Radiograph of the tooth immediately after extraction, root-end preparation and filling, and replantation. **C,** At the 1-year follow-up visit, the tooth was asymptomatic and showed good periradicular healing.

Set of three radiographs are marked A through C as follows:

A) Radiograph of mandibular left second molar shows blur, narrow lines of canals.

B) Radiograph shows two radiopaque spots present at the bottom of the root.

C) Radiograph shows healing of tooth post treatment depicted as increase in radiopaque region of tooth.

Source: (Courtesy Dr. Matt Davis.)

The tooth should be extracted with minimal trauma to the tooth and socket.

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Ideally, elevators are not used and the root surface is not engaged with the forceps. All instruments and materials for root-end preparation and filling should be arranged before extraction to minimize extraoral working time. The root surface must be kept moist by wrapping the root with gauze soaked in a physiologic solution, such as Hank's Balanced Salt Solution. After root-end preparation and filling (as described previously in this chapter), the tooth is replanted and the buccal bone is compressed. The patient may be instructed to bite on a cotton roll or other semisolid object to help position the tooth properly in the socket. Occlusal adjustment is indicated to minimize traumatic forces on the tooth during the initial stage of healing. A splint may be applied, but this is often not necessary. The patient should eat a soft diet and avoid sticky foods, candy, and chewing gum for at least 7 to 10 days. Based on clinical observations and several animal model studies, the prognosis for successful healing after replantation is most closely related to avoiding trauma to the PDL and cementum during extraction and minimizing extraoral time.[21,23,391]

Postoperative care

As previously noted, NSAIDs are generally the preferred class of drugs for managing postoperative pain.[9,41,54,143] Ibuprofen (400 to 800 mg) or an equivalent NSAID typically is given before or immediately after surgery and can be continued for several days postoperatively as needed. When additional pain relief is required, a narcotic such as codeine, hydrocodone, or tramadol may be added to the standard NSAID regimen. This strategy may result in a synergistic effect, and therefore greater pain relief, than would be expected with the separate analgesic value of each drug.[148] A useful short-term approach to the management of moderate to severe pain is a "by the clock" alternating schedule of an NSAID and an acetaminophen/narcotic combination.[250,348] Pain after periradicular surgery is typically only mild to moderate. Postoperative pain usually is managed quite well with NSAIDs only, especially when the previously recommended strategy of preoperative NSAID therapy and a long-acting local anesthetic is combined with a minimally traumatic surgical approach.

Sutures are commonly removed 2 to 4 days after surgery.[92,213] This

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recommendation is based on the current understanding of wound healing and the desire to remove any potential irritants from the incision area as soon as possible. Local anesthesia is rarely required, although application of a topical anesthetic may be helpful, especially to releasing incisions in nonkeratinized mucosa. Sharp suture scissors or a #12 scalpel blade can be used to cut the sutures before they are removed with cotton pliers or tissue forceps. A transient bacteremia can be expected after suture removal, even when a preprocedural chlorhexidine mouth rinse is used.[79] Antibiotic coverage should be considered only for patients at high risk of developing bacterial endocarditis.

If healing is progressing normally at the suture removal appointment, the patient does not need to be seen again in the office until the first scheduled recall examination, typically 3 to 12 months after surgery. However, phone contact with the patient approximately 7 to 10 days after suture removal is recommended to confirm the absence of problems. Patients with questionable healing at the suture removal appointment should be reevaluated in the office in 7 to 10 days or sooner if necessary.

Management of surgical complications

Although serious postoperative surgical complications are rare, the clinician should be prepared to respond to patient concerns and recognize when additional treatment may be necessary. Careful case evaluation, adherence to a minimally traumatic surgical technique, and proper patient management, as described previously in this chapter, should result in a low incidence of postoperative complications. Even so, some patients experience mild to moderate postoperative pain, swelling, ecchymosis, or infection. In a prospective study of 82 patients undergoing endodontic surgical treatment, Tsesis and coworkers[545] reported that 76.4% were pain free 1 day after surgery and 64.7% did not report any swelling. Only 4% of the patients in this study experienced moderate pain, and this sequela was closely related to the presence of presurgical symptoms. Postoperative pain typically peaks the day of surgery and swelling reaches its maximum 1 to 2 days after surgery.[294] As previously noted, good evidence supports the use of prophylactic NSAID therapy and a long-acting local anesthetic to reduce the magnitude and

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duration of postoperative pain.

Patients should be advised that some postoperative oozing of blood is normal, but significant bleeding is uncommon and may require attention. Most bleeding can be controlled by applying steady pressure for 20 to 30 minutes, typically with a piece of moist cotton gauze or a tea bag. Bleeding that persists requires attention by the clinician. Pressure to the area and injection of a local anesthetic containing 1:50,000 epinephrine are reasonable first steps. If bleeding continues, it may be necessary to remove the sutures and search for a small severed blood vessel. When located, the blood vessel can be crushed or cauterized to control bleeding. Cauterization may be performed with a heat source commonly used for warm obturation techniques. Local hemostatic agents, as previously described, may also be used. Occasionally, a patient may require hospitalization and surgical intervention to control bleeding, but this is an extremely rare event. Extraoral ecchymosis (Fig. 11.65) occurs when blood seeps through the interstitial tissues; although it may be alarming to the patient and clinician, this condition is self-limiting and does not affect the prognosis.[281] Moist heat applied to the area may be helpful, although complete resolution of the discoloration may take up to 2 weeks. Heat should not be applied to the face during the first 24 hours after surgery.

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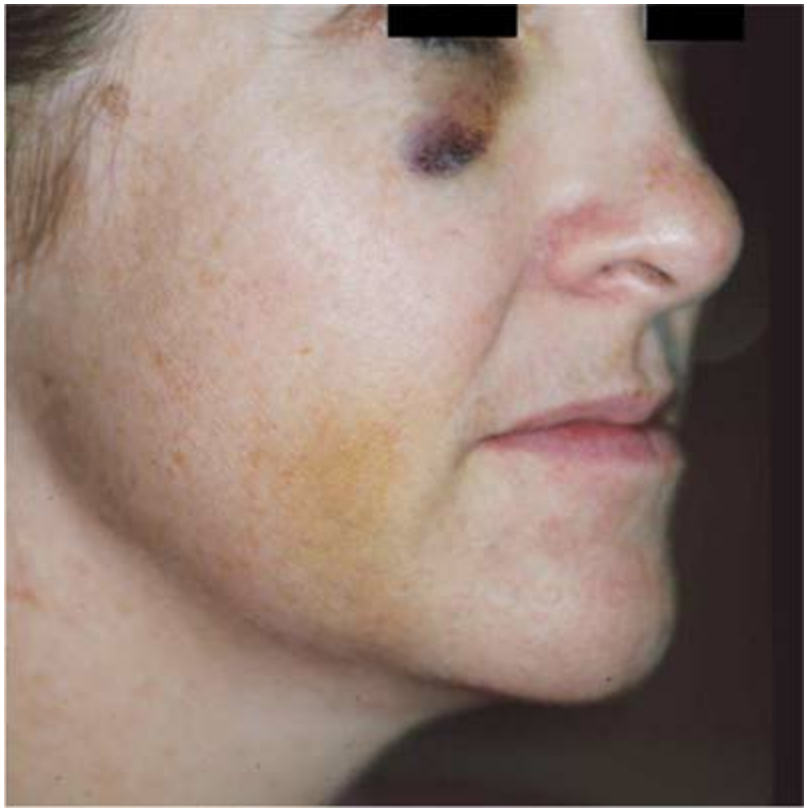

FIG. 11.65 Postoperative ecchymosis can be alarming to the patient but resolves spontaneously within 7 to 14 days.

Lateral view of a patient's face shows purple blue bruise below the eye and a yellow patch present below cheek on the mandibular region.

Sinus exposure during surgical root canal procedures on maxillary posterior teeth is not uncommon. Postoperative antibiotics and decongestants are often recommended[16,29,281,568]; however, this practice is controversial, and no evidence supports the routine use of antibiotics and decongestants in these cases. Walton[576] makes a persuasive argument that antibiotics are not routinely indicated for the management of sinus exposures during periradicular surgery when primary closure of the oral-antral communication is possible. Further support for this position is provided by other clinicians who have observed excellent healing and minimal complications after sinus exposure during periradicular surgery.[291,456,579] Clinical judgment should guide the use of antibiotics and decongestants on a case-by-case basis until more conclusive evidence on this practice is available.

No reliable data are available to provide an accurate estimate of the likelihood of paresthesia after surgical root canal treatment. The incidence of

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paresthesia after third molar removal is estimated to be 1% to 4.4%[443]; however, most reported cases of paresthesia after third molar extraction involved the lingual nerve, which is rarely encountered in mandibular periradicular surgery. The incidence of damage to the inferior alveolar nerve after third molar surgery is approximately 1.3%, with only about 25% of these cases resulting in permanent injury.[551] Unless the nerve is resected during surgery, most patients can be expected to return to normal sensation within 3 to 4 months. If the paresthesia does not show signs of resolving within 10 to 12 weeks, referral and evaluation for possible neuromicrosurgical repair should be considered.[167,431] Robinson and Williams[443] presented a useful method for charting and documenting paresthesias. The area of altered sensation is determined by pinching the skin or mucosa with cotton pliers; alternatively, a pinprick can be applied with a sharp instrument. The area of paresthesia is noted with a series of marks on a diagram of the face and mouth. This method provides a graphic and chronologic record of the paresthesia.

Summary

Periradicular surgery today bears little resemblance to the surgical procedures commonly performed as recently as the 1990s. Enhanced magnification and illumination, microsurgical instruments, ultrasonics, new materials for hemostasis, root-end filling, and GTR, and a greater understanding of the biology of wound healing and the etiology of persistent periradicular disease all have contributed to the rapid evolution of periradicular surgery. With proper case selection and operator skill, periradicular surgery can be considered a predictable, cost-effective alternative to extraction and tooth replacement.

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References

1. Abbott PV. *Analysis of a referral-based endodontic practice: part 2. Treatment provided*, J Endod 1994;20: 253.
2. Abedi HR, Van Mierlo BL, Wilder-Smith P, et al: *Effects of ultrasonic root-end cavity preparation on the root apex*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;80: 207.
3. Abitbol T, Santi E, Scherer W. *Use of a resin-ionomer in guided tissue regeneration: case reports*, Am J Dent 1995;8: 267.
4. Abitbol T, Santi E, Scherer W, et al: *Using a resin-ionomer in guided tissue regenerative procedures: technique and application–case reports*, Periodontal Clin Investig 1996;18: 17.
5. Abramowitz PN, Rankow H, Trope M. *Multidisciplinary approach to apical surgery in conjunction with the loss of buccal cortical plate*, Oral Surg Oral Med Oral Pathol 1994;77: 502.
6. Adamo HL, Buruiana R, Schertzer L, et al: *A comparison of MTA, Super-EBA, composite and amalgam as root-end filling materials using a bacterial microleakage model*, Int Endod J 1999;32: 197.
7. Affairs RotCoS: *Dental management of patients receiving oral bisphosphonate therapy—expert panel recommendations*, American Dental Association.
8. Aframian DJ, Lalla RV, Peterson DE. *Management of dental patients taking common hemostasis-altering medications*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod Suppl 45, 2007;103: e1.

Telegram: @dental_k

www.konkur.in

9. Ahlstrom U, Bakshi R, Nilsson P, et al: *The analgesic efficacy of diclofenac dispersible and ibuprofen in postoperative pain after dental extraction*, Eur J Clin Pharmacol 1993;44: 587.
10. Ainamo J, Loe H. *Anatomical characteristics of gingiva: a clinical and microscopic study of the free and attached gingiva*, J Periodontol 1966;37: 5.
11. Ainsworth G. *Preoperative clindamycin prophylaxis does not prevent postoperative infections in endodontic surgery*, Evid Based Dent 2006;7: 72.
12. Al-Bayaty HF, Murti PR, Thomson ER, et al: *Painful, rapidly growing mass of the mandible*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 7.
13. Alger FA, Solt CW, Vuddhakanok S, et al: *The histologic evaluation of new attachment in periodontally diseased human roots treated with tetracycline-hydrochloride and fibronectin*, J Periodontol 1990;61: 447.
14. Alleyn CD, O'Neal RB, Strong SL, et al: *The effect of chlorhexidine treatment of root surfaces on the attachment of human gingival fibroblasts in vitro*, J Periodontol 1991;62: 434.
15. Altman RD, Latta LL, Keer R, et al: *Effect of nonsteroidal antiinflammatory drugs on fracture healing: a laboratory study in rats*, J Orthop Trauma 1995;9: 392.
16. Altonen M. *Transantral, subperiosteal resection of the palatal root of maxillary molars*, Int J Oral Surg 1975;4: 277.
17. Ambus C, Munksgaard EC. *Dentin bonding agents and composite retrograde root filling*, Am J Dent 1993;6: 35.
18. Anan H, Akamine A, Hara Y, et al: *A histochemical study of bone remodeling during experimental apical periodontitis in rats*, J Endod 1991;17: 332.

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www.konkur.in

19. Anderson HC. *Mechanism of mineral formation in bone*, Lab Invest 1989;60: 320.
20. Anderson HC. *Molecular biology of matrix vesicles*, Clin Orthop Relat Res 1995;314): 266.
21. Andreasen JO, Borum MK, Jacobsen HL, et al: *Replantation of 400 avulsed permanent incisors. 4. Factors related to periodontal ligament healing*, Endod Dent Traumatol 1995;11: 76.
22. Andreasen JO, Munksgaard EC, Fredebo L, et al: *Periodontal tissue regeneration including cementogenesis adjacent to dentin-bonded retrograde composite fillings in humans*, J Endod 1993;19: 151.
23. Andreasen JO, Pitt Ford TR. *A radiographic study of the effect of various retrograde fillings on periapical healing after replantation*, Endod Dent Traumatol 1994;10: 276.
24. Andreasen JO, Rud J. *Correlation between histology and radiography in the assessment of healing after endodontic surgery*, Int J Oral Surg 1972;1: 161.
25. Andreasen JO, Rud J, Munksgaard EC. *[Retrograde root obturations using resin and a dentin bonding agent: a preliminary histologic study of tissue reactions in monkeys]*, Tandlaegebladet 1989;93: 195.
26. *Retroplast*, Rønne, Denmark, 2004, Retroplast Trading.
27. Apaydin ES, Shabahang S, Torabinejad M. *Hard-tissue healing after application of fresh or set MTA as root-end-filling material*, J Endod 2004;30: 21.
28. Ardekian L, Gaspar R, Peled M, et al: *Does low-dose aspirin therapy complicate oral surgical procedures?*, J Am Dent Assoc 2000;131: 331.
29. Arens D. *Surgical endodontics*, Cohen S Burns RC Pathways of the pulp ed 4 1987; Mosby St. Louis.
30. Arnold JW, Rueggeberg FA, Anderson RW, et al: *The*

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www.konkur.in

disintegration of superEBA cement in solutions with adjusted pH and osmolarity, J Endod 1997;23: 663.

31. Artzi Z, Tal H, Dayan D. *Porous bovine bone mineral in healing of human extraction sockets. Part 1: histomorphometric evaluations at 9 months*, J Periodontol 2000;71: 1015.
32. Artzi Z, Wasersprung N, Weinreb M, et al: *Effect of guided tissue regeneration on newly formed bone and cementum in periapical tissue healing after endodontic surgery: an in vivo study in the cat*, J Endod 2012;38: 163.
33. Ashcroft GS, Mills SJ, Lei K, et al: *Estrogen modulates cutaneous wound healing by downregulating macrophage migration inhibitory factor*, J Clin Invest 2003;111: 1309.
34. Aukhil I. *Biology of wound healing*, Periodontology 2000 2000;22: 44.
35. Aurelio J, Chenail B, Gerstein H. *Foreign-body reaction to bone wax: report of a case*, Oral Surg Oral Med Oral Pathol 1984;58: 98.
36. Azzi R, Kenney EB, Tsao TF, et al: *The effect of electrosurgery on alveolar bone*, J Periodontol 1983;54: 96.
37. Babay N. *Comparative SEM study on the effect of root conditioning with EDTA or tetracycline HCl on periodontally involved root surfaces*, Indian J Dent Res 2000;11: 53.
38. Bader JD, Bonito AJ, Shugars DA. *Cardiovascular effects of Epinephrine on hypertensive dental patients: evidence report/technology assessment number 48*, AHRQ Publication No. 02-E006 July 2002; Agency for Healthcare Research and Quality Rockville, MD.
39. Bahcall J, Barss J. *Orascopic visualization technique for conventional and surgical endodontics*, Int Endod J

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2003;36: 441.
40. Bahcall JK, DiFiore PM, Poulakidas TK. *An endoscopic technique for endodontic surgery*, J Endod 1999;25: 132.
41. Bakshi R, Frenkel G, Dietlein G, et al: *A placebo-controlled comparative evaluation of diclofenac dispersible versus ibuprofen in postoperative pain after third molar surgery*, J Clin Pharmacol 1994;34: 225.
42. Bang G, Urist MR. *Bone induction in excavation chambers in matrix of decalcified dentin*, Arch Surg 1967;94: 781.
43. Bang G, Urist MR. *Recalcification of decalcified dentin in the living animal*, J Dent Res 1967;46: 722.
44. Barkhordar RA, Pelzner RB, Stark MM. *Use of glass ionomers as retrofilling materials*, Oral Surg Oral Med Oral Pathol 1989;67: 734.
45. Barnes D, Adachi E, Iwamoto C, et al: Testing of the White Version of ProRoot® MTA Root Canal Repair Material1 2002; Tulsa, Oklahoma DENTSPLY Tulsa Dental.
46. Barry MJ. *Health decision aids to facilitate shared decision making in office practice*, Ann Intern Med 2002;136: 127.
47. Behnia A, Strassler HE, Campbell R. *Repairing iatrogenic root perforations*, J Am Dent Assoc 2000;131: 196.
48. Beling KL, Marshall JG, Morgan LA, et al: *Evaluation for cracks associated with ultrasonic root-end preparation of gutta-percha filled canals*, J Endod 1997;23: 323.
49. Bell E, Ehrlich HP, Sher S, et al: *Development and use of a living skin equivalent*, Plast Reconstr Surg 1981;67: 386.
50. Benninger MS, Sebek BA, Levine HL. *Mucosal regeneration of the maxillary sinus after surgery*,

Telegram: @dental_k

www.konkur.in

Otolaryngol Head Neck Surg 1989;101: 33.
51. Berbari EF, Osmon DR, Carr A, et al: *Dental procedures as risk factors for prosthetic hip or knee infection: a hospital-based prospective case-control study*, Clin Infect Dis 2010;50: 8.
52. Berry JE, Zhao M, Jin Q, et al: *Exploring the origins of cementoblasts and their trigger factors*, Connect Tissue Res 2003;44: 97.
53. Bhaskar SN. *Bone lesions of endodontic origin*, Dent Clin North Am 1967;521:.
54. Biddle C. *Meta-analysis of the effectiveness of nonsteroidal anti-inflammatory drugs in a standardized pain model*, AANA J 2002;70: 111.
55. Biggs JT, Benenati FW, Powell SE. *Ten-year in vitro assessment of the surface status of three retrofilling materials*, J Endod 1995;21: 521.
56. Bigras BR, Johnson BR, BeGole EA, et al: *Differences in clinical decision making: a comparison between specialists and general dentists*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 963.
57. Bjorenson JE, Grove HF, List MG Sr, et al: *Effects of hemostatic agents on the pH of body fluids*, J Endod 1986;12: 289.
58. Block RM, Bushell A, Rodrigues H, et al: *A histopathologic, histobacteriologic, and radiographic study of periapical endodontic surgical specimens*, Oral Surg Oral Med Oral Pathol 1976;42: 656.
59. Blomlof J. *Root cementum appearance in healthy monkeys and periodontitis-prone patients after different etching modalities*, J Clin Periodontol 1996;23: 12.
60. Blomlof J, Jansson L, Blomlof L, et al: *Long-time etching at low pH jeopardizes periodontal healing*, J Clin

Telegram: @dental_k

www.konkur.in

Periodontol 1995;22: 459.
61. Blomlof J, Jansson L, Blomlof L, et al: *Root surface etching at neutral pH promotes periodontal healing*, J Clin Periodontol 1996;23: 50.
62. Blomlof J, Lindskog S. *Periodontal tissue-vitality after different etching modalities*, J Clin Periodontol 1995;22: 464.
63. Blomlof J, Lindskog S. *Root surface texture and early cell and tissue colonization after different etching modalities*, Eur J Oral Sci 1995;103: 17.
64. Blomlof JP, Blomlof LB, Lindskog SF. *Smear removal and collagen exposure after non-surgical root planing followed by etching with an EDTA gel preparation*, J Periodontol 1996;67: 841.
65. Bohsali K, Pertot WJ, Hosseini B, et al: *Sealing ability of super EBA and Dyract as root-end fillings: a study in vitro*, Int Endod J 1998;31: 338.
66. Boioli LT, Penaud J, Miller N. *A meta-analytic, quantitative assessment of osseointegration establishment and evolution of submerged and non-submerged endosseous titanium oral implants*, Clin Oral Implants Res 2001;12: 579.
67. Bonine FL. *Effect of chlorhexidine rinse on the incidence of dry socket in impacted mandibular third molar extraction sites*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 154.
68. Bornstein MM, Lauber R, Sendi P, et al: *Comparison of periapical radiography and limited cone-beam computed tomography in mandibular molars for analysis of anatomical landmarks before apical surgery*, J Endod 2011;37: 151.
69. Bornstein MM, Wasmer J, Sendi P, et al: *Characteristics*

Telegram: @dental_k

www.konkur.in

and dimensions of the Schneiderian membrane and apical bone in maxillary molars referred for apical surgery: a comparative radiographic analysis using limited cone beam computed tomography, J Endod 2012;38: 51.

70. Boskey AL. *Matrix proteins and mineralization: an overview*, Connect Tissue Res 1996;35: 357.
71. Boskey AL. *Biomineralization: an overview*, Connect Tissue Res 2003;44: 5.
72. Boucher Y, Sobel M, Sauveur G. *Persistent pain related to root canal filling and apical fenestration: a case report*, J Endod 2000;26: 242.
73. Bowers GM, Schallhorn RG, McClain PK, et al: *Factors influencing the outcome of regenerative therapy in mandibular Class II furcations: part I*, J Periodontol 2003;74: 1255.
74. Boyes-Varley JG, Cleaton-Jones PE, Lownie JF. *Effect of a topical drug combination on the early healing of extraction sockets in the vervet monkey*, Int J Oral Maxillofac Surg 1988;17: 138.
75. Boykin MJ, Gilbert GH, Tilashalski KR, et al: *Incidence of endodontic treatment: a 48-month prospective study*, J Endod 2003;29: 806.
76. Brent PD, Morgan LA, Marshall JG, et al: *Evaluation of diamond-coated ultrasonic instruments for root-end preparation*, J Endod 1999;25: 672.
77. Briggs PF, Scott BJ. *Evidence-based dentistry: endodontic failure–how should it be managed?*, Br Dent J 1997;183: 159.
78. Britto LR, Katz J, Guelmann M, et al: *Periradicular radiographic assessment in diabetic and control individuals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 449.

Telegram: @dental_k

www.konkur.in

79. Brown AR, Papasian CJ, Shultz P, et al: *Bacteremia and intraoral suture removal: can an antimicrobial rinse help?*, J Am Dent Assoc 1998;129: 1455.
80. Bruce GR, McDonald NJ, Sydiskis RJ. *Cytotoxicity of retrofill materials*, J Endod 1993;19: 288.
81. Brugnami F, Mellonig JT. *Treatment of a large periapical lesion with loss of labial cortical plate using GTR: a case report*, Int J Periodontics Restorative Dent 1999;19: 243.
82. Buckley JA, Ciancio SG, McMullen JA. *Efficacy of epinephrine concentration in local anesthesia during periodontal surgery*, J Periodontol 1984;55: 653.
83. Budnitz DS, Lovegrove MC, Shehab N, et al: *Emergency hospitalizations for adverse drug events in older Americans*, N Engl J Med 2011;365: 2002.
84. Byrne BE, Tibbetts LS. *Conscious sedation and agents for the control of anxiety*, Ciancio SG ADA guide to dental therapeutics ed 3 2003; American Dental Association Chicago 17.
85. Calderwood RG, Hera SS, Davis JR, et al: *A comparison of the healing rate of bone after the production of defects by various rotary instruments*, J Dent Res 1964;43: 207.
86. Calzonetti KJ, Iwanowski T, Komorowski R, et al: *Ultrasonic root end cavity preparation assessed by an in situ impression technique*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 210.
87. Camp MA, Jeansonne BG, Lallier T. *Adhesion of human fibroblasts to root-end-filling materials*, J Endod 2003;29: 602.
88. Campbell JH, Alvarado F, Murray RA. *Anticoagulation and minor oral surgery: should the anticoagulation regimen be altered?*, J Oral Maxillofac Surg 2000;58: 131.
89. Cannon PD, Dharmar VT. *Minor oral surgical procedures*

Telegram: @dental_k

www.konkur.in

in patients on oral anticoagulants: a controlled study, Aust Dent J 2003;48: 115.

90. Caplan DJ, Weintraub JA. *Factors related to loss of root canal filled teeth*, J Public Health Dent 1997;57: 31.
91. Carr G. *Advanced techniques and visual enhancement for endodontic surgery*, End Rep 1992;7: 6.
92. Carr G, Bentkover SK. *Surgical endodontics*, Cohen S Burns RC Pathways of the pulp ed 7 1994; Mosby St. Louis.
93. Carr G, Bentkover SK. *Surgical endodontics*, Cohen S Burns RC Pathways of the pulp ed 7 1998; Mosby St. Louis 608.
94. Carr GB. *Microscopes in endodontics*, J Calif Dent Assoc 1992;20: 55.
95. Carr GB. *Ultrasonic root end preparation*, Dent Clin North Am 1997;41: 541.
96. Chang LK, Whitaker DC. *The impact of herbal medicines on dermatologic surgery*, Dermatol Surg 2001;27: 759.
97. Cheng H, Jiang W, Phillips FM, et al: *Osteogenic activity of the fourteen types of human bone morphogenetic proteins (BMPs)*, J Bone Joint Surg Am 2003;85-A: 1544.
98. Cheung GS. *Endodontic failures: changing the approach*, Int Dent J 1996;46: 131.
99. Cho E, Kopel H, White SN. *Moisture susceptibility of resin-modified glass-ionomer materials [see comment]*, Quintessence Int 1995;26: 351.
100. Chong BS, Ford TR, Kariyawasam SP. *Tissue response to potential root-end filling materials in infected root canals*, Int Endod J 1997;30: 102.
101. Chong BS, Owadally ID, Pitt Ford TR, et al: *Antibacterial activity of potential retrograde root filling materials*, Endod Dent Traumatol 1994;10: 66.

Telegram: @dental_k

www.konkur.in

102. Chong BS, Owadally ID, Pitt Ford TR, et al: *Cytotoxicity of potential retrograde root-filling materials*, Endod Dent Traumatol 1994;10: 129.
103. Chong BS, Pitt Ford TR, Hudson MB. *A prospective clinical study of mineral trioxide aggregate and IRM when used as root-end filling materials in endodontic surgery*, Int Endod J 2003;36: 520.
104. Chong BS, Pitt Ford TR, Kariyawasam SP. *Short-term tissue response to potential root-end filling materials in infected root canals*, Int Endod J 1997;30: 240.
105. Chong BS, Pitt Ford TR, Watson TF. *Light-cured glass ionomer cement as a retrograde root seal*, Int Endod J 1993;26: 218.
106. Chong BS, Pitt Ford TR, Watson TF, et al: *Sealing ability of potential retrograde root filling materials*, Endod Dent Traumatol 1995;11: 264.
107. Ciasca M, Aminoshariae A, Jin G, et al: *A comparison of the cytotoxicity and proinflammatory cytokine production of EndoSequence root repair material and ProRoot mineral trioxide aggregate in human osteoblast cell culture using reverse-transcriptase polymerase chain reaction*, J Endod 2012;38: 486.
108. Clark RA. *Regulation of fibroplasia in cutaneous wound repair*, Am J Med Sci 1993;306: 42.
109. Clark RA, Stone RD, Leung DY, et al: *Role of macrophages in wound healing*, Surg Forum 1976;27: 16.
110. Clokie CM, Moghadam H, Jackson MT, et al: *Closure of critical sized defects with allogenic and alloplastic bone substitutes*, J Craniofac Surg 2002;13: 111.
111. Costich ER, Youngblood PJ, Walden JM. *A study of the effects of high-speed rotary instruments on bone repair in dogs*, Oral Surg Oral Med Oral Pathol 1964;17: 563.

Telegram: @dental_k

www.konkur.in

112. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J Endod 2007;33: 1121.
113. Coulthard P, Worthington H, Esposito M, et al: *Tissue adhesives for closure of surgical incisions*, Cochrane Database Syst Rev 2004; CD004287.
114. Craig KR, Harrison JW. *Wound healing following demineralization of resected root ends in periradicular surgery*, J Endod 1993;19: 339.
115. Creasy JE, Mines P, Sweet M. *Surgical trends among endodontists: the results of a web-based survey*, J Endod 2009;35: 30.
116. Crooks WG, Anderson RW, Powell BJ, et al: *Longitudinal evaluation of the seal of IRM root end fillings*, J Endod 1994;20: 250.
117. Culliford AT, Cunningham JN Jr, Zeff RH, et al: *Sternal and costochondral infections following open-heart surgery: a review of 2,594 cases*, J Thorac Cardiovasc Surg 1976;72: 714.
118. Cutright B, Quillopa N, Schubert W. *An anthropometric analysis of the key foramina for maxillofacial surgery*, J Oral Maxillofac Surg 2003;61: 354.
119. Dahlin C, Gottlow J, Linde A, et al: *Healing of maxillary and mandibular bone defects using a membrane technique: an experimental study in monkeys*, Scand J Plast Reconstr Surg Hand Surg 1990;24: 13.
120. Dahlin C, Linde A, Gottlow J, et al: *Healing of bone defects by guided tissue regeneration*, Plast Reconstr Surg 1988;81: 672.
121. Dahlkemper P, Wolcott JF, Pringle GA, et al: *Periapical central giant cell granuloma: a potential endodontic misdiagnosis [see comment][erratum appears in Oral*

Telegram: @dental_k

www.konkur.in

Surg Oral Med Oral Pathol Oral Radiol Endod 92:2–3, 2001; PMID: 11458236], Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 739.

122. Damas BA, Wheater MA, Bringas JS, et al: *Cytotoxicity comparison of mineral trioxide aggregates and EndoSequence bioceramic root repair materials*, J Endod 2011;37: 372.
123. Danesh-Meyer MJ. *Guided tissue regeneration in the management of severe periodontal-endodontic lesions*, N Z Dent J 1999;95: 7.
124. Danforth RA, Clark DE. *Effective dose from radiation absorbed during a panoramic examination with a new generation machine*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 236.
125. Danielsson K, Evers H, Holmlund A, et al: *Long-acting local anaesthetics in oral surgery: clinical evaluation of bupivacaine and etidocaine for mandibular nerve block*, Int J Oral Maxillofac Surg 1986;15: 119.
126. Danin J, Linder L, Sund ML, et al: *Quantitative radioactive analysis of microleakage of four different retrograde fillings*, Int Endod J 1992;25: 183.
127. Danin J, Linder LE, Lundqvist G, et al: *Outcomes of periradicular surgery in cases with apical pathosis and untreated canals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 227.
128. Danin J, Stromberg T, Forsgren H, et al: *Clinical management of nonhealing periradicular pathosis: surgery versus endodontic retreatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 213.
129. Daoudi MF, Saunders WP. *In vitro evaluation of furcal perforation repair using mineral trioxide aggregate or resin modified glass ionomer cement with and without the*

Telegram: @dental_k

www.konkur.in

use of the operating microscope, J Endod 2002;28: 512.

130. Davis JR, Steinbronn KK, Graham AR, et al: *Effects of Monsel's solution in uterine cervix*, Am J Clin Pathol 1984;82: 332.

130a Dayer MJ, Jones S, Prendergast B, et al: *Incidence of infective endocarditis in England, 2000–13: a secular trend, interrupted time-series analysis*, Lancet 2015;385: 1219.

131. De Deus QD. *Frequency, location, and direction of the lateral, secondary, and accessory canals*, J Endod 1975;1: 361.

132. de Lange J, Putters T, Baas EM, et al: *Ultrasonic root-end preparation in apical surgery: a prospective randomized study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 841.

133. DeGrood ME, Oguntebi BR, Cunningham CJ, et al: *A comparison of tissue reactions to Ketac-Fil and amalgam*, J Endod 1995;21: 65.

134. Del Fabbro M, Ceresoli V, Lolato A, et al: *Effect of platelet concentrate on quality of life after periradicular surgery: a randomized clinical study*, J Endod 2012;38: 733.

135. Del Fabbro M, Taschieri S, Testori T, et al: *Surgical versus non-surgical endodontic re-treatment for periradicular lesions*, Cochrane Database Syst Rev 2007; CD005511.

136. Denio D, Torabinejad M, Bakland LK. *Anatomical relationship of the mandibular canal to its surrounding structures in mature mandibles*, J Endod 1992;18: 161.

137. Desmouliere A, Gabbiani G. *Myofibroblast differentiation during fibrosis*, Exp Nephrol 1995;3: 134.

138. Desmouliere A, Redard M, Darby I, et al: *Apoptosis*

Telegram: @dental_k

www.konkur.in

mediates the decrease in cellularity during the transition between granulation tissue and scar, Am J Pathol 1995;146: 56.

139. Dewhirst FE. *Structure-activity relationships for inhibition of prostaglandin cyclooxygenase by phenolic compounds*, Prostaglandins 1980;20: 209.
140. Diekwisch TG. *The developmental biology of cementum*, J Dev Biol 2001;45: 695.
141. Dietrich T, Zunker P, Dietrich D, et al: *Apicomarginal defects in periradicular surgery: classification and diagnostic aspects*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 233.
142. Dietrich T, Zunker P, Dietrich D, et al: *Periapical and periodontal healing after osseous grafting and guided tissue regeneration treatment of apicomarginal defects in periradicular surgery: results after 12 months*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 474.
143. Dionne RA, Snyder J, Hargreaves KM. *Analgesic efficacy of flurbiprofen in comparison with acetaminophen, acetaminophen plus codeine, and placebo after impacted third molar removal*, J Oral Maxillofac Surg 1994;52: 919.
144. Dionne RA, Wirdzek PR, Fox PC, et al: *Suppression of postoperative pain by the combination of a nonsteroidal anti-inflammatory drug, flurbiprofen, and a long-acting local anesthetic, etidocaine*, J Am Dent Assoc 1984;108: 598.
145. Distler JH, Hirth A, Kurowska-Stolarska M, et al: *Angiogenic and angiostatic factors in the molecular control of angiogenesis*, Q J Nucl Med 2003;47: 149.
146. Dodson T, Halperin L. *Prophylactic antibiotics reduce complications of orthognathic surgery*, Evid Based Dent 2000;2: 66.

Telegram: @dental_k

www.konkur.in

147. Dorn SO, Gartner AH. *Retrograde filling materials: a retrospective success-failure study of amalgam, EBA, and IRM*, J Endod 1990;16: 391.
148. Doroschak AM, Bowles WR, Hargreaves KM. *Evaluation of the combination of flurbiprofen and tramadol for management of endodontic pain*, J Endod 1999;25: 660.
149. Douthitt JC, Gutmann JL, Witherspoon DE. *Histologic assessment of healing after the use of a bioresorbable membrane in the management of buccal bone loss concomitant with periradicular surgery*, J Endod 2001;27: 404.
150. Dragoo MR. *Resin-ionomer and hybrid-ionomer cements: part I. Comparison of three materials for the treatment of subgingival root lesions*, Int J Periodontics Restorative Dent 1996;16: 594.
151. Dragoo MR. *Resin-ionomer and hybrid-ionomer cements: part II, human clinical and histologic wound healing responses in specific periodontal lesions*, Int J Periodontics Restorative Dent 1997;17: 75.
152. Duarte MA, Demarchi AC, Yamashita JC, et al: *pH and calcium ion release of 2 root-end filling materials*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 345.
153. Dugas NN, Lawrence HP, Teplitsky P, et al: *Quality of life and satisfaction outcomes of endodontic treatment*, J Endod 2002;28: 819.
154. Duggins LD, Clay JR, Himel VT, et al: *A combined endodontic retrofill and periodontal guided tissue regeneration technique for the repair of molar endodontic furcation perforations: report of a case*, Quintessence Int 1994;25: 109.

Telegram: @dental_k

www.konkur.in

155. Edlund M, Nair MK, Nair UP. *Detection of vertical root fractures by using cone-beam computed tomography: a clinical study*, J Endod 2011;37: 768.
156. Enqvist B, von Konow L, Bystedt H. *Pre- and perioperative suggestion in maxillofacial surgery: effects on blood loss and recovery*, Int J Clin Exp Hypn 1995;43: 284.
157. Ericson S, Finne K, Persson G. *Results of apicoectomy of maxillary canines, premolars and molars with special reference to oroantral communication as a prognostic factor*, Int J Oral Surg 1974;3: 386.
158. Eriksson A, Albrektsson T, Grane B, et al: *Thermal injury to bone: a vital-microscopic description of heat effects*, Int J Oral Surg 1982;11: 115.
159. Eriksson AR, Albrektsson T. *Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit*, J Prosthet Dent 1983;50: 101.
160. Eriksson AR, Albrektsson T, Albrektsson B. *Heat caused by drilling cortical bone: temperature measured in vivo in patients and animals*, Acta Orthop Scand 1984;55: 629.
161. Eriksson RA, Albrektsson T, Magnusson B. *Assessment of bone viability after heat trauma: a histological, histochemical and vital microscopic study in the rabbit*, Scand J Plast Reconstr Surg 1984;18: 261.
162. Esposito M, Coulthard P, Oliver R, et al: *Antibiotics to prevent complications following dental implant treatment*, Cochrane Database Syst Rev 2003; CD004152.
163. Estrela C, Bueno MR, Leles CR, et al: *Accuracy of cone beam computed tomography and panoramic and periapical radiography for detection of apical periodontitis*, J Endod 2008;34: 273.

Telegram: @dental_k

www.konkur.in

164. Evans BE. *Local hemostatic agents*, N Y J Dent 1977;47: 109.
165. Fayad MI, Ashkenaz PJ, Johnson BR. *Different representations of vertical root fractures detected by cone-beam volumetric tomography: a case series report*, J Endod 2012;38: 1435.
166. Ferreira FB, Ferreira AL, Gomes BP, et al: *Resolution of persistent periapical infection by endodontic surgery*, Int Endod J 2004;37: 61.
167. Fielding AF, Rachiele DP, Frazier G. *Lingual nerve paresthesia following third molar surgery: a retrospective clinical study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84: 345.
168. Finn MD, Schow SR, Schneiderman ED. *Osseous regeneration in the presence of four common hemostatic agents*, J Oral Maxillofac Surg 1992;50: 608.
169. Fister J, Gross BD. *A histologic evaluation of bone response to bur cutting with and without water coolant*, Oral Surg Oral Med Oral Pathol 1980;49: 105.
170. Fitzpatrick EL, Steiman HR. *Scanning electron microscopic evaluation of finishing techniques on IRM and EBA retrofillings*, J Endod 1997;23: 423.
171. Fogel HM, Peikoff MD. *Microleakage of root-end filling materials [erratum appears in J Endod 27:634, 2001]*, J Endod 2001;27: 456.
172. Folke LE, Stallard RE. *Periodontal microcirculation as revealed by plastic microspheres*, J Periodontal Res 1967;2: 53.
173. Ford TR, Torabinejad M, McKendry DJ, et al: *Use of mineral trioxide aggregate for repair of furcal perforations*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 756.

Telegram: @dental_k

www.konkur.in

174. Forte SG, Hauser MJ, Hahn C, et al: *Microleakage of super-EBA with and without finishing as determined by the fluid filtration method*, J Endod 1998;24: 799.
175. Fouad AF, Burleson J. *The effect of diabetes mellitus on endodontic treatment outcome: data from an electronic patient record*, J Am Dent Assoc 2003;134: 43.
176. Frank RJ, Antrim DD, Bakland LK. *Effect of retrograde cavity preparations on root apexes*, Endod Dent Traumatol 1996;12: 100.
177. Freedman A, Horowitz I. *Complications after apicoectomy in maxillary premolar and molar teeth*, Int J Oral Maxillofac Surg 1999;28: 192.
178. Friedman S. *Management of post-treatment endodontic disease: a current concept of case selection*, Aust Endod J 2000;26: 104.
179. Frosch DL, Kaplan RM. *Shared decision making in clinical medicine: past research and future directions*, Am J Prev Med 1999;17: 285.
180. Fyffe HE, Kay EJ. *Assessment of dental health state utilities*, Community Dent Oral Epidemiol 1992;20: 269.
181. Gagliani M, Taschieri S, Molinari R. *Ultrasonic root-end preparation: influence of cutting angle on the apical seal*, J Endod 1998;24: 726.
182. Gajraj NM. *The effect of cyclooxygenase-2 inhibitors on bone healing*, Reg Anesth Pain Med 2003;28: 456.
183. Galan D. *Clinical application of Geristore glass-ionomer restorative in older dentitions*, J Esthet Dent 1991;3: 221.
184. Gallagher CS, Mourino AP. *Root-end induction*, J Am Dent Assoc 1979;98: 578.
185. Garlock JA, Pringle GA, Hicks ML. *The odontogenic keratocyst: a potential endodontic misdiagnosis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 452.

Telegram: @dental_k

www.konkur.in

186. Garrett K, Kerr M, Hartwell G, et al: *The effect of a bioresorbable matrix barrier in endodontic surgery on the rate of periapical healing: an in vivo study*, J Endod 2002;28: 503.
187. Gatti JJ, Dobeck JM, Smith C, et al: *Bacteria of asymptomatic periradicular endodontic lesions identified by DNA-DNA hybridization*, Endod Dent Traumatol 2000;16: 197.
188. Gemalmaz D, Yoruc B, Ozcan M, et al: *Effect of early water contact on solubility of glass ionomer luting cements*, J Prosthet Dent 1998;80: 474.
189. Georgelin-Gurgel M, Diemer F, Nicolas E, et al: *Surgical and nonsurgical endodontic treatment-induced stress*, J Endod 2009;35: 19.
190. Gerhards F, Wagner W. *Sealing ability of five different retrograde filling materials*, J Endod 1996;22: 463.
191. Gerstenfeld LC, Cullinane DM, Barnes GL, et al: *Fracture healing as a post-natal developmental process: molecular, spatial, and temporal aspects of its regulation*, J Cell Biochem 2003;88: 873.
192. Giannoudis PV, MacDonald DA, Matthews SJ, et al: *Nonunion of the femoral diaphysis: the influence of reaming and non-steroidal anti-inflammatory drugs [see comment]*, J Bone Joint Surg Br 2000;82: 655.
193. Gibbard LL, Zarb G. *A 5-year prospective study of implant-supported single-tooth replacements*, J Can Dent Assoc 2002;68: 110.
194. Gibbs SJ. *Effective dose equivalent and effective dose: comparison for common projections in oral and maxillofacial radiology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 538.
195. Gilheany PA, Figdor D, Tyas MJ. *Apical dentin*

Telegram: @dental_k

www.konkur.in

permeability and microleakage associated with root end resection and retrograde filling, J Endod 1994;20: 22.

196. Giray CB, Atasever A, Durgun B, et al: *Clinical and electron microscope comparison of silk sutures and n-butyl-2-cyanoacrylate in human mucosa*, Aust Dent J 1997;42: 255.
197. Glick M, Abel SN, Muzyka BC, et al: *Dental complications after treating patients with AIDS*, J Am Dent Assoc 1994;125: 296.
198. Glowacki J. *Angiogenesis in fracture repair*, Clin Orthop Relat Res 1998;S82:.
199. Goaslind GD, Robertson PB, Mahan CJ, et al: *Thickness of facial gingiva*, J Periodontol 1977;48: 768.
200. Gondim E Jr, Figueiredo Almeida de Gomes BP, Ferraz CC, et al: *Effect of sonic and ultrasonic retrograde cavity preparation on the integrity of root apices of freshly extracted human teeth: scanning electron microscopy analysis*, J Endod 2002;28: 646.
201. Goodchild J, Glick M. *A different approach to medical risk assessment*, Endod Topics 2003;4: 1.
202. Goodman S, Ma T, Trindade M, et al: *COX-2 selective NSAID decreases bone ingrowth in vivo*, J Orthop Res 2002;20: 1164.
203. Goodman SB, Ma T, Genovese M, et al: *COX-2 selective inhibitors and bone*, Int J Immunopathol Pharmacol 2003;16: 201.
204. Gordon SM, Dionne RA, Brahim J, et al: *Blockade of peripheral neuronal barrage reduces postoperative pain*, Pain 1997;70: 209.
205. Gorman MC, Steiman HR, Gartner AH. *Scanning electron microscopic evaluation of root-end preparations*, J Endod 1995;21: 113.

Telegram: @dental_k

www.konkur.in

206. Goyal B, Tewari S, Duhan J, et al: *Comparative evaluation of platelet-rich plasma and guided tissue regeneration membrane in the healing of apicomarginal defects: a clinical study*, J Endod 2011;37: 773.
207. Gray GJ, Hatton JF, Holtzmann DJ, et al: *Quality of root-end preparations using ultrasonic and rotary instrumentation in cadavers*, J Endod 2000;26: 281.
208. Grbic JT, Landesberg R, Lin SQ, et al: *Incidence of osteonecrosis of the jaw in women with postmenopausal osteoporosis in the health outcomes and reduced incidence with zoledronic acid once yearly pivotal fracture trial*, J Am Dent Assoc 2008;139: 32.
209. Greer BD, West LA, Liewehr FR, et al: *Sealing ability of Dyract, Geristore, IRM, and super-EBA as root-end filling materials*, J Endod 2001;27: 441.
210. Grzesik WJ, Narayanan AS. *Cementum and periodontal wound healing and regeneration*, Crit Rev Oral Biol Med 2002;13: 474.
211. Gutmann JL. *Perspectives on root-end resection*, J Hist Dent 1999;47: 135.
212. Gutmann JL, Harrison JW. Surgical endodontics 1991; Blackwell Scientific Publications London 468.
213. Gutmann JL, Harrison JW. Surgical endodontics ed 1 1994; Ishiyaku EuroAmerica St. Louis 468.
214. Gutmann JL, Saunders WP, Nguyen L, et al: *Ultrasonic root-end preparation. Part 1. SEM analysis*, Int Endod J 1994;27: 318.
215. Haasch GC, Gerstein H, Austin BP. *Effects of two hemostatic agents on osseous healing*, J Endod 1989;15: 310.
216. Hall RM. *The effect of high-speed bone cutting without the use of water coolant*, Oral Surg Oral Med Oral Pathol

Telegram: @dental_k

www.konkur.in

1965;20: 150.
217. Hanks CT, Wataha JC, Parsell RR, et al: *Delineation of cytotoxic concentrations of two dentin bonding agents in vitro*, J Endod 1992;18: 589.
218. Happonen RP. *Periapical actinomycosis: a follow-up study of 16 surgically treated cases*, Endod Dent Traumatol 1986;2: 205.
219. Harada S, Rodan GA. *Control of osteoblast function and regulation of bone mass*, Nature 2003;423: 349.
220. Harder AT, An YH. *The mechanisms of the inhibitory effects of nonsteroidal anti-inflammatory drugs on bone healing: a concise review*, J Clin Pharmacol 2003;43: 807.
221. Harrison JW, Johnson SA. *Excisional wound healing following the use of IRM as a root-end filling material*, J Endod 1997;23: 19.
222. Harrison JW, Jurosky KA. *Wound healing in the tissues of the periodontium following periradicular surgery. I. The incisional wound*, J Endod 1991;17: 425.
223. Harrison JW, Jurosky KA. *Wound healing in the tissues of the periodontium following periradicular surgery. 2. The dissectional wound*, J Endod 1991;17: 544.
224. Hart J. *Inflammation. 1: Its role in the healing of acute wounds*, J Wound Care 2002;11: 205.
225. Hauman CH, Chandler NP, Tong DC. *Endodontic implications of the maxillary sinus: a review*, Int Endod J 2002;35: 127.
226. Hayashi M, Kinomoto Y, Takeshige F, et al: *Prognosis of intentional replantation of vertically fractured roots reconstructed with dentin-bonded resin*, J Endod 2004;30: 145.
227. Haynes SR, Lawler PG. *An assessment of the consistency of ASA physical status classification allocation*,

Telegram: @dental_k

www.konkur.in

Anaesthesia 1995;50: 195.
228. Held SA, Kao YH, Wells DW. *Endoscope: an endodontic application*, J Endod 1996;22: 327.
229. Hellstein JW, Adler RA, Edwards B, et al: *Managing the care of patients receiving antiresorptive therapy for prevention and treatment of osteoporosis: executive summary of recommendations from the American Dental Association Council on Scientific Affairs*, J Am Dent Assoc 2011;142: 1243.
230. Hepworth MJ, Friedman S. *Treatment outcome of surgical and non-surgical management of endodontic failures*, J Can Dent Assoc 1997;63: 364.
231. Herman WW, Konzelman JL Jr, Prisant LM. *New national guidelines on hypertension: a summary for dentistry*, J Am Dent Assoc 2004;135: 576.
232. Higa RK, Torabinejad M, McKendry DJ, et al: *The effect of storage time on the degree of dye leakage of root-end filling materials*, Int Endod J 1994;27: 252.
233. Hirsch JM, Ahlstrom U, Henrikson PA, et al: *Periapical surgery*, Int J Oral Surg 1979;8: 173.
234. Hlava GL, Reinhardt RA, Kalkwarf KL. *Etidocaine HCl local anesthetic for periodontal flap surgery*, J Periodontol 1984;55: 364.
235. Ho ML, Chang JK, Chuang LY, et al: *Effects of nonsteroidal anti-inflammatory drugs and prostaglandins on osteoblastic functions*, Biochem Pharmacol 1999;58: 983.
236. Holland R, de Souza V, Nery MJ, et al: *Reaction of dogs' teeth to root canal filling with mineral trioxide aggregate or a glass ionomer sealer*, J Endod 1999;25: 728.
237. Holland R, de Souza V, Nery MJ, et al: *Reaction of rat connective tissue to implanted dentin tubes filled with*

Telegram: @dental_k

www.konkur.in

mineral trioxide aggregate or calcium hydroxide, J Endod 1999;25: 161.

238. Holland R, Souza V, Nery MJ, et al: *Reaction of rat connective tissue to implanted dentin tubes filled with a white mineral trioxide aggregate*, Braz Dent J 2002;13: 23.
239. Hollinger J. *Factors for osseous repair and delivery: part II*, J Craniofac Surg 1993;4: 135.
240. Hollinger J, Wong ME. *The integrated processes of hard tissue regeneration with special emphasis on fracture healing*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 594.
241. Huggins CB, Urist MR. *Dentin matrix transformation: rapid induction of alkaline phosphatase and cartilage*, Science 1970;167: 896.
242. Hull TE, Robertson PB, Steiner JC, et al: *Patterns of endodontic care for a Washington state population*, J Endod 2003;29: 553.
243. Hume WR. *An analysis of the release and the diffusion through dentin of eugenol from zinc oxide-eugenol mixtures*, J Dent Res 1984;63: 881.
244. Hume WR. *Effect of eugenol on respiration and division in human pulp, mouse fibroblasts, and liver cells in vitro*, J Dent Res 1984;63: 1262.
245. Hume WR. *In vitro studies on the local pharmacodynamics, pharmacology and toxicology of eugenol and zinc oxide-eugenol*, Int Endod J 1988;21: 130.
246. Hunt LM, Benoit PW. *Evaluation of a microcrystalline collagen preparation in extraction wounds*, J Oral Surg 1976;34: 407.
247. Hunt TK, Hopf H, Hussain Z. *Physiology of wound healing*, Adv Skin Wound Care 2000;13: 6.
248. Hunt TK, Knighton DR, Thakral KK, et al: *Studies on*

Telegram: @dental_k

www.konkur.in

inflammation and wound healing: angiogenesis and collagen synthesis stimulated in vivo by resident and activated wound macrophages, Surgery 1984;96: 48.

249. Huumonen S, Kvist T, Grondahl K, et al: *Diagnostic value of computed tomography in re-treatment of root fillings in maxillary molars*, Int Endod J 2006;39: 827.
250. Huynh MP, Yagiela JA. *Current concepts in acute pain management*, J Calif Dent Assoc 2003;31: 419.
251. Iasella JM, Greenwell H, Miller RL, et al: *Ridge preservation with freeze-dried bone allograft and a collagen membrane compared to extraction alone for implant site development: a clinical and histologic study in humans*, J Periodontol 2003;74: 990.
252. Inoue S, Yoshimura M, Tinkle JS, et al: *A 24-week study of the microleakage of four retrofilling materials using a fluid filtration method*, J Endod 1991;17: 369.
253. Ishikawa H, Sawada N, Kobayashi C, et al: *Evaluation of root-end cavity preparation using ultrasonic retrotips*, Int Endod J 2003;36: 586.
254. Iwu C, MacFarlane TW, MacKenzie D, et al: *The microbiology of periapical granulomas*, Oral Surg Oral Med Oral Pathol 1990;69: 502.
255. Jafri SM. *Periprocedural thromboprophylaxis in patients receiving chronic anticoagulation therapy*, Am Heart J 2004;147: 3.
256. Jeansonne BG, Boggs WS, Lemon RR. *Ferric sulfate hemostasis: effect on osseous wound healing. II. With curettage and irrigation*, J Endod 1993;19: 174.
257. Jeffcoat MK, Reddy MS, Wang IC, et al: *The effect of systemic flurbiprofen on bone supporting dental implants*, J Am Dent Assoc 1995;126: 305.
258. Jensen JA, Hunt TK, Scheuenstuhl H, et al: *Effect of*

Telegram: @dental_k

www.konkur.in

lactate, pyruvate, and pH on secretion of angiogenesis and mitogenesis factors by macrophages, Lab Invest 1986;54: 574.

259. Jerome CE, Hill AV. *Preventing root tip loss in the maxillary sinus during endodontic surgery*, J Endod 1995;21: 422.
260. Jeske AH, Suchko GD. *Lack of a scientific basis for routine discontinuation of oral anticoagulation therapy before dental treatment*, J Am Dent Assoc 2003;134: 1492.
261. Jesslen P, Zetterqvist L, Heimdahl A. *Long-term results of amalgam versus glass ionomer cement as apical sealant after apicectomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 101.
262. Johnson BR, Schwartz A, Goldberg J, et al: *A chairside aid for shared decision making in dentistry: a randomized controlled trial*, J Dent Educ 2006;70: 133.
263. Johnson P, Fromm D. *Effects of bone wax on bacterial clearance*, Surgery 1981;89: 206.
264. Kadohiro G. *A comparative study of the sealing quality of zinc-free amalgam and Diaket when used as a retrograde filling material*, Hawaii Dent J 1984;15: 8.
265. Kalbermatten DF, Kalbermatten NT, Hertel R. *Cotton-induced pseudotumor of the femur*, Skeletal Radiol 2001;30: 415.
266. Kassab MM, Radmer TW, Glore JW, et al: *A retrospective review of clinical international normalized ratio results and their implications*, J Am Dent Assoc 2011;142: 1252.
267. Kawaguchi H, Pilbeam CC, Harrison JR, et al: *The role of prostaglandins in the regulation of bone metabolism*, Clin Orthop Relat Res 1995;313: 36.

Telegram: @dental_k

www.konkur.in

268. Kawai K, Masaka N. *Vertical root fracture treated by bonding fragments and rotational replantation*, Dent Traumatol 2002;18: 42.
269. Kay EJ, Nuttall NM, Knill-Jones R. *Restorative treatment thresholds and agreement in treatment decision-making*, Community Dent Oral Epidemiol 1992;20: 265.
270. Kay WW, Kurylo E, Chong G, et al: *Inhibition and enhancement of platelet aggregation by collagen derivatives*, J Biomed Mater Res 1977;11: 365.
271. Kay WW, Swanson R, Chong G, et al: *Binding of collagen by canine blood platelets*, Thromb Haemost 1977;37: 309.
272. Keller J. *Effects of indomethacin and local prostaglandin E2 on fracture healing in rabbits*, Dan Med Bull 1996;43: 317.
273. Kellert M, Chalfin H, Solomon C. *Guided tissue regeneration: an adjunct to endodontic surgery*, J Am Dent Assoc 1994;125: 1229.
274. Kerawala CJ, Martin IC, Allan W, et al: *The effects of operator technique and bur design on temperature during osseous preparation for osteosynthesis self-tapping screws*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88: 145.
275. Kersten HW, Wesselink PR, Thoden van Velzen SK. *The diagnostic reliability of the buccal radiograph after root canal filling*, Int Endod J 1987;20: 20.
276. Kettering JD, Torabinejad M. *Cytotoxicity of root canal sealers: a study using HeLa cells and fibroblasts*, Int Endod J 1984;17: 60.
277. Kettering JD, Torabinejad M. *Investigation of mutagenicity of mineral trioxide aggregate and other commonly used root-end filling materials*, J Endod

Telegram: @dental_k

www.konkur.in

1995;21: 537.
278. Khamaisi M, Regev E, Yarom N, et al: *Possible association between diabetes and bisphosphonate-related jaw osteonecrosis*, J Clin Endocrinol Metab 2007;92: 1172.
279. Kim E, Song JS, Jung IY, et al: *Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal-endodontic origin*, J Endod 2008;34: 546.
280. Kim S. *Principles of endodontic microsurgery*, Dent Clin North Am 1997;41: 481.
281. Kim S. *Endodontic microsurgery*, Cohen S Burns RC Pathways of the pulp ed 8 2002; Mosby St. Louis.
282. Kim S, Kratchman S. *Modern endodontic surgery concepts and practice: a review*, J Endod 2006;32: 601.
283. Kim S, Rethnam S. *Hemostasis in endodontic microsurgery*, Dent Clin North Am 1997;41: 499.
284. Kim SG, Solomon C. *Cost-effectiveness of endodontic molar retreatment compared with fixed partial dentures and single-tooth implant alternatives*, J Endod 2011;37: 321.
285. Kim TS, Caruso JM, Christensen H, et al: *A comparison of cone-beam computed tomography and direct measurement in the examination of the mandibular canal and adjacent structures*, J Endod 2010;36: 1191.
286. Kirsner RS, Eaglstein WH. *The wound healing process*, Dermatol Clin 1993;11: 629.
287. Kiryu T, Hoshino E, Iwaku M. *Bacteria invading periapical cementum*, J Endod 1994;20: 169.
288. Kleier DJ. *The continuous locking suture technique*, J Endod 2001;27: 624.

Telegram: @dental_k

www.konkur.in

289. Knighton DR, Hunt TK, Scheuenstuhl H, et al: *Oxygen tension regulates the expression of angiogenesis factor by macrophages*, Science 1983;221: 1283.
290. Kocher T, Plagmann HC. *Heat propagation in dentin during instrumentation with different sonic scaler tips*, Quintessence Int 1996;27: 259.
291. Kretzschmar D. *In reply*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 3.
292. Kuc I, Peters E, Pan J. *Comparison of clinical and histologic diagnoses in periapical lesions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 333.
293. Kvist T, Reit C. *Results of endodontic retreatment: a randomized clinical study comparing surgical and onsurgical procedures*, J Endod 1999;25: 814.
294. Kvist T, Reit C. *Postoperative discomfort associated with surgical and nonsurgical endodontic retreatment*, Endod Dent Traumatol 2000;16: 71.
295. Lalonde ER, Luebke RG. *The frequency and distribution of periapical cysts and granulomas: an evaluation of 800 specimens*, Oral Surg Oral Med Oral Pathol 1968;25: 861.
296. Lang NP, Loe H. *The relationship between the width of keratinized gingiva and gingival health*, J Periodontol 1972;43: 623.
297. Langeland K, Block RM, Grossman LI. *A histopathologic and histobacteriologic study of 35 periapical endodontic surgical specimens*, J Endod 1977;3: 8.
298. Larson PO. *Topical hemostatic agents for dermatologic surgery [see comment]*, J Dermatol Surg Oncol 1988;14: 623.
299. Layton CA, Marshall JG, Morgan LA, et al: *Evaluation of cracks associated with ultrasonic root-end preparation*, J Endod 1996;22: 157.

Telegram: @dental_k

www.konkur.in

300. Lee SJ, Monsef M, Torabinejad M. *Sealing ability of a mineral trioxide aggregate for repair of lateral root perforations*, J Endod 1993;19: 541.
301. Lemon RR, Steele PJ, Jeansonne BG. *Ferric sulfate hemostasis: effect on osseous wound healing. Left in situ for maximum exposure*, J Endod 1993;19: 170.
302. Leonhardt A, Grondahl K, Bergstrom C, et al: *Long-term follow-up of osseointegrated titanium implants using clinical, radiographic and microbiological parameters*, Clin Oral Implants Res 2002;13: 127.
303. Lin CP, Chou HG, Chen RS, et al: *Root deformation during root-end preparation*, J Endod 1999;25: 668.
304. Lin CP, Chou HG, Kuo JC, et al: *The quality of ultrasonic root-end preparation: a quantitative study*, J Endod 1998;24: 666.
305. Lin L, Skribner J, Shovlin F, et al: *Periapical surgery of mandibular posterior teeth: anatomical and surgical considerations*, J Endod 1983;9: 496.
306. Lin LM, Gaengler P, Langeland K. *Periradicular curettage*, Int Endod J 1996;29: 220.
307. Lin LM, Pascon EA, Skribner J, et al: *Clinical, radiographic, and histologic study of endodontic treatment failures*, Oral Surg Oral Med Oral Pathol 1991;71: 603.
308. Lin S, Cohenca N, Muska EA, et al: *Ridge preservation in cases requiring tooth extraction during endodontic surgery: a case report*, Int Endod J 2008;41: 448.
309. Lindh T, Gunne J, Tillberg A, et al: *A meta-analysis of implants in partial edentulism*, Clin Oral Implants Res 1998;9: 80.
310. Lindskog S, Blomlof L, Hammarstrom L. *Repair of periodontal tissues in vivo and in vitro*, J Clin Periodontol 1983;10: 188.

Telegram: @dental_k

www.konkur.in

311. Little JW, Falace DA, Miller CS, et al: Dental management of the medically compromised patient ed 6 2002; Mosby St. Louis.
312. Littner MM, Kaffe I, Tamse A, et al: *Relationship between the apices of the lower molars and mandibular canal: a radiographic study*, Oral Surg Oral Med Oral Pathol 1986;62: 595.
313. Lloyd A, Gutmann J, Dummer P, et al: *Microleakage of Diaket and amalgam in root-end cavities prepared using MicroMega sonic retro-prep tips*, Int Endod J 1997;30: 196.
314. Lofthag-Hansen S, Huumonen S, Grondahl K, et al: *Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 114.
315. Loftus D: Assessment of MTA, White MTA, Diaket, and Geristore when used as surgical root-end fillings in dogs. In *Endodontics 1*, Dallas, 2003, Baylor College of Dentistry, the Texas A&M University System Health Science Center.
316. Lorena D, Uchio K, Costa AM, et al: *Normal scarring: importance of myofibroblasts*, Wound Repair Regen 2002;10: 86.
317. Lovato KF, Sedgley CM. *Antibacterial activity of endosequence root repair material and proroot MTA against clinical isolates of Enterococcus faecalis*, J Endod 2011;37: 1542.
318. Low KM, Dula K, Burgin W, et al: *Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery*, J Endod 2008;34: 557.
319. Lowenguth RA, Blieden TM. *Periodontal regeneration:*

Telegram: @dental_k

www.konkur.in

root surface demineralization, Periodontology 2000;1: 54.
320. Lowenguth RA, Polson AM, Caton JG. *Oriented cell and fiber attachment systems in vivo*, J Periodontol 1993;64: 330.
321. Ludlow JB, Davies-Ludlow LE, Brooks SL, et al: *Dosimetry of 3 CBCT devices for oral and maxillofacial radiology: CB Mercuray, NewTom 3G and i-CAT*, Dentomaxillofac Radiol 2006;35: 219.
322. Luebke RG. *Surgical endodontics*, Dent Clin North Am 1974;18: 379.
323. Ma J, Shen Y, Stojicic S, et al: *Biocompatibility of two novel root repair materials*, J Endod 2011;37: 793.
324. Machtei EE, Oettinger-Barak O, Peled M. *Guided tissue regeneration in smokers: effect of aggressive anti-infective therapy in Class II furcation defects*, J Periodontol 2003;74: 579.
325. Macphee TC, Cowley G. Essentials of periodontology and periodontics ed 3 1981; Blackwell Scientific Publications Oxford 273.
326. Maddalone M, Gagliani M. *Periapical endodontic surgery: a 3-year follow-up study*, Int Endod J 2003;36: 193.
327. Madison JG 3rd, Hokett SD. *The effects of different tetracyclines on the dentin root surface of instrumented, periodontally involved human teeth: a comparative scanning electron microscope study*, J Periodontol 1997;68: 739.
328. Maeda H, Hashiguchi I, Nakamuta H, et al: *Histological study of periapical tissue healing in the rat molar after retrofilling with various materials*, J Endod 1999;25: 38.
329. Maher WP, Johnson RL, Hess J, et al: *Biocompatibility of retrograde filling materials in the ferret canine: amalgam*

Telegram: @dental_k

www.konkur.in

and IRM, Oral Surg Oral Med Oral Pathol 1992;73: 738.
330. Mariotti A. *Efficacy of chemical root surface modifiers in the treatment of periodontal disease: a systematic review*, Ann Periodontol 2003;8: 205.
331. Marmulla R, Wortche R, Muhling J, et al: *Geometric accuracy of the NewTom 9000 Cone Beam CT*, Dentomaxillofac Radiol 2005;34: 28.
332. Marx RE. *Pamidronate (Aredia) and zoledronate (Zometa) induced avascular necrosis of the jaws: a growing epidemic*, J Oral Maxillofac Surg 2003;61: 1115.
333. Marx RE, Cillo JE Jr, Ulloa JJ. *Oral bisphosphonate-induced osteonecrosis: risk factors, prediction of risk using serum CTX testing, prevention, and treatment*, J Oral Maxillofac Surg 2007;65: 2397.
334. Marx RE, Sawatari Y, Fortin M, et al: *Bisphosphonate-induced exposed bone (osteonecrosis/osteopetrosis) of the jaws: risk factors, recognition, prevention, and treatment*, J Oral Maxillofac Surg 2005;63: 1567.
335. Mason RG, Read MS. *Some effects of a microcrystalline collagen preparation on blood*, Haemostasis 1974;3: 31.
336. Mason RG, Read MS. *Effects of collagen and artificial surfaces on platelets that influence blood coagulation*, Thromb Res 1975;7: 471.
337. Mastromihalis N, Goldstein S, Greenberg M, et al: *Applications for guided bone regeneration in endodontic surgery*, N Y State Dent J 1999;65: 30.
338. Matthews LS, Hirsch C. *Temperatures measured in human cortical bone when drilling*, J Bone Joint Surg Am 1972;54: 297.
339. Mauger MJ, Schindler WG, Walker WA 3rd. *An evaluation of canal morphology at different levels of root resection in mandibular incisors*, J Endod 1998;24: 607.

Telegram: @dental_k

www.konkur.in

340. Mavrokokki T, Cheng A, Stein B, et al: *Nature and frequency of bisphosphonate-associated osteonecrosis of the jaws in Australia*, J Oral Maxillofac Surg 2007;65: 415.
341. Mazeland GR. *Longitudinal aspects of gingival width*, J Periodontal Res 1980;15: 429.
342. McAllister BS, Haghighat K. *Bone augmentation techniques*, J Periodontol 2007;78: 377.
343. McCaul LK, McHugh S, Saunders WP. *The influence of specialty training and experience on decision making in endodontic diagnosis and treatment planning*, Int Endod J 2001;34: 594.
344. McDonald NJ, Dumsha TC. *A comparative retrofill leakage study utilizing a dentin bonding material*, J Endod 1987;13: 224.
345. McDonald NJ, Dumsha TC. *Evaluation of the retrograde apical seal using dentine bonding materials*, Int Endod J 1990;23: 156.
346. McQuay H, Moore R. An evidence-based resource for pain relief 1998; Oxford University Press Oxford.
347. Mehlhaff DS, Marshall JG, Baumgartner JC. *Comparison of ultrasonic and high-speed-bur root-end preparations using bilaterally matched teeth*, J Endod 1997;23: 448.
348. Mehlisch DR. *The efficacy of combination analgesic therapy in relieving dental pain*, J Am Dent Assoc 2002;133: 861.
349. Meryon SD, Riches DW. *A comparison of the in vitro cytotoxicity of four restorative materials assessed by changes in enzyme levels in two cell types*, J Biomed Mater Res 1982;16: 519.
350. Michaelides PL. *Use of the operating microscope in dentistry [erratum appears in J Calif Dent Assoc 24:9,*

Telegram: @dental_k

www.konkur.in

1996], J Calif Dent Assoc 1996;24: 45.
351. Migliorati CA. *Bisphosphonates and oral cavity avascular bone necrosis*, J Clin Oncol 2003;21: 4253.
352. Migliorati CA, Woo SB, Hewson I, et al: *A systematic review of bisphosphonate osteonecrosis (BON) in cancer*, Support Care Cancer 2010;18: 1099.
353. Miles DA, Anderson RW, Pashley DH. *Evaluation of the bond strength of dentin bonding agents used to seal resected root apices*, J Endod 1994;20: 538.
354. Min MM, Brown CE Jr, Legan JJ, et al: *In vitro evaluation of effects of ultrasonic root-end preparation on resected root surfaces*, J Endod 1997;23: 624.
355. Moiseiwitsch JR. *Avoiding the mental foramen during periapical surgery*, J Endod 1995;21: 340.
356. Moiseiwitsch JR. *Position of the mental foramen in a North American, white population*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 457.
357. Morgan LA, Marshall JG. *The topography of root ends resected with fissure burs and refined with two types of finishing burs*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 585.
358. Morgan LA, Marshall JG. *A scanning electron microscopic study of in vivo ultrasonic root-end preparations*, J Endod 1999;25: 567.
359. Mortensen H, Winther JE, Birn H. *Periapical granulomas and cysts: an investigation of 1,600 cases*, Scand J Dent Res 1970;78: 241.
360. Moss RW. *Histopathologic reaction of bone to surgical cutting*, Oral Surg Oral Med Oral Pathol 1964;17: 405.
361. Munksgaard EC, Rud J, Asmussen E. *[Retrograde root obturations employing composite and a dentin bonding agent: adaptions of the filling materials and bond*

Telegram: @dental_k

www.konkur.in

strength], Tandlaegebladet 1989;93: 157.

362. Murray PE, Hafez AA, Windsor LJ, et al: *Comparison of pulp responses following restoration of exposed and non-exposed cavities*, J Dent 2002;30: 213.
363. Nair PN. *Apical periodontitis: a dynamic encounter between root canal infection and host response*, Periodontology 2000 1997;13: 121.
364. Nair PN. *New perspectives on radicular cysts: do they heal?*, Int Endod J 1998;31: 155.
365. Nair PN. *Cholesterol as an aetiological agent in endodontic failures: a review*, Aust Endod J 1999;25: 19.
366. Nair PN, Pajarola G, Luder HU. *Ciliated epithelium-lined radicular cysts*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 485.
367. Nair PN, Sjogren U, Figdor D, et al: *Persistent periapical radiolucencies of root-filled human teeth, failed endodontic treatments, and periapical scars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 617.
368. Nair PN, Sjogren U, Krey G, et al: *Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study*, J Endod 1990;16: 580.
369. Nair PN, Sjogren U, Krey G, et al: *Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth*, J Endod 1990;16: 589.
370. Nair PN, Sjogren U, Sundqvist G. *Cholesterol crystals as an etiological factor in non-resolving chronic inflammation: an experimental study in guinea pigs*, Eur J Oral Sci 1998;106: 644.
371. Nakata K, Naitoh M, Izumi M, et al: *Effectiveness of dental computed tomography in diagnostic imaging of*

Telegram: @dental_k

www.konkur.in

periradicular lesion of each root of a multirooted tooth: a case report, J Endod 2006;32: 583.

372. Nakazawa Y, Mitsui K, Hirai Y, et al: *Histo-pathological study of a glass-ionomer/resin (Geristore) restoration system*, Bull Tokyo Dent Coll 1994;35: 197.
373. Nappi JF, Lehman JA Jr. *The effects of surgicel on bone formation*, Cleft Palate J 1980;17: 291.
374. Nash KD, Brown LJ, Hicks ML. *Private practicing endodontists: production of endodontic services and implications for workforce policy*, J Endod 2002;28: 699.
375. Navarre SW, Steiman HR. *Root-end fracture during retropreparation: a comparison between zirconium nitride-coated and stainless steel microsurgical ultrasonic instruments*, J Endod 2002;28: 330.
376. Neaverth EJ. *Disabling complications following inadvertent overextension of a root canal filling material*, J Endod 1989;15: 135.
377. Nedderman TA, Hartwell GR, Protell FR. *A comparison of root surfaces following apical root resection with various burs: scanning electron microscopic evaluation*, J Endod 1988;14: 423.
378. Nelson DR, Buxton TB, Luu QN, et al: *The promotional effect of bone wax on experimental staphylococcus aureus osteomyelitis [see comment]*, J Thorac Cardiovasc Surg 1990;99: 977.
379. Nencka D, Walia H, Austin BP. *Histological evaluation of the biocompatability of Diaket*, J Dent Res 1995;74: 101.
380. Nicoll BK, Peters RJ. *Heat generation during ultrasonic instrumentation of dentin as affected by different irrigation methods*, J Periodontol 1998;69: 884.
381. Nixon KC, Adkins KF, Keys DW. *Histological*

Telegram: @dental_k

www.konkur.in

evaluation of effects produced in alveolar bone following gingival incision with an electrosurgical scalpel, J Periodontol 1975;46: 40.

382. Nobuhara WK, del Rio CE. *Incidence of periradicular pathoses in endodontic treatment failures*, J Endod 1993;19: 315.
383. Nohl FS, Gulabivala K. *Odontogenic keratocyst as periradicular radiolucency in the anterior mandible: two case reports*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 103.
384. Norred CL. *Complementary and alternative medicine use by surgical patients*, AOM J 2002;76: 1013.
385. Norred CL, Brinker F. *Potential coagulation effects of preoperative complementary and alternative medicines*, Altern Ther Health Med 2001;7: 58.
386. O'Connor AM, Bennett C, Stacey D, et al: *Do patient decision aids meet effectiveness criteria of the international patient decision aid standards collaboration? A systematic review and meta-analysis*, Med Decis Making 2007;27: 554.
387. O'Connor AM, Stacey D, Rovner D, et al: *Decision aids for people facing health treatment or screening decisions*, Cochrane Database Syst Rev 2001; CD001431.
388. O'Connor RP, Hutter JW, Roahen JO. *Leakage of amalgam and Super-EBA root-end fillings using two preparation techniques and surgical microscopy*, J Endod 1995;21: 74.
389. O'Neal RB, Alleyn CD. *Suture materials and techniques*, Curr Opin Periodontol 1997;4: 89.
390. Oguntebi BR, Barker BF, Anderson DM, et al: *The effect of indomethacin on experimental dental periapical lesions in rats*, J Endod 1989;15: 117.

Telegram: @dental_k

www.konkur.in

391. Oikarinen KS, Stoltze K, Andreasen JO. *Influence of conventional forceps extraction and extraction with an extrusion instrument on cementoblast loss and external root resorption of replanted monkey incisors*, J Periodontol Res 1996;31: 337.
392. Olson RA, Roberts DL, Osbon DB. *A comparative study of polylactic acid, Gelfoam, and Surgicel in healing extraction sites*, Oral Surg Oral Med Oral Pathol 1982;53: 441.
393. Olsson B, Wennberg A. *Early tissue reaction to endodontic filling materials*, Endod Dent Traumatol 1985;1: 138.
394. Osorio RM, Hefti A, Vertucci FJ, et al: *Cytotoxicity of endodontic materials*, J Endod 1998;24: 91.
395. Owens WD, Felts JA, Spitznagel EL Jr. *ASA physical status classifications: a study of consistency of ratings*, Anesthesiology 1978;49: 239.
396. Oynick J, Oynick T. *A study of a new material for retrograde fillings*, J Endod 1978;4: 203.
397. Pantschev A, Carlsson AP, Andersson L. *Retrograde root filling with EBA cement or amalgam: a comparative clinical study*, Oral Surg Oral Med Oral Pathol 1994;78: 101.
398. Patel S, Dawood A. *The use of cone beam computed tomography in the management of external cervical resorption lesions*, Int Endod J 2007;40: 730.
399. Peciuliene V, Reynaud AH, Balciuniene I, et al: *Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis*, Int Endod J 2001;34: 429.
400. Pecora G, Kim S, Celletti R, et al: *The guided tissue regeneration principle in endodontic surgery: one-year postoperative results of large periapical lesions*, Int Endod

Telegram: @dental_k

www.konkur.in

J 1995;28: 41.

401. Peer M. *Intentional replantation: a "last resort" treatment or a conventional treatment procedure? Nine case reports*, Dent Traumatol 2004;20: 48.
402. Peltola M, Salo T, Oikarinen K. *Toxic effects of various retrograde root filling materials on gingival fibroblasts and rat sarcoma cells*, Endod Dent Traumatol 1992;8: 120.
403. Perez M, Fernandez I, Marquez D, et al: *Use of N-butyl-2-cyanoacrylate in oral surgery: biological and clinical evaluation*, Artif Organs 2000;24: 241.
404. Pertot WJ, Stephan G, Tardieu C, et al: *Comparison of the intraosseous biocompatibility of Dyract and Super EBA*, J Endod 1997;23: 315.
405. Peters CI, Peters OA, Barbakow F. *An in vitro study comparing root-end cavities prepared by diamond-coated and stainless steel ultrasonic retrotips*, Int Endod J 2001;34: 142.
406. Peters E, Monopoli M, Woo SB, et al: *Assessment of the need for treatment of postendodontic asymptomatic periapical radiolucencies in bone marrow transplant recipients*, Oral Surg Oral Med Oral Pathol 1993;76: 45.
407. Peters LB, Harrison JW. *A comparison of leakage of filling materials in demineralized and non-demineralized resected root ends under vacuum and non-vacuum conditions*, Int Endod J 1992;25: 273.
408. Petersen JK, Krogsgaard J, Nielsen KM, et al: *A comparison between 2 absorbable hemostatic agents: gelatin sponge (Spongostan) and oxidized regenerated cellulose (Surgicel)*, Int J Oral Surg 1984;13: 406.
409. Philipsen HP, Srisuwan T, Reichart PA. *Adenomatoid odontogenic tumor mimicking a periapical (radicular)*

Telegram: @dental_k

www.konkur.in

cyst: a case report, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 246.

410. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 1. Size, orientation, and positional relationship to the mandibular second premolar*, J Endod 1990;16: 221.
411. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 2. Radiographic position in relation to the mandibular second premolar*, J Endod 1992;18: 271.
412. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 3. Size and position on panoramic radiographs*, J Endod 1992;18: 383.
413. Phillips SJ. *Physiology of wound healing and surgical wound care*, ASAIO J 2000;46: S2.
414. Pinto VS, Zuolo ML, Mellonig JT. *Guided bone regeneration in the treatment of a large periapical lesion: a case report*, Pract Periodontics Aesthet Dent 1995;7: 76.
415. Pissiotis E, Sapounas G, Spangberg LS. *Silver glass ionomer cement as a retrograde filling material: a study in vitro*, J Endod 1991;17: 225.
416. Pissiotis E, Spangberg L. *Reaction of bony tissue to implanted silver glass ionomer and a reinforced zinc oxide-eugenol cement*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 623.
417. Pitt Ford TR, Andreasen JO, Dorn SO, et al: *Effect of IRM root end fillings on healing after replantation*, J Endod 1994;20: 381.
418. Pitt Ford TR, Andreasen JO, Dorn SO, et al: *Effect of super-EBA as a root end filling on healing after replantation*, J Endod 1995;21: 13.
419. Pitt Ford TR, Andreasen JO, Dorn SO, et al: *Effect of various zinc oxide materials as root-end fillings on healing after replantation*, Int Endod J 1995;28: 273.

Telegram: @dental_k

www.konkur.in

420. Pitt Ford TR, Andreasen JO, Dorn SO, et al: *Effect of various sealers with gutta-percha as root-end fillings on healing after replantation*, Endod Dent Traumatol 1996;12: 33.
421. Pompa DG. *Guided tissue repair of complete buccal dehiscences associated with periapical defects: a clinical retrospective study*, J Am Dent Assoc 1997;128: 989.
422. Posen S. *Alkaline phosphatase*, Ann Intern Med 1967;67: 183.
423. Posen S, Neale FC, Brudenell-Woods J, et al: *Continuous determination of enzyme activity during heat inactivation*, Lancet 1966;1: 264.
424. Rahbaran S, Gilthorpe MS, Harrison SD, et al: *Comparison of clinical outcome of periapical surgery in endodontic and oral surgery units of a teaching dental hospital: a retrospective study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 700.
425. Rainwater A, Jeansonne BG, Sarkar N. *Effects of ultrasonic root-end preparation on microcrack formation and leakage*, J Endod 2000;26: 72.
426. Raisz LG. *Bone cell biology: new approaches and unanswered questions*, J Bone Miner Res 1993;8: S457.
427. Raisz LG, Pilbeam CC, Fall PM. *Prostaglandins: mechanisms of action and regulation of production in bone*, Osteoporos Int 1993;3: 136.
428. Rakich DR, Wataha JC, Lefebvre CA, et al: *Effects of dentin bonding agents on macrophage mitochondrial activity*, J Endod 1998;24: 528.
429. Rakich DR, Wataha JC, Lefebvre CA, et al: *Effect of dentin bonding agents on the secretion of inflammatory mediators from macrophages*, J Endod 1999;25: 114.
430. Rakusin H, Harrison JW, Marker VA. *Alteration of the*

Telegram: @dental_k

www.konkur.in

manipulative properties of plain gut suture material by hydration, J Endod 1988;14: 121.

431. Ramadas Y, Sealey CM. *Third molar removal and nerve injury*, N Z Dent J 2001;97: 25.
432. Rankow HJ, Krasner PR. *Endodontic applications of guided tissue regeneration in endodontic surgery*, Oral Health 1996;86: 33.
433. Regan JD, Gutmann JL, Witherspoon DE. *Comparison of Diaket and MTA when used as root-end filling materials to support regeneration of the periradicular tissues*, Int Endod J 2002;35: 840.
434. Register AA. *Bone and cementum induction by dentin, demineralized in situ*, J Periodontol 1973;44: 49.
435. Register AA. *Induced reattachment in periodontic-endodontic lesions by root demineralization in situ*, Oral Surg Oral Med Oral Pathol 1978;45: 774.
436. Register AA, Burdick FA. *Accelerated reattachment with cementogenesis to dentin, demineralized in situ. I. Optimum range*, J Periodontol 1975;46: 646.
437. Register AA, Burdick FA. *Accelerated reattachment with cementogenesis to dentin, demineralized in situ. II. Defect repair*, J Periodontol 1976;47: 497.
438. Register AA, Scopp IW, Kassouny DY, et al: *Human bone induction by allogeneic dentin matrix*, J Periodontol 1972;43: 459.
439. Reit C, Kvist T. *Endodontic retreatment behaviour: the influence of disease concepts and personal values*, Int Endod J 1998;31: 358.
440. Resillez-Urioste F, Sanandajt K, Davidson RM. *Use of a resin-ionomer in the treatment of mechanical root perforation: report of a case*, Quintessence Int 1998;29: 115.

Telegram: @dental_k

www.konkur.in

441. Reuben HL, Apotheker H. *Apical surgery with the dental microscope*, Oral Surg Oral Med Oral Pathol 1984;57: 433.
442. Rigolone M, Pasqualini D, Bianchi L, et al: *Vestibular surgical access to the palatine root of the superior first molar: "low-dose cone-beam" CT analysis of the pathway and its anatomic variations*, J Endod 2003;29: 773.
443. Robinson RC, Williams CW. *Documentation method for inferior alveolar and lingual nerve paresthesias*, Oral Surg Oral Med Oral Pathol 1986;62: 128.
444. Rosales JI, Vallecillo M, Osorio R, et al: *An in vitro comparison of micro leakage in three glass ionomer cements used as retrograde filling materials*, Int Dent J 1996;46: 15.
445. Rosenberg ES, Cutler SA. *The effect of cigarette smoking on the long-term success of guided tissue regeneration: a preliminary study*, Ann R Australas Coll Dent Surg 1994;12: 89.
446. Roy CO, Jeansonne BG, Gerrets TF. *Effect of an acid environment on leakage of root-end filling materials*, J Endod 2001;27: 7.
447. Rubin MR, Bilezikian JP. *Clinical review 151: The role of parathyroid hormone in the pathogenesis of glucocorticoid-induced osteoporosis: a re-examination of the evidence*, J Clin Endocrinol Metab 2002;87: 4033.
448. Rubinstein RA, Kim S. *Long-term follow-up of cases considered healed one year after apical microsurgery*, J Endod 2002;28: 378.
449. Rud J, Andreasen JO. *A study of failures after endodontic surgery by radiographic, histologic and stereomicroscopic methods*, Int J Oral Surg 1972;1: 311.
450. Rud J, Andreasen JO, Jensen JF. *A multivariate analysis*

Telegram: @dental_k

www.konkur.in

of the influence of various factors upon healing after endodontic surgery, Int J Oral Surg 1972;1: 258.

451. Rud J, Andreasen JO, Rud V. *[Retrograde root filling utilizing resin and a dentin bonding agent: frequency of healing when compared to retrograde amalgam]*, Tandlaegebladet 1989;93: 267.
452. Rud J, Munksgaard EC. *[Retrograde root canal filling using resin and a dentin bonding agent: analysis of failures]*, Tandlaegebladet 1989;93: 343.
453. Rud J, Munksgaard EC, Andreasen JO, et al: *Retrograde root filling with composite and a dentin-bonding agent. 2*, Endod Dent Traumatol 1991;7: 126.
454. Rud J, Munksgaard EC, Andreasen JO, et al: *Retrograde root filling with composite and a dentin-bonding agent. 1*, Endod Dent Traumatol 1991;7: 118.
455. Rud J, Munksgaard EC, Rud V. *[Retrograde root canal filling using resin and a dentin bonding agent: operative procedures]*, Tandlaegebladet 1989;93: 401.
456. Rud J, Rud V. *Surgical endodontics of upper molars: relation to the maxillary sinus and operation in acute state of infection*, J Endod 1998;24: 260.
457. Rud J, Rud V, Munksgaard EC. *[Retrograde root filling utilizing resin and a dentin bonding agent: indication and applications]*, Tandlaegebladet 1989;93: 223.
458. Rud J, Rud V, Munksgaard EC. *Long-term evaluation of retrograde root filling with dentin-bonded resin composite*, J Endod 1996;22: 90.
459. Rud J, Rud V, Munksgaard EC. *Retrograde root filling with dentin-bonded modified resin composite*, J Endod 1996;22: 477.
460. Rud J, Rud V, Munksgaard EC. *Effect of root canal contents on healing of teeth with dentin-bonded resin*

Telegram: @dental_k

www.konkur.in

composite retrograde seal, J Endod 1997;23: 535.

461. Russo G, Corso LD, Biasiolo A, et al: *Simple and safe method to prepare patients with prosthetic heart valves for surgical dental procedures*, Clin Appl Thromb Hemost 2000;6: 90.
462. Sabeti M, Simon JH, Nowzari H, et al: *Cytomegalovirus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns*, J Endod 2003;29: 321.
463. Safavi K, Kazemi R, Watkins D. *Adherence of enamel matrix derivatives on root-end filling materials*, J Endod 1999;25: 710.
464. Sakamoto M, Siqueira JF Jr, Rocas IN, et al: *Molecular analysis of the root canal microbiota associated with endodontic treatment failures*, Oral Microbiol Immunol 2008;23: 275.
465. Sakellariou PL. *Periapical actinomycosis: report of a case and review of the literature*, Endod Dent Traumatol 1996;12: 151.
466. Sammonds JH. Drug evaluations ed 6 1986; American Medical Association Chicago 658.
467. Satchell PG, Gutmann JL, Witherspoon DE. *Apoptosis: an introduction for the endodontist*, Int Endod J 2003;36: 237.
468. Saunders WP. *A prospective clinical study of periradicular surgery using mineral trioxide aggregate as a root-end filling*, J Endod 2008;34: 660.
469. Saunders WP, Saunders EM, Gutmann JL. *Ultrasonic root-end preparation, Part 2. Microleakage of EBA root-end fillings*, Int Endod J 1994;27: 325.
470. Sauveur G, Boccara E, Colon P, et al: *A photoelastimetric analysis of stress induced by root-end resection*, J Endod 1998;24: 740.

Telegram: @dental_k

www.konkur.in

471. Saygin NE, Giannobile WV, Somerman MJ. *Molecular and cell biology of cementum*, Periodontology 2000;24: 73.
472. Scarfe WC, Farman AG, Sukovic P. *Clinical applications of cone-beam computed tomography in dental practice*, J Can Dent Assoc 2006;72: 75.
473. Schamberg M. *The surgical treatment of chronic alveolar abscess*, Dent Cosmos 1906;48: 15.
474. Scheerer SQ, Steiman HR, Cohen J. *A comparative evaluation of three root-end filling materials: an in vitro leakage study using Prevotella nigrescens*, J Endod 2001;27: 40.
475. Scherer W, Dragoo MR. *New subgingival restorative procedures with Geristore resin ionomer*, Pract Periodontics Aesthet Dent 1995;7: 1.
476. Schilephake H. *Bone growth factors in maxillofacial skeletal reconstruction*, Int J Oral Maxillofac Surg 2002;31: 469.
477. Sciubba JJ, Waterhouse JP, Meyer J. *A fine structural comparison of the healing of incisional wounds of mucosa and skin*, J Oral Pathol 1978;7: 214.
478. Scully C, Wolff A. *Oral surgery in patients on anticoagulant therapy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 57.
479. Sedghizadeh PP, Stanley K, Caligiuri M, et al: *Oral bisphosphonate use and the prevalence of osteonecrosis of the jaw: an institutional inquiry*, J Am Dent Assoc 2009;140: 61.
480. Selden HS. *Bone wax as an effective hemostat in periapical surgery*, Oral Surg Oral Med Oral Pathol 1970;29: 262.
481. Seltzer S, Soltanoff W, Bender IB, et al: *Biologic aspects*

Telegram: @dental_k

www.konkur.in

of endodontics. 1. Histological observations of the anatomy and morphology of root apices and surroundings, Oral Surg Oral Med Oral Pathol 1966;22: 375.

482. Setzer FC, Kohli MR, Shah SB, et al: *Outcome of endodontic surgery: a meta-analysis of the literature—Part 2: Comparison of endodontic microsurgical techniques with and without the use of higher magnification*, J Endod 2012;38: 1.
483. Setzer FC, Shah SB, Kohli MR, et al: *Outcome of endodontic surgery: a meta-analysis of the literature–part 1: Comparison of traditional root-end surgery and endodontic microsurgery*, J Endod 2010;36: 1757.
484. Shah PM, Chong BS, Sidhu SK, et al: *Radiopacity of potential root-end filling materials*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 476.
485. Shaw N. *Textured collagen, a hemostatic agent: a pilot study*, Oral Surg Oral Med Oral Pathol 1991;72: 642.
486. Shulman BB, Leung A. *Endoscopic surgery: an alternative technique*, Dent Today 1996;15: 42.
487. Shuman IE. *Repair of a root perforation with a resin-ionomer using an intentional replantation technique*, General Dent 1999;47: ß392.
488. Simon JH. *Incidence of periapical cysts in relation to the root canal*, J Endod 1980;6: 845.
489. Simon JH, Enciso R, Malfaz JM, et al: *Differential diagnosis of large periapical lesions using cone-beam computed tomography measurements and biopsy*, J Endod 2006;32: 833.
490. Siqueira JF Jr, Lopes HP. *Bacteria on the apical root surfaces of untreated teeth with periradicular lesions: a scanning electron microscopy study*, Int Endod J 2001;34: 216.

Telegram: @dental_k

www.konkur.in

491. Siqueira JF Jr, Rocas IN. *Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 85.
492. Siqueira Junior JF Jr. *Aetiology of root canal treatment failure: why well-treated teeth can fail*, Int Endod J 2001;34: 1.
493. Sisk AL, Dionne RA, Wirdzek PR. *Evaluation of etidocaine hydrochloride for local anesthesia and postoperative pain control in oral surgery*, J Oral Maxillofac Surg 1984;42: 84.
494. Sisk AL, Mosley RO, Martin RP. *Comparison of preoperative and postoperative diflunisal for suppression of postoperative pain*, J Oral Maxillofac Surg 1989;47: 464.
495. Sjogren U, Happonen RP, Kahnberg KE, et al: *Survival of Arachnia propionica in periapical tissue*, Int Endod J 1988;21: 277.
496. Sjogren U, Ohlin A, Sundqvist G, et al: *Gutta-percha-stimulated mouse macrophages release factors that activate the bone resorptive system of mouse calvarial bone*, Eur J Oral Sci 1998;106: 872.
497. Sjogren U, Sundqvist G, Nair PN. *Tissue reaction to gutta-percha particles of various sizes when implanted subcutaneously in guinea pigs*, Eur J Oral Sci 1995;103: 313.
498. Skaar DD, O'Connor H, Hodges JS, et al: *Dental procedures and subsequent prosthetic joint infections: findings from the medicare current beneficiary survey*, J Am Dent Assoc 2011;142: 1343.
499. Skoglund A, Persson G. *A follow-up study of apicoectomized teeth with total loss of the buccal bone*

Telegram: @dental_k

www.konkur.in

plate, Oral Surg Oral Med Oral Pathol 1985;59: 78.

499a Sollecito TP, Abt E, Lockhart PB, et al: *The use of prophylactic antibiotics prior to dental procedures in patients with prosthetic joints*, J Am Dent Assoc 2014;146: 11.

500. Song M, Chung W, Lee SJ, et al: *Long-term outcome of the cases classified as successes based on short-term follow-up in endodontic microsurgery*, J Endod 2012;38: 1192.

501. Song M, Kim E. *A prospective randomized controlled study of mineral trioxide aggregate and super ethoxy-benzoic acid as root-end filling materials in endodontic microsurgery*, J Endod 2012;38: 875.

502. Spangberg L. *Biological effects of root canal filling materials. 7. Reaction of bony tissue to implanted root canal filling material in guinea pigs*, Odontologisk Tidskrift 1969;77: 133.

503. Spector JA, Mehrara BJ, Greenwald JA, et al: *Osteoblast expression of vascular endothelial growth factor is modulated by the extracellular microenvironment*, Am J Physiol Cell Physiol 2001;280: C72.

504. Stein MD, Salkin LM, Freedman AL, et al: *Collagen sponge as a topical hemostatic agent in mucogingival surgery*, J Periodontol 1985;56: 35.

505. Subramanian G, Cohen HV, Quek SY. *A model for the pathogenesis of bisphosphonate-associated osteonecrosis of the jaw and teriparatide's potential role in its resolution*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 744.

506. Sugaya T, Kawanami M, Noguchi H, et al: *Periodontal healing after bonding treatment of vertical root fracture*, Dent Traumatol 2001;17: 174.

Telegram: @dental_k

www.konkur.in

507. Sukovic P. *Cone beam computed tomography in craniofacial imaging*, Orthod Craniofac Res suppl 1, 2003;6: 31.
508. Suliman AA, Schulein TM, Boyer DB, et al: *Effects of etching and rinsing times and salivary contamination on etched glass-ionomer cement bonded to resin composites*, Dent Mater 1989;5: 171.
509. Sunde PT, Tronstad L, Eribe ER, et al: *Assessment of periradicular microbiota by DNA-DNA hybridization*, Endod Dent Traumatol 2000;16: 191.
510. Sundqvist G, Figdor D, Persson S, et al: *Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 86.
511. Sundqvist G, Reuterving CO. *Isolation of Actinomyces israelii from periapical lesion*, J Endod 1980;6: 602.
512. Swift EJ Jr, Pawlus MA, Vargas MA, et al: *Depth of cure of resin-modified glass ionomers*, Dent Mater 1995;11: 196.
513. Sykaras N, Opperman LA. *Bone morphogenetic proteins (BMPs): how do they function and what can they offer the clinician?*, J Oral Sci 2003;45: 57.
514. Tai KW, Chang YC. *Cytotoxicity evaluation of perforation repair materials on human periodontal ligament cells in vitro*, J Endod 2000;26: 395.
515. Taschieri S, Corbella S, Tsesis I, et al: *Effect of guided tissue regeneration on the outcome of surgical endodontic treatment of through-and-through lesions: a retrospective study at 4-year follow-up*, Oral Maxillofac Surg 2011;15: 153.
516. Taub DD, Oppenheim JJ. *Chemokines, inflammation and the immune system*, Therapeutic Immunology 1994;1: 229.

Telegram: @dental_k

www.konkur.in

517. Tenenbaum H, Tenenbaum M. *A clinical study of the width of the attached gingiva in the deciduous, transitional and permanent dentitions*, J Clin Periodontol 1986;13: 270.
518. Testori T, Capelli M, Milani S, et al: *Success and failure in periradicular surgery: a longitudinal retrospective analysis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 493.
519. Tetsch P. *Development of raised temperature after osteotomies*, J Maxillofac Oral Surg 1974;2: 141.
520. Thirawat J, Edmunds DH. *Sealing ability of materials used as retrograde root fillings in endodontic surgery*, Int Endod J 1989;22: 295.
521. Thomas S. *Platelet membrane glycoproteins in haemostasis*, Clin Lab 2002;48: 247.
522. Thomson TS, Berry JE, Somerman MJ, et al: *Cementoblasts maintain expression of osteocalcin in the presence of mineral trioxide aggregate*, J Endod 2003;29: 407.
523. Tidmarsh BG, Arrowsmith MG. *Dentinal tubules at the root ends of apicected teeth: a scanning electron microscopic study*, Int Endod J 1989;22: 184.
524. Tomasek JJ, Gabbiani G, Hinz B, et al: *Myofibroblasts and mechano-regulation of connective tissue remodeling*, Nat Rev Mol Cell Biol 2002;3: 349.
525. Tonetti MS, Pini-Prato G, Cortellini P. *Effect of cigarette smoking on periodontal healing following GTR in infrabony defects: a preliminary retrospective study*, J Clin Periodontol 1995;22: 229.
526. Tong DC, Rothwell BR. *Antibiotic prophylaxis in dentistry: a review and practice recommendations*, J Am Dent Assoc 2000;131: 366.

Telegram: @dental_k

www.konkur.in

527. Torabinejad M, Higa RK, McKendry DJ, et al: *Dye leakage of four root end filling materials: effects of blood contamination*, J Endod 1994;20: 159.
528. Torabinejad M, Hong CU, Lee SJ, et al: *Investigation of mineral trioxide aggregate for root-end filling in dogs*, J Endod 1995;21: 603.
529. Torabinejad M, Hong CU, McDonald F, et al: *Physical and chemical properties of a new root-end filling material*, J Endod 1995;21: 349.
530. Torabinejad M, Hong CU, Pitt Ford TR, et al: *Tissue reaction to implanted super-EBA and mineral trioxide aggregate in the mandible of guinea pigs: a preliminary report*, J Endod 1995;21: 569.
531. Torabinejad M, Hong CU, Pitt Ford TR, et al: *Cytotoxicity of four root end filling materials*, J Endod 1995;21: 489.
532. Torabinejad M, Pitt Ford TR, McKendry DJ, et al: *Histologic assessment of mineral trioxide aggregate as a root-end filling in monkeys*, J Endod 1997;23: 225.
533. Torabinejad M, Rastegar AF, Kettering JD, et al: *Bacterial leakage of mineral trioxide aggregate as a root-end filling material*, J Endod 1995;21: 109.
534. Torabinejad M, Smith PW, Kettering JD, et al: *Comparative investigation of marginal adaptation of mineral trioxide aggregate and other commonly used root-end filling materials*, J Endod 1995;21: 295.
535. Torabinejad M, Watson TF, Pitt Ford TR. *Sealing ability of a mineral trioxide aggregate when used as a root end filling material*, J Endod 1993;19: 591.
536. Traub EF, Tennen JS. *Permanent pigmentation following the application of iron salts*, JAMA 1936;106: 1711.
537. Trent CS. *Electrocautery versus epinephrine-injection*

Telegram: @dental_k

www.konkur.in

tonsillectomy [see comment], Ear Nose Throat J 1993;72: 520.

538. Trombelli L, Kim CK, Zimmerman GJ, et al: *Retrospective analysis of factors related to clinical outcome of guided tissue regeneration procedures in intrabony defects*, J Clin Periodontol 1997;24: 366.
539. Trombelli L, Scabbia A. *Healing response of gingival recession defects following guided tissue regeneration procedures in smokers and non-smokers*, J Clin Periodontol 1997;24: 529.
540. Tronstad L, Barnett F, Cervone F. *Periapical bacterial plaque in teeth refractory to endodontic treatment*, Endod Dent Traumatol 1990;6: 73.
541. Tronstad L, Kreshtool D, Barnett F. *Microbiological monitoring and results of treatment of extraradicular endodontic infection*, Endod Dent Traumatol 1990;6: 129.
542. Trope M, Lost C, Schmitz HJ, et al: *Healing of apical periodontitis in dogs after apicoectomy and retrofilling with various filling materials*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 221.
543. Trowbridge HO, Emling RC. Inflammation: a review of the process ed 5 1997; Quintessence Books Chicago 1.
544. Tseng CC, Harn WM, Chen YH, et al: *A new approach to the treatment of true-combined endodontic-periodontic lesions by the guided tissue regeneration technique*, J Endod 1996;22: 693.
545. Tsesis I, Fuss Z, Lin S, et al: *Analysis of postoperative symptoms following surgical endodontic treatment*, Quintessence Int 2003;34: 756.
546. Tsesis I, Rosen E, Schwartz-Arad D, et al: *Retrospective evaluation of surgical endodontic treatment: traditional versus modern technique*, J Endod 2006;32: 412.

Telegram: @dental_k

www.konkur.in

547. Tsesis I, Rosen E, Tamse A, et al: *Effect of guided tissue regeneration on the outcome of surgical endodontic treatment: a systematic review and meta-analysis*, J Endod 2011;37: 1039.
548. Tyas MJ. *Clinical evaluation of five adhesive systems*, Am J Dent 1994;7: 77.
549. Uchin RA. *Use of a bioresorbable guided tissue membrane at an adjunct to bony regeneration in cases requiring endodontic surgical intervention*, J Endod 1996;22: 94.
550. Urist MR. *Bone histogenesis and morphogenesis in implants of demineralized enamel and dentin*, J Oral Surg 1971;29: 88.
551. Valmaseda-Castellon E, Berini-Aytes L, Gay-Escoda C. *Inferior alveolar nerve damage after lower third molar surgical extraction: a prospective study of 1117 surgical extractions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 377.
552. Veis A. *Mineral-matrix interactions in bone and dentin*, J Bone Miner Res 1993;8: S493.
553. Veis A, Sfeir C, Wu CB. *Phosphorylation of the proteins of the extracellular matrix of mineralized tissues by casein kinase-like activity*, Crit Rev Oral Biol Med 1997;8: 360.
554. Velvart P. *Papilla base incision: a new approach to recession-free healing of the interdental papilla after endodontic surgery*, Int Endod J 2002;35: 453.
555. Velvart P, Ebner-Zimmermann U, Ebner JP. *Comparison of long-term papilla healing following sulcular full thickness flap and papilla base flap in endodontic surgery*, Int Endod J 2004;37: 687.
556. Velvart P, Hecker H, Tillinger G. *Detection of the apical lesion and the mandibular canal in conventional*

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radiography and computed tomography, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 682.

557. Velvart P, Peters CI. *Soft tissue management in endodontic surgery*, J Endod 2005;31: 4.
558. Vickers FJ, Baumgartner JC, Marshall G. *Hemostatic efficacy and cardiovascular effects of agents used during endodontic surgery*, J Endod 2002;28: 322.
559. Vigil GV, Wayman BE, Dazey SE, et al: *Identification and antibiotic sensitivity of bacteria isolated from periapical lesions*, J Endod 1997;23: 110.
560. Vignaroli PA, Anderson RW, Pashley DH. *Longitudinal evaluation of the microleakage of dentin bonding agents used to seal resected root apices*, J Endod 1995;21: 509.
561. Vishteh A, Thomas I, Imamura T. *Eugenol modulation of the immune response in mice*, Immunopharmacology 1986;12: 187.
562. Viswanathan HL, Berry JE, Foster BL, et al: *Amelogenin: a potential regulator of cementum-associated genes*, J Periodontol 2003;74: 1423.
563. Voigt JP, Goran ML, Flesher RM. *The width of lingual mandibular attached gingiva*, J Periodontol 1978;49: 77.
564. von Arx T. *Failed root canals: the case for apicoectomy (periradicular surgery)*, J Oral Maxillofac Surg 2005;63: 832.
565. von Arx T, Gerber C, Hardt N. *Periradicular surgery of molars: a prospective clinical study with a one-year follow-up*, Int Endod J 2001;34: 520.
566. von Arx T, Jensen SS, Hanni S, et al: *Five-year longitudinal assessment of the prognosis of apical microsurgery*, J Endod 2012;38: 570.
567. Vy C. *Cardiovascular effects and efficacy of hemostatic agent in periradicular surgery*, J Endod 2004;30: 379.

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568. Wada M, Takase T, Nakanuma K, et al: *Clinical study of refractory apical periodontitis treated by apicectomy. Part 1. Root canal morphology of resected apex*, Int Endod J 1998;31: 53.
569. Wahl MJ. *Dental surgery in anticoagulated patients*, Arch Intern Med 1998;158: 1610.
570. Waldorf H, Fewkes J. *Wound healing*, Adv Dermatol 1995;10: 77.
571. Walia HD, Newlin S, Austin BP. *Electrochemical analysis of retrofilling microleakage in extracted human teeth*, J Dent Res 1995;74: 101.
572. Wallace JA. *Transantral endodontic surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 80.
573. Walmsley AD, Lumley PJ, Johnson WT, et al: *Breakage of ultrasonic root-end preparation tips*, J Endod 1996;22: 287.
574. Walsh WR, Morberg P, Yu Y, et al: *Response of a calcium sulfate bone graft substitute in a confined cancellous defect*, Clin Orthop Relat Res 2003;406:.
575. Waltimo T, Kuusinen M, Jarvensivu A, et al: *Examination on Candida spp. in refractory periapical granulomas*, Int Endod J 2003;36: 643.
576. Walton RE. *Iatrogenic maxillary sinus exposure during maxillary posterior root-end surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 3- author reply 3.
577. Wang N, Knight K, Dao T, et al: *Treatment outcome in endodontics—The Toronto study. Phases I and II: apical surgery*, J Endod 2004;30: 751.
578. Watters W 3rd, Rethman MP, Hanson NB, et al: *Prevention of orthopaedic implant infection in patients undergoing dental procedures*, J Am Acad Orthop Surg 2013;21: 180.

Telegram: @dental_k

www.konkur.in

579. Watzek G, Bernhart T, Ulm C. *Complications of sinus perforations and their management in endodontics*, Dent Clin North Am 1997;41: 563.
580. Wayman BE, Murata SM, Almeida RJ, et al: *A bacteriological and histological evaluation of 58 periapical lesions*, J Endod 1992;18: 152.
581. Weinstein RS, Chen JR, Powers CC, et al: *Promotion of osteoclast survival and antagonism of bisphosphonate-induced osteoclast apoptosis by glucocorticoids*, J Clin Investig 2002;109: 1041.
582. Weinstein RS, Jilka RL, Parfitt AM, et al: *Inhibition of osteoblastogenesis and promotion of apoptosis of osteoblasts and osteocytes by glucocorticoids. Potential mechanisms of their deleterious effects on bone*, J Clin Investig 1998;102: 274.
583. Wessel JH, Dodson TB, Zavras AI. *Zoledronate, smoking, and obesity are strong risk factors for osteonecrosis of the jaw: a case-control study*, J Oral Maxillofac Surg 2008;66: 625.
584. Weston GD, Moule AJ, Bartold PM. *A comparison in vitro of fibroblast attachment to resected root-ends*, Int Endod J 1999;32: 444.
585. Weston GD, Moule AJ, Bartold PM. *A scanning electron microscopic evaluation of root surfaces and the gutta-percha interface following root-end resection in vitro*, Int Endod J 1999;32: 450.
586. Wiggins KL, Malkin S. *Drilling of bone*, J Biomech 1976;9: 553.
587. Williams SS, Gutmann JL. *Periradicular healing in response to Diaket root-end filling material with and without tricalcium phosphate*, Int Endod J 1996;29: 84.
588. Wilson W, Taubert KA, Gewitz M, et al: *Prevention of*

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infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group, J Am Dent Assoc 2007;138: 739.

589. Witherspoon DE, Gutmann JL. *Haemostasis in periradicular surgery*, Int Endod J 1996;29: 135.
590. Witherspoon DE, Gutmann JL. *Analysis of the healing response to gutta-percha and Diaket when used as root-end filling materials in periradicular surgery*, Int Endod J 2000;33: 37.
591. Wu MK, Kontakiotis EG, Wesselink PR. *Long-term seal provided by some root-end filling materials*, J Endod 1998;24: 557.
592. Wuchenich G, Meadows D, Torabinejad M. *A comparison between two root end preparation techniques in human cadavers*, J Endod 1994;20: 279.
593. Yaccino JM, Walker WA 3rd, Carnes DL Jr, et al: *Longitudinal microleakage evaluation of Super-EBA as a root-end sealing material*, J Endod 1999;25: 552.
594. Yagiela JA. *Injectable and topical local anesthetics*, Ciancio SG ADA guide to dental therapeutics ed 3 2003; American Dental Association Chicago 1.
595. Yajima A, Otonari-Yamamoto M, Sano T, et al: *Cone-beam CT (CB Throne) applied to dentomaxillofacial region*, Bull Tokyo Dent Coll 2006;47: 133.
596. Yao K, Chien M, Kohara O, et al: *Effect of water isolation and early finishing on hardness of glass ionomer*

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www.konkur.in

cements, J Osaka Dent Univ 1990;24: 141.

597. Yeomans JD, Urist MR. *Bone induction by decalcified dentine implanted into oral, osseous and muscle tissues*, Arch Oral Biol 1967;12: 999.

598. Young MP, Korachi M, Carter DH, et al: *The effects of an immediately pre-surgical chlorhexidine oral rinse on the bacterial contaminants of bone debris collected during dental implant surgery*, Clin Oral Implants Res 2002;13: 20.

599. Yucel EA, Oral O, Olgac V, et al: *Effects of fibrin glue on wound healing in oral cavity*, J Dent 2003;31: 569.

600. Yusuf H. *The significance of the presence of foreign material periapically as a cause of failure of root treatment*, Oral Surg Oral Med Oral Pathol 1982;54: 566.

601. Zaman KU, Sugaya T, Hongo O, et al: *A study of attached and oriented human periodontal ligament cells to periodontally diseased cementum and dentin after demineralizing with neutral and low pH etching solution*, J Periodontol 2000;71: 1094.

602. Zetterqvist L, Hall G, Holmlund A. *Apicectomy: a comparative clinical study of amalgam and glass ionomer cement as apical sealants*, Oral Surg Oral Med Oral Pathol 1991;71: 489.

603. Zhang X, Schwarz EM, Young DA, et al: *Cyclooxygenase-2 regulates mesenchymal cell differentiation into the osteoblast lineage and is critically involved in bone repair [erratum appears in J Clin Invest 110:1211, 2002]*, J Clin Investi 2002;109: 1405.

604. Zhu Q, Haglund R, Safavi KE, et al: *Adhesion of human osteoblasts on root-end filling materials*, J Endod 2000;26: 404.

605. Zhu Q, Safavi KE, Spangberg LS. *Cytotoxic evaluation of*

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root-end filling materials in cultures of human osteoblast-like cells and periodontal ligament cells, J Endod 1999;25: 410.

606. Ziegler CM, Woertche R, Brief J, et al: *Clinical indications for digital volume tomography in oral and maxillofacial surgery*, Dentomaxillofac Radiol 2002;31: 126.
607. Zubery Y, Kozlovsky A. *Two approaches to the treatment of true combined periodontal-endodontal lesions*, J Endod 1993;19: 414.
608. Zubillaga G, Von Hagen S, Simon BI, et al: *Changes in alveolar bone height and width following post-extraction ridge augmentation using a fixed bioabsorbable membrane and demineralized freeze-dried bone osteoinductive graft*, J Periodontol 2003;74: 965.
609. Zuolo ML, Ferreira MO, Gutmann JL. *Prognosis in periradicular surgery: a clinical prospective study*, Int Endod J 2000;33: 91.
610. Zuolo ML, Perin FR, Ferreira MO, et al: *Ultrasonic root-end preparation with smooth and diamond-coated tips*, Endod Dent Traumatol 1999;15: 265.

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*The authors would like to acknowledge the contributions of David Witherspoon for his involvement with previous editions.

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12: Regenerative endodontics

Anibal Diogenes, Stéphane Simon, Alan S. Law

CHAPTER OUTLINE

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Overview of regenerative dentistry

Advancements in tissue engineering are dramatically changing medicine and dentistry. Tissue engineering is an interdisciplinary field that applies the principles of engineering and the life sciences toward restoring, maintaining, or replacing biologic function. It involves the interplay between stem cells, growth factors, and scaffolds (biologic matrices). It has become increasingly clear that the intentional manipulation of these three factors can lead to the regeneration of tissue function that would not otherwise take place if repair had taken place without intervention.[68] This relatively young field was first applied to medicine with many examples of regenerative medicine approaches used in the clinical practice.[175,222,306] Although the inclusion of tissue engineering in dentistry is more recent, it is also fundamentally changing the way clinicians are treating patients while providing a fertile research field that fosters future advancements and therapies.

Most of the history of dentistry is marked by the evolution of dental materials and techniques tailored to the replacement of lost or diseased tissues with inert materials. This prosthetic replacement of missing dental tissues has prevailed in dentistry since the primordial examples of dental treatments in ancient civilizations.[23,86,145,193,320] In contrast, the goal of regenerative dentistry is to induce biologic replacement of dental tissues and their supporting structures. The potential for regenerative dentistry is in large part due to advancements in biologic therapies that apply principles of tissue engineering with the spatial and temporal assembly of stem cells, growth factors, and scaffolds to achieve the functional regeneration of a missing tissue.

The foundations of regenerative endodontics date back to more than a century ago with the first attempts of vital pulp therapy with the goal of

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maintaining the physiologic function of an injured pulp.[71] Phillip Pfaff in 1756 performed the first reported pulp capping procedure. He concluded that the exposed injured pulp had the potential of repair if the tissue was further irritated with a caustic agent or heated instruments to promote the cauterization of the pulp stomps.[98,113] It was not until 1920 when Datwyler introduced zinc eugenol–based cement as a direct pulp capping agent.[125] Next, D.W. Herman introduced calcium hydroxide as a biocompatible agent to be used in both vital and nonvital therapies in 1921. In 1960 Professor Nygaard-Østby evaluated a revascularization method for reestablishing a pulp-dentin complex in permanent teeth with pulpal necrosis (see later).[220,221] Over the past several decades, the scope and clinical application of regenerative dental procedures have continuously advanced to now include guided tissue regeneration (GTR) or guided bone regeneration (GBR) procedures and distraction osteogenesis,[56,114] the application of platelet-rich plasma (PRP) for bone augmentation,[117] Emdogain for regeneration of periodontal tissues and pulp,[7,55] recombinant human bone morphogenic protein (rhBMP) for augmentation of bone,[61] and clinical trials on the use of fibroblast growth factor 2 (FGF-2) for periodontal tissue regeneration.[171] The potential of regenerative procedures in endodontics has been emphasized by elegant studies demonstrating the regeneration of pulp and dentin using scaffold materials and stem cells.[105,142,144,146,147,251] Thus regenerative dental procedures are emerging as a vital, evolving field of dental care, creating a paradigm shift in many dental specialties, including endodontics.[208] This chapter will review the current status of regenerative endodontic procedures (REPs) with an emphasis on biologic principles and the advantages and limitations of currently available clinical procedures.

Overview of regenerative endodontics

The developing dentition is at risk for pulpal necrosis due to trauma, caries, and developmental dental anomalies such as dens evaginatus.[17-19,67,77,243,244,325] It is important to note that full radicular maturation occurs up to 3 years after the eruption of a tooth in the oral cavity,[204] and the loss of pulp vitality during this period arrests further root development, rendering these teeth susceptible to premature tooth loss. This untoward event in young

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patients with mixed dentition can have devastating consequences that include: altered maxillary and mandibular bone development, interferences with phonics, breathing, and mastication, and importantly, there is a severe detrimental psychosocial effect on young patients.[158,287]

Immature teeth diagnosed with pulp necrosis have been traditionally treated with apexification procedures using either long-term calcium hydroxide treatment[65,66] or immediate placement of a mineral trioxide aggregate (MTA) apical plug.[315] Although these treatments often result in the resolution of signs and symptoms of pathosis, they provide little to no benefit for continued root development.[39] The process of root maturation includes root elongation, apical closure, and further root thickening (Fig. 12.1). This process is mediated through epithelial-mesenchymal interactions between the Hertwig root sheath (HERS) and the apical papilla.[317] Throughout this process, the pulp-dentin complex is formed at the expense of the apical papilla as stem cells migrate and differentiate forming odontoblasts and fibroblasts, reducing the apical papilla size as the root matures (see Fig. 12.1). Unfortunately, this elaborate developmental process can be disrupted by untoward events such as trauma and infection. Thus immature teeth treated with these procedures are considered in a state of "arrested development," and no further root growth, normal pulpal nociception, and immune defense should be expected.

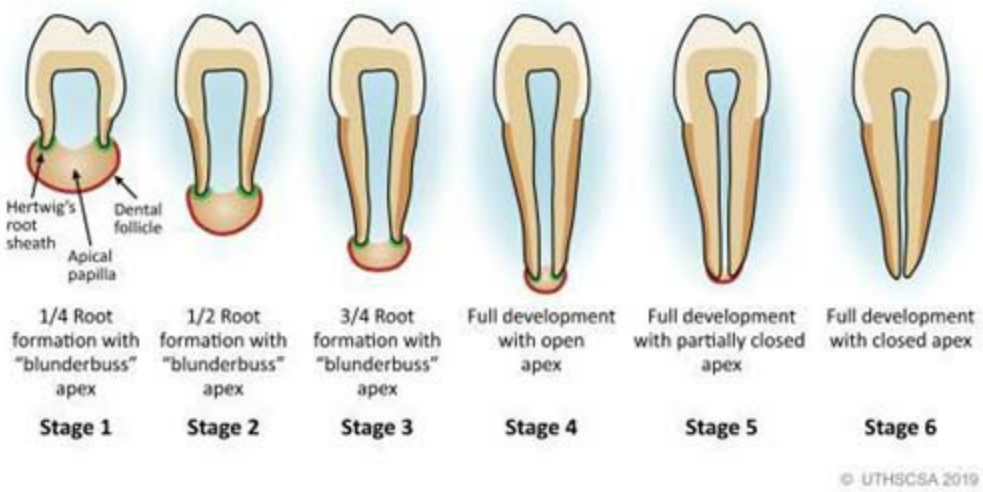

FIG. 12.1 Schematic drawing demonstrating the different stages of root development and the subsequent reduction of the apical papilla

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and Hertwig root sheath.

Lateral cutaway diagram of tooth is labeled from left to right as Hertwig's root sheath, apical papilla and dental follicle. The stages of root development are marked from left to right as:

Stage 1: One-fourth root formation with "blunderbuss" apex.

Stage 2: Half root formation with "blunderbuss" apex.

Stage 3: Three-fourth root formation with "blunderbuss" apex.

Stage 4: full development with open apex.

Stage 5: full development with partially closed apex.

Stage 6: Full development with closed apex.

REPs have been defined as biologically based procedures designed to replace damaged structures such as dentin, root structures, and cells of the pulp-dentin complex.[208] This new treatment modality has emerged as a treatment alternative for these teeth that, in addition to healing of apical periodontitis, aims to promote normal pulpal physiologic functions. These include continued root development, immune competency,[24] and normal nociception, as seen in some published cases.[77] Thus the ultimate goal of these procedures is to regenerate the components and normal function of the pulp-dentin complex to promote tooth longevity and function.

Regenerative endodontics has its foundation on the seminal work by Dr. Nygaard-Østby more than 50 years ago.[226] He hypothesized that a blood clot could be the first step in the healing of a damaged dental pulp, similar to the role of the blood clot in the healing process observed in other areas (e.g., alveolar bone following extraction). To test the hypothesis that the presence of a blood clot within a root canal system promotes healing, mature teeth diagnosed with either vital or necrotic pulp received pulp space débridement followed by foraminal enlargement, medicament dressing for the necrotic cases, evoked intracanal bleeding, and a kloroperka obturation placed coronal to the formed blood clot. Patients (n = 17) were followed for various time periods (17 days to 3.5 years), and then the treated tooth was extracted and the newly formed tissues were histologically examined. The outcomes were similar for all teeth: (1) resolution of symptoms of inflammation related to

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foraminal enlargement and overinstrumentation in as early as 17 days, (2) resolution of signs and symptoms of pathosis for the necrotic cases, and in certain cases, (3) radiographic evidence of apical closure. For the histologic analysis, it was observed that there was ingrowth of connective tissue into the canal space and varied levels of mineralized tissue found along the canal walls as well as "islands" of mineralized tissue embedded within the newly formed tissue (see Fig. 12.1). Because dental pulp is a type of connective tissue with a rich supply of fibroblasts, this general finding was quite promising. However, the inclusion of undesired cell types (e.g., cementoblasts) and the lack of desired cell types (e.g., odontoblasts) indicate that this protocol did not lead to complete histologic regeneration of dental pulp. Despite its shortcomings, this pioneer study laid the foundation for the subsequent studies in the field of regenerative endodontics.

In 1966 a study was published reporting that disinfection could be established primarily with interappointment medication with polyantibiotic mixes (three different formulations used in five cases).[245] The investigators did not purposely evoke intracanal bleeding in this study but instrumented canals short of what they believed to be vital tissue determined by visualization of tissue and pain sensation upon instrumentation. Resolution of signs and symptoms of disease and continued root development was seen for all reported cases. It represented the first reported case where polyantibiotic pastes were used in immature necrotic teeth for disinfection and to promote root development. Five years later, another study was published that included the use of antibiotics in the disinfection protocol and the intentional promotion of intracanal bleeding.[221] Resolution of symptoms and continued root development was a common finding. However, histology of the extracted teeth demonstrated that connective tissue was formed in 28 out of 35 teeth, whereas cellular cementum was formed in 18 out of 35 teeth. Again, these protocols generated acceptable clinical outcomes (e.g., healing of apical periodontitis, lack of symptoms) with only partial evidence of dental pulp phenotype. Collectively, these findings laid the foundation for contemporary regenerative endodontics, demonstrating that repair could take place following root canal disinfection in immature teeth.

The emergence of the field of regenerative endodontics was catalyzed in the early 2000s with the publication of two remarkable case reports.[27,149]

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Since then, there has been an exponential increase in published cases reporting unprecedented clinical outcomes such as resolution of signs and symptoms of apical periodontitis, continued root development, and, in certain cases, normal nociceptive responses to vitality testing.[77,130,173] Among the many published cases, there are two randomized clinical trials[185,211] that, despite limited sample sizes, support the hypothesis that patients with otherwise limited treatment options benefit from these procedures.[297] Importantly, the field of regenerative endodontics has seen a dramatic increase in knowledge gained from translational basic science studies evaluating the interplay of the tissue-engineering components (stem cells, growth factors, and scaffolds) applied to the clinical need and challenges.

Preclinical studies on regenerative endodontics

Applying the principles of tissue engineering to the development of REPs requires research on the correct spatial assembly of distinct stem cells, growth factors/morphogens, and scaffolds to form a functional pulp-dentin complex.[131,142,144,176] In this section, we will review each of these critical components in turn.

Stem cells

Stem cells are defined as a distinct subpopulation of undifferentiated cells with the ability of self-renewal and differentiation into cell lineages of their tissue of origin.[203] They can be classified as pluripotent or multipotent cells. Pluripotent stem cells have the capacity of becoming specialized cells belonging to all three germ layers. Embryonic stem cells are the best example of pluripotent cells. There is a significant body of research on embryonic stem cells, but ethical, legal, and medical (tissue-rejection) issues can render these cell types unsuitable for clinical applications.[208] True pluripotent stem cells can be found only in the developing embryo, and the harvesting of these cells requires destruction of the embryo, hence the legal and ethical concerns with such practice. Yamanka and colleagues reported the groundbreaking finding that somatic cells can be transformed into pluripotent stem cells,

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namely induced pluripotent stem cells (iPSCs).[225] The use of iPSCs does not have the same legal and ethical concerns as the use of embryonic stem cells, but there are still concerns regarding their inherent pluripotent capacity and lack of proliferative control. These cells tend to form teratomas after implantation into a host, a true testament of their high proliferative and differentiation capacities, but making their use in clinical applications challenging, requiring that differentiation into single phenotypically differentiated cells occurs in vitro prior to implantation.[165] On the other hand, all adult mesenchymal stem cells (MSCs) are more restricted in their capacity to differentiate, forming tissues only of mesenchymal origin, and therefore are classified as multipotent.[50] These cells can be found compartmentalized within tissues in "stem cell niches." The mesenchymal tissues (e.g., bone, dental pulp, periodontal ligament) appear to have an enriched population of adult stem cells.[50] These cells were first found in bone marrow decades ago and characterized as self-renewing and plastic-adherent and formed cell colonies with a fibroblastic appearance.[100,101] They were initially called stromal stem cells but later received the now widely accepted terminology "mesenchymal stem cells."[50] It is worth mentioning this terminology is not unanimous, and the characteristics or criteria that define a "stem cell" remain controversial.[51] Nonetheless, most undifferentiated progenitor cells found in the orofacial region are considered MSCs.[83]

Different populations of adult stem cells have been identified in tissue compartments in the oral region. These include stem cells of the apical papilla (SCAPs),[279] inflammatory periapical progenitor cells (iPAPCs),[184] dental follicle stem cells (DFSCs),[205] dental pulp stem cells (DPSCs),[124] periodontal ligament stem cells (PDLSCs),[261] bone marrow stem cells (BMSCs),[100] tooth germ progenitor cells (TGPCs),[285] salivary gland stem cells (SGSCs),[260] stem cells from human exfoliated deciduous teeth (SHEDs),[201] oral epithelial stem cells (OESCs), gingiva-derived mesenchymal stem cells (GMSCs),[156] and periosteal-derived stem cells (PSCs) (Fig. 12.2).[326] Although stem cells have been identified in most oral tissues, the stem cells more likely to be involved in REPs are localized around the periapical region. These include SCAPs, PDLSCs, BMSCs, iPAPCs, and DPSCs (if vital pulp is still present apically).

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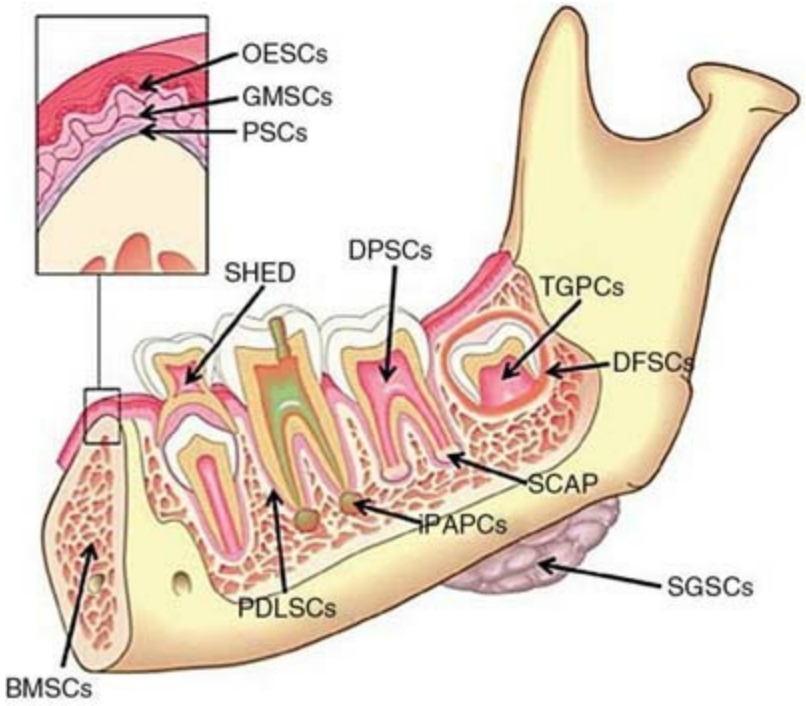

FIG. 12.2 Schematic drawing illustrating potential sources of postnatal stem cells in the oral environment. Cell types include tooth germ progenitor cells *(TGPCs)*, dental follicle stem cells *(DFSCs)*, salivary gland stem cells *(SGSCs)*, stem cells of the apical papilla *(SCAPs)*, dental pulp stem cells *(DPSCs)*, inflamed periapical progenitor cells *(iPAPCs)*, stem cells from human exfoliated deciduous teeth *(SHEDs)*, periodontal ligament stem cells *(PDLSCs)*, bone marrow stem cells *(BMSCs)*, and, as illustrated in the inset, oral epithelial stem cells *(OESCs)*, gingiva-derived mesenchymal stem cells *(GMSCs)*, and periosteal stem cells *(PSCs)*.

Lateral cutaway diagram of postnatal stem cell with layers of oral epithelial stem cells, gingiva-derived mesenchymal stem cells and pluripotent stem cells are marked clockwise as; stem cells from human exfoliated deciduous teeth, dental pulp stem cells, tooth germ progenitor cells, dental follicle stem cells, stem cells from apical papilla, salivary gland stem cells, inflamed periapical progenitor cells, periodontal ligament stem cells and bone marrow-derived mesenchymal stem cells.

Source: (From Hargreaves KM, Diogenes A, Teixeira FB: Treatment options: biological basis of regenerative endodontic procedures, *J Endod* 39:s30–s43, 2013.)

The apical papilla and its residing stem cells (SCAPs) were first characterized in 2006.[279] The apical papilla (Fig. 12.3) is a dense reservoir of undifferentiated MSCs with great proliferative and odontogenic

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differentiation capacity and are directly involved in root formation during development.[140,246,317] Furthermore, the close proximity of the apical papilla to the apices of teeth in continuum with the root canal space makes this rich source of stem cells readily available for regenerative endodontic therapeutics through autologous cell transfer without the necessity to harvest and manipulate these cells exogenously. The iPAPCs represent another important potential source of stem cells for regenerative endodontics in teeth with well-established apical periodontitis.[58,184] These cells appear to be largely localized in the vasculature within apical granulomatous tissues. Lastly, PDLSCs and BMSCs should be also considered as stem cells sources for regenerative procedures because the action of mechanical disruption of the apical tissue (evoked bleeding) could also trigger the release of these cells, albeit their relative abundance is believed to be significantly less than SCAPs and iPAPCs.

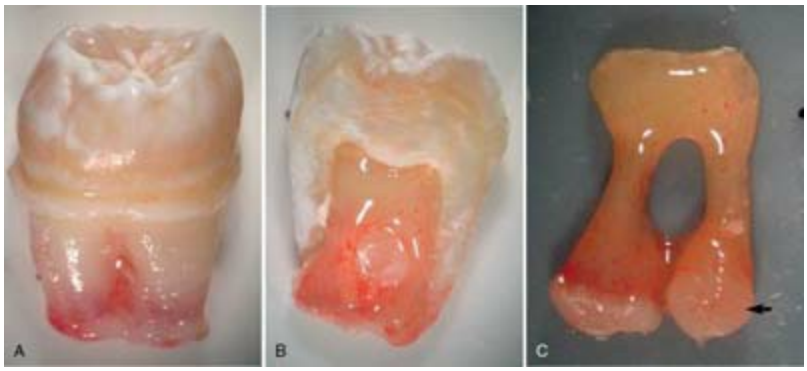

FIG. 12.3 Dissection of an immature permanent tooth indicating the extent of the apical papilla (**A–C**). Note that this structure is likely lacerated during the evoked bleeding step of revascularization cases and thus cells from this structure, including mesenchymal stem cells of the apical papilla (SCAPs), are likely to be delivered into the root canal space. *Arrow* in **C** denotes junction of apical papilla and dental pulp.

A) Lateral close up of a tooth shows crown, neck and roots covered in a sheath of white transopaque membrane.

B) Lateral close up of a tooth shows part of crown and outline of the lower tooth covered in a sheath of white transopaque membrane. A part of root canals is exposed with a circular scar formed between them.

C) Lateral close up of a tooth shows a thinned crown and a hollow neck with enlarged root ends. An arrow pointing to a root end shows the presence of

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apical papilla and dental pulp.

Source: (Courtesy Dr. Michael Henry.)

In 2011 a study was conducted to evaluate the presence of MSCs following the evoked-bleeding step in regenerative procedures.[191] It was found that there is a substantial influx of MSCs into root canals during regenerative procedures resulting in an increase greater than 700-fold in the expression of MSC markers (Fig. 12.4). In addition, the cells could be harvested from clinical samples and examined under confocal microscopy (Fig. 12.5). This was the first demonstration that REPs are stem cell–based therapies.[77] Although this study did not evaluate whether the MSCs detected in REPs are derived from the apical papilla, it was assumed that these cells were SCAPs because the evoked-bleeding step lacerated the apical papilla. However, these MSCs are a heterogeneous population of cells that could come from any of the periradicular tissues after the mechanical step of evoking bleeding into the root canal system.

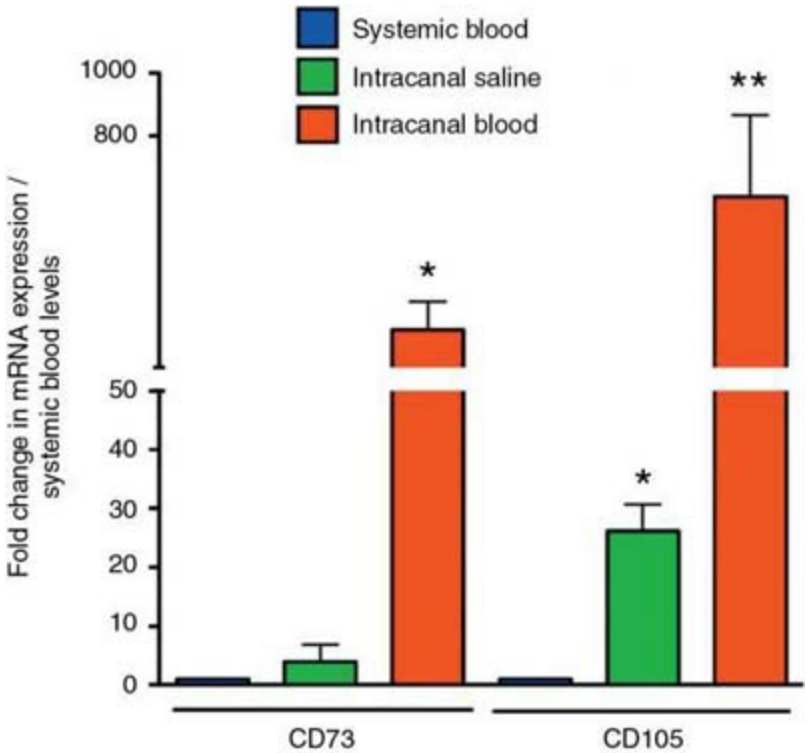

FIG. 12.4 Evoked-bleeding step in endodontic regenerative procedures in immature teeth with open apices leads to significant

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increase in expression of undifferentiated mesenchymal stem cell markers in the root canal space. Systemic blood, saline irrigation, and intracanal blood samples were collected during second visit of regenerative procedures. Real-time RT-PCR was performed by using RNA isolated from each sample as template, with validated specific primers for target genes and 18S ribosomal RNA endogenous control. Expression of mesenchymal stem cell markers CD73 and CD105 was upregulated after the evoked-bleeding step in regenerative procedures. Data were normalized to the housekeeping gene 18S levels and presented as mean ± standard deviation fold increase in relation to systemic blood levels for each gene and analyzed with one-way analysis of variance with Bonferroni post hoc test (n = 8; *P <.05; **P <.01; n.s., not statistically significant).

Plot shows cluster of differentiation 73 and cluster of differentiation 105 against fold change in mRNA expression and systemic blood levels. The horizontal axis has three bars of systematic blood, intracanal saline and intracanal blood under cluster of differentiation 73 and 105 each. The segmented vertical axis ranges from 0 to 50 in increments of 10 and 800 to 1000. The data is as follows:

Cluster of differentiation 73:

Systemic blood: 1, intracanal saline: 4, intracanal blood: 50 (lower vertical axis) : 100 (upper vertical axis).

Cluster of differentiation 105:

Systemic blood: 1, intracanal saline: 25, intracanal blood: 50 (lower vertical axis) : 600 (upper vertical axis).

Source: (From Lovelace TW, Henry MA, Hargreaves KM, Diogenes A: Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure, *J Endod* 37:133–138, 2011.)

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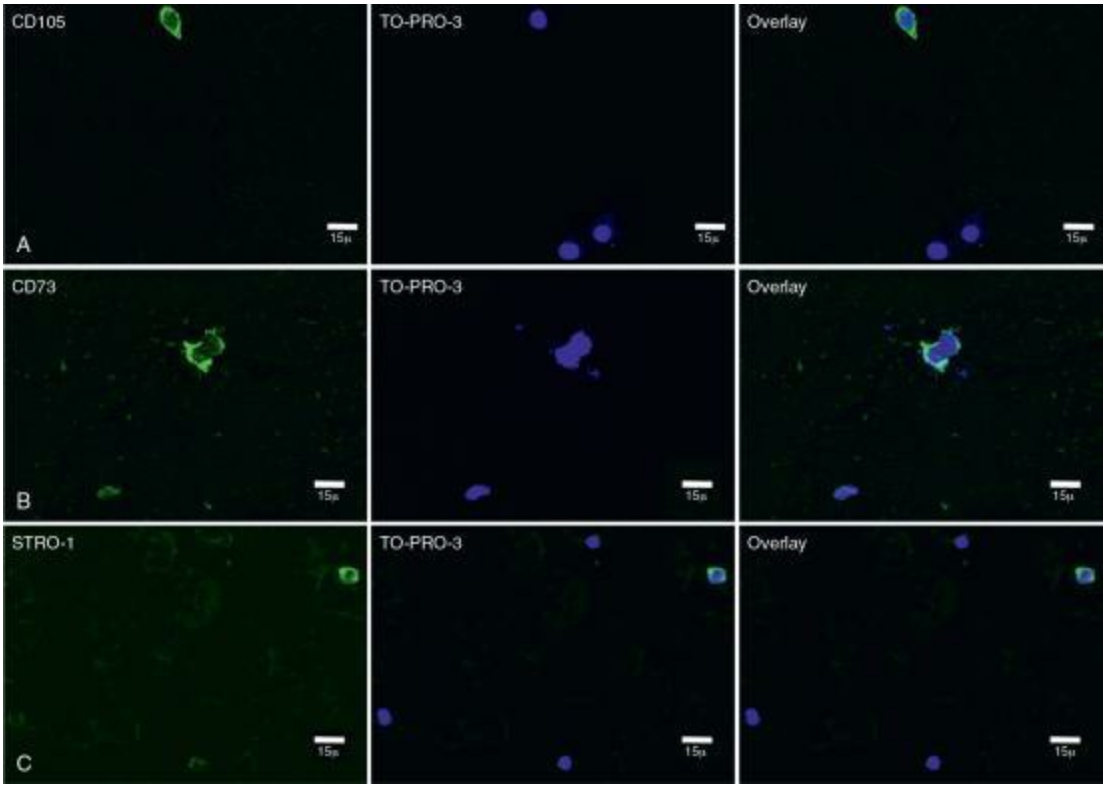

FIG. 12.5 Mesenchymal stem cells were delivered into root canal spaces during regenerative procedures in immature teeth with open apices. Cells collected from intracanal blood samples after the evoked-bleeding step or from systemic blood were stained with antibodies against CD105, CD73, or Stro-1 and evaluated with a laser-scanning confocal microscope. (**A**) Cells in intracanal blood samples collected after evoked-bleeding step showed expression of mesenchymal stem cell marker and CD105 (*green* in **A**), CD73 (*green* in **B**), and STRO-1 (*green* in **C**), whereas nuclei appear blue as stained with TO-PRO-3.

A) Scan for the cluster of differentiation 105, to-pro 3 and overlay shows data as:

Cluster of differentiation 105: A green pigmented stain in the top.

To-pro 3: Three purple dots, one in the top and two in the bottom.

Overlay: A purple dot over a pigmented green dot at the top and two purple dots in the bottom.

B) Scan for the cluster of differentiation 73, to-pro 3 and overlay shows data as:

Scan for cluster of differentiation 73: A large green-pigmented stain in the center with three green stains in the left and bottom.

To-pro 3: A large purple stain over the center and bottom.

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Overlay: A purple stain over a green-pigmented stain in the center, purple stain with green pigments and green dot in the bottom.

C) Scan for the STRO-1, to-pro 3 and overlay shows data as:

Scan for STRO-1 (mesenchyme): A large green pigmented stain in the top right with small green stain in the bottom right.

To-pro 3: Purple dots in top right, left bottom, bottom and overlying purple dot over a green pigment in top right.

Overlay: Purple dots in top right, left bottom, bottom and overlying purple dot over a green pigment in top right.

Source: (From Lovelace TW, Henry MA, Hargreaves KM, Diogenes A: Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure, *J Endod* 37:133–138, 2011.)

The delivery of substantial concentrations of MSCs into the root canal space, despite advanced apical periodontitis or abscess, points to an impressive survival capacity of these cells. In these clinical presentations, low oxygen tension, low pH, and high concentration of endotoxins and inflammatory mediators are expected. Indeed, the finding of high concentrations of the immune cell marker CD14 in those clinical samples indicates that there was still a substantial chronic inflammatory exudate present at the apical region of those teeth. These findings raise the question of how MSCs such as SCAPs can survive during apical periodontitis where a complex microflora, an array of inflammatory mediators, immune cells, and presumably low oxygen tension are commonly encountered. The biologic reason for this apparent resilient survival, despite challenging conditions, may be explained by the relatively low density of blood vessels in the apical papilla in comparison with the adjacent dental pulp, whereas the dental follicle surrounding the apical papilla is highly vascularized and may act as a capillary bed to supply nutrients to SCAPs.[77] Indeed, the apical papilla was found to remain vital despite complete pulpal necrosis and advanced apical periodontitis in animal model of endodontic infection in rodents[290] and dogs.[229] Further evidence of this survival capacity was shown in a clinical case when an apical papilla was harvested through the canal space with a Hedstrom file. This tissue was processed for immunohistochemistry, and

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cells were cultured and found to be multipotent and expressing all recognized marked of MSCs.[59] The cells cultured from the apical papilla of that patient with a history of apical abscess due to pulp necrosis for more than 1 year showed greater mineralization potential than apical papilla cells cultured from healthy extracted third molars.[59] Interestingly, it has been demonstrated that hypoxic environments enhance the proliferation, survival, and angiogenic potential of dental stem cells[4,21,84,305] and that similar enhancing effects were observed when dental stem cells were exposed to bacterial by-products or antigens.[84,308] Thus it appears that SCAPs and surrounding stem cells are equipped to survive and maintain their potential for differentiation in adverse conditions such as apical periodontitis and apical abscesses. Nonetheless, stem cells delivered into root canals after bleeding is evoked from the periradicular tissues are likely from various apical sources or niches, and not restricted to the apical papilla.

A clinical study in 2014 evaluated the intracanal transfer of undifferentiated MSCs from the apical region in adult patients through evoked bleeding.[58] A substantial increase in MSC markers could be detected in intracanal blood samples compared with systemic blood using quantitative real-time polymerase chain reaction (qRT-PCR). In addition, cells were cultured from the intracanal blood sample from tooth #8 in a 35-year-old female patient diagnosed with pulp necrosis and symptomatic apical periodontitis. The cells cultured were adherent to plastic, formed single colonies, coexpressed MSC markers, had high proliferative rates, and differentiated into a mineralizing phenotype.[58] This study supports that apical lesions contain a substantial number of undifferentiated cells and that these cells could participate in an intracanal regenerative or reparative process. Indeed, these procedures have been attempted in adults with fully formed teeth and published as case report[232,318,331] and case series.[252] Although significant root development is not expected in mature roots, it can be argued that regaining nociception and immune defense, a tooth regains surveillance and reparative functions, respectively. However, further evidence is needed to compare the predictability and the long-term success and survival of mature teeth treated with a regenerative approach versus conventional root canal therapy. Regardless of the clinical outcome of current techniques, evidence strongly supports the presence of apical undifferentiated cells in mature teeth

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in adults, opening the possibility for future biologic-based therapies for all patients and not only children with immature teeth.

The dental pulp can be viewed as a core of innervated and vascularized loose connective tissue surrounded by a layer of odontoblasts. The major cell type of this core region is the fibroblast. Together with blood vessels, lymphatics, and neurons, this core tissue is embedded in an extracellular matrix consisting of collagen and other fiber types (see also Chapter 13). DPSCs can be found throughout the dental pulp but are known to accumulate in the perivascular region and the cell-rich zone of Hohl adjacent to the odontoblastic layer.[96,262] Thus DPSCs from both sources are believed to be active participants in the process of reparative dentinogenesis. These cells are recruited to the site of injury following a gradient of chemotactic agents released by resident immune cells and from the damaged dentin.[1,270] The reparative dentin formed by these cells is distinct from the primary, secondary, and reactionary dentin that has been lost.[8,324] It is often called "osteodentin" when found to be disorganized and atubular and having cellular inclusions.[302] This process of cellular repair is known to be enhanced by bioactive materials (e.g., MTA and Biodentine).[168] These materials are known to increase the inherent mineralizing potential of the dental pulp when used in both indirect and direct pulp applications. These biologic events form the basis of contemporary vital pulp therapies that include direct pulp capping and pulpotomies with well-documented high success rates.[137,188,266] However, the physiologic process of tertiary dentinogenesis requires a vital pulp and the resolution of the etiology (e.g., caries or trauma). This process becomes disrupted when the pulp succumbs to injury, resulting in liquefaction necrosis of the dental pulp. Regeneration or repair in this case is only possible with the recruitment or delivery of autologous stem cells to the canal space following adequate disinfection.[77]

Odontoblasts are one of the most specialized cells of the pulp-dentin complex with dentinogenic, immunogenic, and possibly sensorial functions.[40,82,85] Identification of odontoblasts in the intact pulp-dentin complex is easily achieved based on their location and their distinct morphologic characteristics (i.e., columnar polarized cell body with cellular projections into the dentinal tubules). However, identification and characterization of an odontoblast-like cell are far more challenging. One of

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the main reasons for this is that these cells lack a primary odontoblast morphology and unique markers that could be used for identification.[139] Indeed, many markers used for the identification of odontoblast-like cells are also expressed in other mineralizing cell types such as osteoblasts. For example, both odontoblasts-like cells and osteoblasts are similar in the formation of mineralized nodules and in the expression of several proteins such as dentin sialoprotein (DSP), although DSP levels are nearly 400 times greater in odontoblasts than osteoblasts.[309] Measuring only one or two characteristics of a cell might not conclusively identify whether the cell is a true odontoblast. Even among odontoblasts, the phenotype varies in cells located in the apical (squamous shape) versus coronal (tall columnar) pulpal tissue. Importantly, molecular studies have identified many of the genes selectively expressed in odontoblasts.[189,227,228] Lastly, an intermediate filament protein called “nestin” has been shown to be preferentially expressed in odontoblasts or odontoblast-like cells when in the active secretory function. Nestin expression could be used in conjunction with other markers to better identify odontoblast-like cells.[2,3] This knowledge is expected to aid future studies characterizing the conditions necessary for mesenchymal cells of multiple origins to differentiate into odontoblast-like cells. It is likely that definitive cellular identification depends upon both the morphology of the cell and an assessment of the expression of multiple genes.

At least five different types of postnatal MSCs, in addition to DPSCs, have been reported to differentiate into odontoblast-like cells, including SHEDs,[201] SCAPs,[140] iPAPCs,[184] dental follicle progenitor cells (DFPCs),[205] and bone marrow mesenchymal stem cells (BMMSCs).[26] As stated previously, overinstrumentation into the periradicular tissues followed by bleeding into the canal space results in a robust influx of cells with MSC markers in fully mature teeth, similarly to that seen in immature teeth.[58,191] Thus it appears that MSCs from the apical region can be delivered into the root canal spaces in both immature and mature teeth. However, there is growing evidence that MSCs have decreased proliferative and differentiation potential with aging.[166,215] Further research is required to elucidate the age limit for the use of autologous dental-derived stem cells, but these findings suggest that regenerative procedures may be applicable to mature fully formed teeth in adults. Thus it is recognized that more research and development are required

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to make regenerative procedures more predictable for immature teeth and that these procedures may transition to be applicable to fully formed teeth.

Growth factors/morphogens

Dentin is composed of collagen fibers (90%, collagen type I) and noncollagenous matrix molecules (proteoglycans, phosphoproteins, and phospholipids). The collagen fibers act as a grid or matrix, and this structure behaves as a scaffold upon which mineralization can occur. Dentin phosphoprotein (DPP) and DSP are the most abundant dentin-specific proteins among the noncollagenous proteins of organic matrix.[46] DSP resembles other sialoproteins such as bone sialoprotein, but its precise function is still unclear; it may have a role in matrix mineralization.[126] Both DSP and DPP are a part of the small integrin-binding, ligand N-linked glycoproteins (SIBLINGs) which include dentin matrix acidic phosphoprotein 1 (DMP-1), bone sialoprotein, osteopontin, osteocalcin, and osteonectin. These proteins are only a small part of the whole cocktail of noncollagenous proteins which form components of the dentin.[119]

Research on dentin structure and composition has highlighted that the matrix contains some components that may be important in regulating tissue due to their bioactive properties. Based on this, dentin is currently considered a reservoir of growth factors and cytokines.[32,270,274–276] These growth factors/cytokines are secreted by the odontoblasts during primary dentinogenesis, becoming sequestered and trapped or “fossilized” into the dentin matrix after biomineralization (Table 12.1). However, they may become solubilized by demineralization of the matrix, by bacterial acid (caries decay), chemical treatment (ethylenediaminetetraacetic acid [EDTA] rinsing solution, calcium hydroxide or acid etching for bonded restorations), or by restorative materials such as MTA and Biodentine (Fig. 12.6).[272,292]

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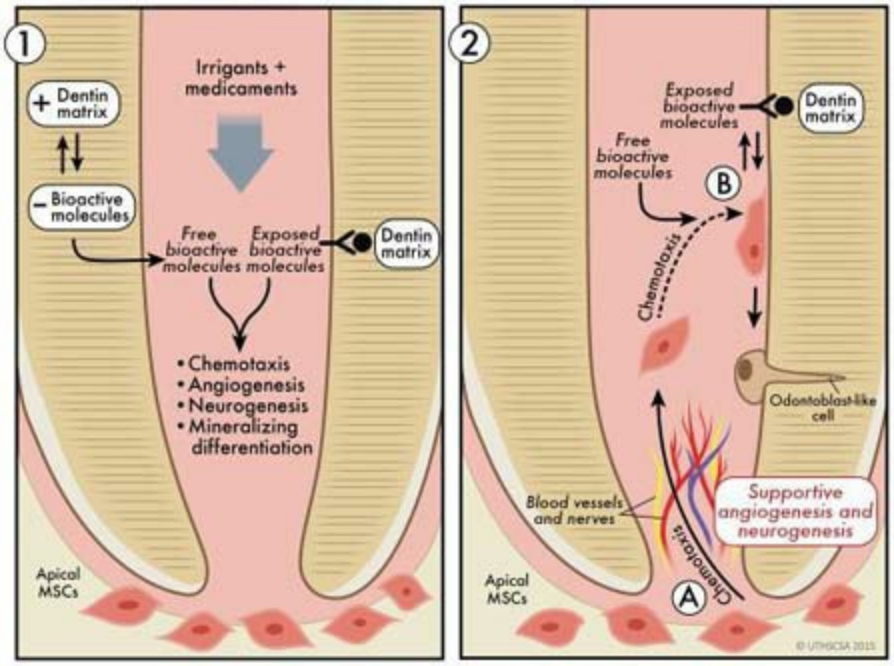

FIG. 12.6 Schematic illustrating the potential actions of irrigants and medicaments in the release and/or exposure of bioactive molecules sequestered in dentin *(1)* and their influences on regenerative events, including chemotaxis of apical undifferentiated cells *(A),* odontoblast-like cell differentiation *(B),* mineralization, angiogenesis, and neurogenesis *(2)*.

1) Lateral diagram of the roots of tooth and apical mesenchymal stem cells show the irrigants and medicaments that are labeled from top to bottom as; irrigants plus medicaments, positive dentin matrix, negative bio-active molecules, free bioactive molecules, exposed bioactive molecules, dentin matrix, chemotaxis, angiotaxis, neurogenesis and mineralizing differentiation.

2) Lateral diagram of the roots of tooth and gingival surface show the irrigants and medicaments that are labeled from top to bottom as; dentin matrix, exposed bioactive molecules, free bioactive molecules, chemotaxis (B), odonoblast like cell, blood vessels and nerves and chemotaxis (A).

Source: (From Smith AJ, Duncan HF, Diogenes A, Simon S, Cooper PR. Exploiting the bioactive properties of the dentin-pulp complex in regenerative endodontics, *J Endod* 42(1):47–56, 2016.)

Table 12.1

List of Growth Factors Found into the Mineralized Dentin Matrix

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Growth Factors in Dentin Matrix	
Transforming growth factor beta-1 (TGF-β1)	Cassidy et al. (1997)[53]
Transforming growth factor beta-2 (TGF-β2)	Cassidy et al. (1997)[53]
Transforming growth factor beta-3 (TGF-β3)	Cassidy et al. (1997)[53]
Bone morphogenic protein 2 (BMP-2)	Thomadakis et al. (1999)[289a]
Bone morphogenic protein 4 (BMP-4)	About et al. (2000)[3]
Bone morphogenic protein 7 (BMP-7)	Thomadakis et al. (1999)[289a]
Insulin-like growth factor-1 (IGF-I)	Finkelman et al. (1990)[95]
Insulin-like growth factor-2 (IGF-II)	Finkelman et al. (1990)[95]
Hepatocyte growth factor (HGF)	Tomson et al. (2013)[292a]
Vascular endothelial growth factor (VEGF)	Roberts-Clark and Smith (2000)[242a]
Adrenomedullin (ADM)	Musson et al. (2010)[208a]

Other growth factors and their receptors have been shown to be present at the enamel organ–dental papilla interface by immunohistochemistry and in situ hybridization during tooth development and have been implicated in odontoblast differentiation:

- Growth hormone (GH) plays a paracrine and/or autocrine role in dental development.[329]
- Insulin-like growth factor beta-1 (IGF-β1) and beta-2 (IFG-β2)[31,157]
- Transforming growth factor beta-1 (TGF-β1), TGF-β2, and TGF-β3[69,288] and bone morphogenic protein 2 (BMP-2), BMP-4, and BMP-6[303] play a role in the polarization and the differentiation of odontoblasts.[31] Notably in adult pulp, TGF-β1 plays an important role in the regulation of the inflammatory response and tissue regenerative processes.[183]

The dental pulp has well-recognized regenerative potential observed in the process of reparative dentinogenesis.[207,271] In this process, dentin-derived growth factors are believed to play a key role to be deciphered into the regulation of progenitor cell recruitment, cell proliferation, and differentiation of new dentin-secreting cells.[183,270] Indeed, the differentiation of new odontoblast-like cells has also been reported following pulp capping with fibroblast growth factor (FGF), TGF-β1 (Li et al. 2014), and BMP-7.[118,183]

The sequestration of these growth factors in the dentin matrix and their subsequent “fossilization” during the mineralization process appear key to the

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pulp healing process where their release from the matrix may be responsible for various signaling events. These growth factors are extremely potent and have a variety of cell signaling properties. However, their precise localization in the dentin[273] and their various biologic roles remain to be elucidated.

It is possible to imagine opportunities for therapeutic stimulation, inducing a targeted release of these proteins. For example, treatment of dentin with EDTA solution has been shown to dissolve the mineral phase, liberating growth factors that orchestrate the stimulation of progenitors or stem cell differentiation.[104,105,110,270,274,276] Etching with orthophosphoric acid, used for conditioning the dentin in bonding procedures, also promotes demineralization of the dentin and liberation of biologic factors.[81,93] For a long time, calcium hydroxide has been used as a protective lining, especially beneath amalgams fillings, or as a canal disinfection medication. It has been shown to have the ability to release bioactive components from the dentin, including growth factors.[123] Unlike dentin-etching acids which have only brief contact with the dentin, calcium hydroxide remains in place beneath restorations or in canals, allowing for a gentle and continued dissolution, thus releasing growth factors; its action is prolonged and potentially controllable depending on the form of the product. Lastly, calcium hydroxide, a by-product of the use of MTA and Biodentine, appears to underlie release of bioactive dentin-derived growth factors by these two bioactive materials.[292] Thus clinicians may take advantage of potent growth factors stored within dentin with the use of chemical treatments and materials that promote the release of these factors (see Fig. 12.6).

Morphogens

It is also important to keep in mind that a second level of regulation exists during dental development (and thus during pulp regeneration process)—transcription factors. Notably, Msx1 is expressed in polarized preodontoblasts, whereas Msx2 is present in mature odontoblasts.[31] Protein and transcripts for Msx1 have been identified in the pulp mesenchyme at early stages of tooth development and their concentrations decreased at the bell stage.[63] The expression of these transcription factors is under the control of growth factors, and they can ultimately have broad-ranging effects. Significantly, BMP-4 upregulates Msx1 and Msx2 expression. In turn,

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transcription factors regulate further growth factor expression; for example, Msx1 upregulates BMP-4 synthesis in the mesenchyme and Msx2 regulates Runx2 and osteocalcin gene expression during odontogenesis.[37,38]

Growth factors and transcription factors are central to the cascade of molecular and cellular events during tooth development and are responsible for many of the temporospatial morphologic changes observed in the developing tooth germ. For these reasons, they are also likely involved in the regeneration process.

It is also important to consider the nature of the signaling process between the injurious agent and the pulp cells. Bacteria and their toxins are key candidates in the direct stimulation of pulp cells.[82] Lipopolysaccharides (LPS) and other bacterial toxins initiate intrapulpal inflammatory processes by activation of Toll-like receptors (e.g., TLR4 activation by LPS).[76,280] Importantly, both progenitor and dental stem cells have been shown to express these receptors.[40,41,133,134,291,330] Thus stem cells within the dental pulp or periradicular tissues are equipped to detect microorganisms. Exposure of these cells to microbial antigens has been shown to directly modulate the proliferation and differentiation potential of these cells.[134,135,190] A study demonstrated that SCAPs differentiation into odontoblast-like cells was blocked when these cells were in contact with a previously infected dentin, resulting in osteoblastic differentiation instead.[308] This study showed that microbial factors can alter the ultimate differentiation fate of stem cells, reinforcing the notion that the microenvironment in which these cells are found dictates their final phenotype. Lastly, cytokines commonly found in the inflammatory milieu (including that of the dental pulp) have a profound effect on stem cells. For example, it has been recently shown that tumor necrosis factor-α (TNF-α) stimulates differentiation of dental pulp cells toward an odontoblastic phenotype via mitogen-activated protein (MAP) kinase pathway activation and p38 phosphorylation.[233,267] Therefore stem cell fate within the dental pulp is ultimately dictated by a complex cascade of intracellular signaling pathways activated by agents released from microorganisms, dentin, and immune cells.

Interestingly, morphogens are not the only naturally occurring factors found in within teeth. Several growth factors have also been evaluated for their ability to trigger the differentiation of selected MSC populations into

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odontoblast-like cells (Table 12.2). Interestingly, several case studies have reported that patients taking long-term corticosteroids often present with dramatic reduction of the radiographic size of the pulp chamber and up to a fivefold increase in the thickness of the predentin layer.[216,217,283] Although these patients were medically complex patients (e.g., renal failure) taking multiple drugs, the use of corticosteroids appeared to be associated with the observed increased activity of human odontoblasts. Furthermore, these unexpected "side-effects" were also observed in a retrospective study evaluating the association of pulp calcifications and the long-term use of statins.[236] These incidental effects of commonly prescribed medications were further evaluated in translational studies that have extended this general observation by demonstrating that the application of dexamethasone or statins greatly increased the differentiation of human dental pulp cells into odontoblast-like cells.[139,224] This was particularly evident when dexamethasone was combined with 1,25-dihydroxyvitamin D_3.[139] Merely changing the composition of growth factors completely altered the differentiation of these cells, with the same population of cells able to express markers of odontoblasts, chondrocytes, or adipocytes, depending on their exposure to different combinations of growth factors.[309] Such findings emphasize the importance of growth factors in guiding the differentiation of these cells. Other studies have evaluated growth factors administered alone or in various combinations for promoting differentiation of odontoblast-like cells.

Table 12.2

Effects of Selected Growth Factors on the Differentiation of Odontoblast-Like Cells

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Growth Factors	Cell Source	Phenotype	Condition	Authors
Dexamethasone	Human dental pulp	Odontoblast-like	In vitro × 8 weeks	Huang et al. (2006)[139]
Dexamethasone and vitamin D_3	Human dental pulp	Odontoblast-like	In vitro × 8 weeks	Huang et al. (2006)[139]
Dexamethasone and ascorbate-2-phosphate and β-glycerophosphate	Human or rat dental pulp	Odontoblast-like	In vitro × 3 weeks	Wei et al. (2007)[309] Zhang et al. (2005)
Insulin and Indomethacin and 3-Isobatyl-1-methylxanthine (IMBX)	Human dental pulp	Adipocyte	In vitro × 19 days	Wei et al. (2007)[309]
Dexamethasone and Insulin and Ascorbate-2-phosphate and Sodium pyruvate and TGF-β1	Human dental pulp	Chondrocyte	In vitro × 8 weeks	Wei et al. (2007)[309]
Growth/differentiation factor 11 (Gdf11)	Dental pulp	Odontoblast-like	In vitro/in vivo 10 days	Nakashima et al. (2004)
Simvastatin (statins)	Human dental pulp	Odontoblast-like	In vitro/in vivo	Okamoto et al. (2009)[224]
LIM mineralization protein 1 (LMP-1)	Human dental pulp	Odontoblast-like	In vitro/in vivo	Wang et al. (2007)
Bone morphogenetic proteins	Dental pulp	Odontoblast-like	In vitro	Saito et al. (2004) Sloan et al. (2000) Chen et al. (2008)
TGF-β1–3	Rat/monkey dental pulp	Odontoblast-like	In vitro	Sloan et al. (1999)
Demineralized dentin	Human or rodent pulp	Odontoblast-like	In vitro/in vivo	Smith et al. (1990)[274] Smith et al. (2001) Tziafas (2004)
Nerve growth factor (NGF)	Immortalized apical papilla	Odontoblast-like	In vitro	Arany et al. (2009)
Fibroblast growth factor 2	Human dental pulp	Odontoblast-like	In vitro	He et al. (2008)
Dentin matrix protein 1	Rat dental pulp	Odontoblast-like	In vivo	Almushayt et al. (2006)

Several of the approaches using compounds later found to have growth factor–like effects have immediate clinical implications. First, it is unlikely that a single growth factor will result in maximal differentiation, so combinations of growth factors may be required for evaluation in clinical trials. Related to this point, many of the studied growth factors (e.g., dexamethasone, insulin) are drugs already approved for human use in other medical/dental applications. Second, the demonstration that statins promote the differentiation of an odontoblast-like phenotype suggests that patients clinically taking statins may also have narrowing of the pulp chamber space, similar to the findings previously described for corticosteroids. This would be an important future area of research. Third, clinicians have long used demineralized human bone to augment healing after surgical procedures.[240] Demineralized human bone is thought to contain a natural combination of

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appropriate growth factors and scaffolds, thereby providing an appropriate environment for osteoblast differentiation or function. Extending this concept, several research groups have demonstrated that demineralized human dentin has significant benefit for promoting the differentiation of odontoblast-like cells. Importantly, translational studies in regenerative endodontics have demonstrated that irrigation of dentin with 17% EDTA increases the survival of stem cells[194,300] and odontoblastic differentiation,[106,194] possibly due to the release of bioactive molecules from dentin.[270] Collectively these findings suggest that EDTA irrigation of the dentinal walls as part of a REP could result in improved clinical outcomes.

Scaffolds

An important component of tissue engineering is a physical scaffold.[176,214] Tissues are organized as three-dimensional structures, and appropriate scaffolding is necessary to (1) provide a spatially correct position of cell location and (2) regulate differentiation, proliferation, or metabolism while promoting nutrient and gaseous exchanges. It is known that extracellular matrix molecules control the differentiation of stem cells,[238,239,321] and an appropriate scaffold might selectively bind and localize cells, contain growth factors,[107] and undergo biodegradation over time.[327] Thus a scaffold is far more than a simple lattice to contain cells but instead can be viewed as the "blueprint" of the engineered tissue.

Scaffolds can be classified as either natural or synthetic. Examples of natural scaffolds include collagen,[211,213] glycosaminoglycans, hyaluronic acid (HA), demineralized or native dentin matrix,[28,57,127,212] and fibrin.[107] On the other hand, examples of synthetic scaffolds include poly-L-lactic acid (PLLA),[74] polyglycolic acid (PGA), polylactic-coglycolic acid (PLGA),[87] polyepsilon caprolactone,[322] hydroxyapatite/tricalcium phosphate,[16] bioceramics, and hydrogels such as self-assembly peptide hydrogels.[109] The great majority of currently published REPs involve evoked bleeding and the formation of a blood clot to serve as a scaffold.[77,173] Although it relatively straightforward because it does not require ex vivo manipulation, this simplistic approach is not without challenges. The blood clot is often difficult to achieve, and it does not have many of the properties of the ideal scaffold.

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These properties include easy delivery, adequate mechanical properties, controllable biodegradation, and incorporation of growth factors.[107] In addition, the blood clot contains a great number of hematopoietic cells, which eventually undergo cell death releasing their toxic intracellular enzymes in the microenvironment that may be detrimental to stem cell survival.

Another approach for creating a scaffold involves the use of autologous PRP. It requires minimal ex vivo manipulation, being fairly easy to prepare in a dental setting. PRP is rich in growth factors, degrades over time, and forms a three-dimensional fibrin matrix.[20,148,223] Platelet-rich fibrin (PRF) is an alternative to PRP because it has a three-dimensional architecture conducive with stem cell proliferation and differentiation and contains bioactive molecules.[73,75] These autologous scaffolds have been used successfully in regenerative cases.[173] However, it should be emphasized that, despite their reported use, there are some drawbacks to their clinical use; they require collection of intravenous blood that can be challenging in children, the diversity and concentration of growth factors within PRP and PRF preparations are not controllable, and they lack temporal degradation control and the mechanical strength to support a coronal restoration. Thus, despite some desirable characteristics, other scaffold alternatives to PRP and PRF should be carefully considered.

Hydrogels are a class of scaffolds composed of three-dimensional hydrophilic polymers that absorb water or tissue fluids up to several times their weight.[94,109] These water-swollen materials are easily injectable in their colloidal form, undergoing gelation by chemical (e.g., changes in pH and osmolarity) and/or physical (e.g., temperature change) cues. These materials are highly tunable and biocompatible and can be designed to resemble naturally occurring extracellular matrices.[319] They are of particular interest for regenerative endodontics because they can be easily injected into narrow root canal spaces and be modified to deliver chemotactic and angiogenic agents to drive stem cell homing and supportive angiogenesis.[77,121] Hydrogels made of self-assembly peptides (e.g., Puramatrix)[54] show great potential to be used in endodontic tissue engineering because their sequence includes short peptide sequences, similar to those naturally occurring in tissues, enhancing cell attachment and proliferation.[111] Similarly, fibrin-based scaffolds have been shown to have excellent properties that they may

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be well suited for use in regenerative endodontics. These scaffolds have the potential to be functionalized because they can be loaded with growth factors or dentin matrix protein extracts that promote in vitro and in vivo proliferation and proper differentiation of stem cells.[103,311] In addition, fibrin is a natural scaffold that already is used in medicine (e.g., surgical fibrin glue) and could potentially be adopted for use in REPs.

Decellularized human and animal tissues have been used as scaffolds for many decades in regenerative medicine and dentistry.[116] An adequate decellularization procedure is needed to obtain a suitable ECM scaffold from native tissues. It should minimize the loss or damage of the extracellular matrix (ECM) components and maximize the removal of the cellular component to reduce the immunologic responses.[116,316] There are many examples of decellularized tissues that have been approved by the US Food and Drug Administration (FDA) to be used as scaffolds, including dermal allograft (Alloderm), human peripheral nerves (Avance Nerve Graft, Axogen), porcine small intestine submucosa (SurgiSIS, Cook Biotech), porcine urinary bladder (MatriStem, Acell), and porcine heart valves (Syntergraft, Cryolife).[12,44] Unfortunately, these scaffolds do not share the intricate ultrastructure and molecular composition of the dental pulp. A study demonstrated that human dental pulp from extracted healthy third molars can be successfully decellularized and used as a scaffold.[277] SCAPs, when seeded in the decellularized dental pulp scaffold, proliferated in three dimensions and differentiated into odontoblast-like cells extending cellular processes into dentinal tubules (Fig. 12.7).[277] This study introduced the concept that harvested dental pulp, from otherwise discarded extracted molars, could be made into scaffolds for use in regenerative endodontics, similarly to many other tissues currently available for tissue specific regeneration in medicine. The results of this study have been confirmed by another study that evaluated the use of decellularized swine dental pulp in REPs.[14] The authors demonstrated that the decellularized scaffold allowed for cell proliferation in vitro and that its implantation into pulpectomized teeth in dogs favored the recruitment of apical stem cells resulting in the formation of a vascularized pulplike tissue with expression of an odontoblastic marker (i.e., DSP).[14] Collectively, these studies demonstrated that, similarly to other tissues, dental pulp could be decellularized and used as scaffold for regenerative purposes.

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This allograft approach is further facilitated by the readily available source of this tissue because millions of healthy third molars are extracted and discarded every year in the United States alone. Alternatively, dental pulp from animals such as swine could be used as the source of a xenograft scaffold.

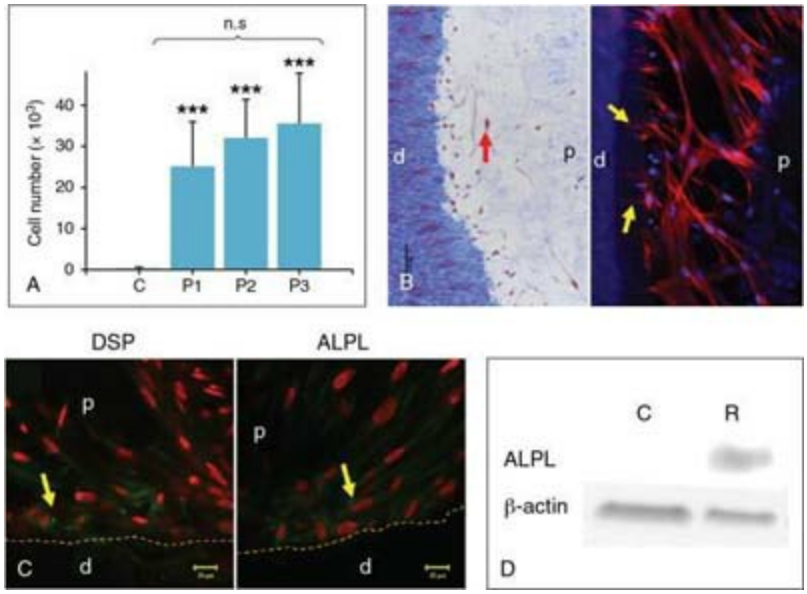

FIG. 12.7 SCAP repopulation in decellularized dental pulp scaffold. (**A**) Cell viability in repopularized human dental pulp (rHDP) at 2 weeks after cell seeding. The data are presented as mean number of viable cells and standard deviation values. ****P* <.001 with one-way ANOVA analysis followed by post hoc Bonferroni test compared with control (b) Masson trichrome staining (a) and fluorescent staining with 4',6-diamidino-2-phenylindole (DAPI) and phalloidin at 2 weeks after cell seeding. *Arrowhead* in left side indicates repopulated cells which have spindle-like shape, those in the right side indicate odontoblastic processes (**B**). There was no statistically significance between other experimental groups. (**C**) Immunofluorescence cell staining at 8 weeks after cell seeding for dentin sialoprotein (DSP) and alkaline phosphatase, liver/bone/kidney (ALPL). Positive signals *(green)* were mainly found in the cells adjacent to dentin. (**D**) Western blot analysis at 8 weeks after cell seeding revealed elevation of ALPL expression in rHDP group. Scale bars: 50 μm in **B** and 20 μm in **C** and **D.** *C,* Control; *P1,* protocol 1; *P2,* protocol 2; *P3,* protocol 3; *p,* dental pulp; *d,* dentin; *R,* repopulation.

Plot shows C, P1, P2 and P3 against cell number (multiplied with 10 raised to power 3). The horizontal axis shows bars for C, P1, P2 and P3. The vertical axis ranges from 0 to 40 in increments of 10.

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The data is as follows:

C: 0.5, P1: 25, P2: 33 and P3: 35.

B) Light scan of the vertically segmented p and d region surfaces shows an arrow pointing to a red stain in p region. Dark scan of the vertically segmented p and d region surfaces shows two arrows pointing to two purple stains in p surface.

C) Dark scan of the laterally segmented p and d region surfaces shows an arrow pointing to a green dot in p region. Dark scan of the laterally segmented p and d region surfaces shows two arrows pointing to a red stain in p surface.

D) The data for blot analysis is as follows:

ALPL: C: blank

ALPL: R: the data is blurred.

ALPL: the data is blurred : the data is blurred.

Source: (Reproduced with permission from Song JS, Takimoto K, Jeon M, et al: Decellularized human dental pulp as a scaffold for regenerative endodontics, *J Dent Res* 96[6]:640–646, 2017.)

Delivery system

Even with selection of the appropriate cell source, growth factors, and scaffold, the resultant mixture must be delivered in a spatially appropriate fashion into the space of the root canal system. For example, nearly all cells of the body are within 0.1 to 1 mm of a blood vessel to maintain adequate diffusion of oxygen and nutrients.[122,136] This represents a challenge still to be overcome in the currently performed REPs that recruit stem cells[58,191] to a canal space devoid of lateral vascularity and several millimeters away from apical blood vessels. If one were to inject cells, in a cell-based approach, along the entire coronal-apical extent of a root canal system, the vast majority of cells would be expected to succumb to tissue hypoxia. Interestingly, it has been demonstrated that, under hypoxic conditions, stem cells proliferate faster and release greater levels of angiogenic factors such as vascular endothelial growth factor 1 (VEGF-1) that promote targeted angiogenesis into the engineered space.[21] Thus an alternative approach would be to inject a scaffold with chemotactic factors into the root canal. This approach is called

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cell homing because cells are attracted to the scaffold along with supportive blood vessels in a progressive manner;[170] instead of being abruptly delivered to an avascular space (i.e., similar to the current revascularization procedures), the cell-homing approach can be applied in a cell-free[108] (no cells implanted along the chemotactic factors) (Fig. 12.8) or cell-based approach (cells are delivered in the chemotactic-containing scaffold).[77,208] Because dental pulp can be approximated as a loose connective tissue core surrounded by a layer of odontoblasts, the spatial arrangement of cells and growth factors within the scaffold may be particularly important to promote odontogenesis without having complete calcification of the root canal system. Complete recapitulation of the pulp-dentin complex architecture requires additional research effort.

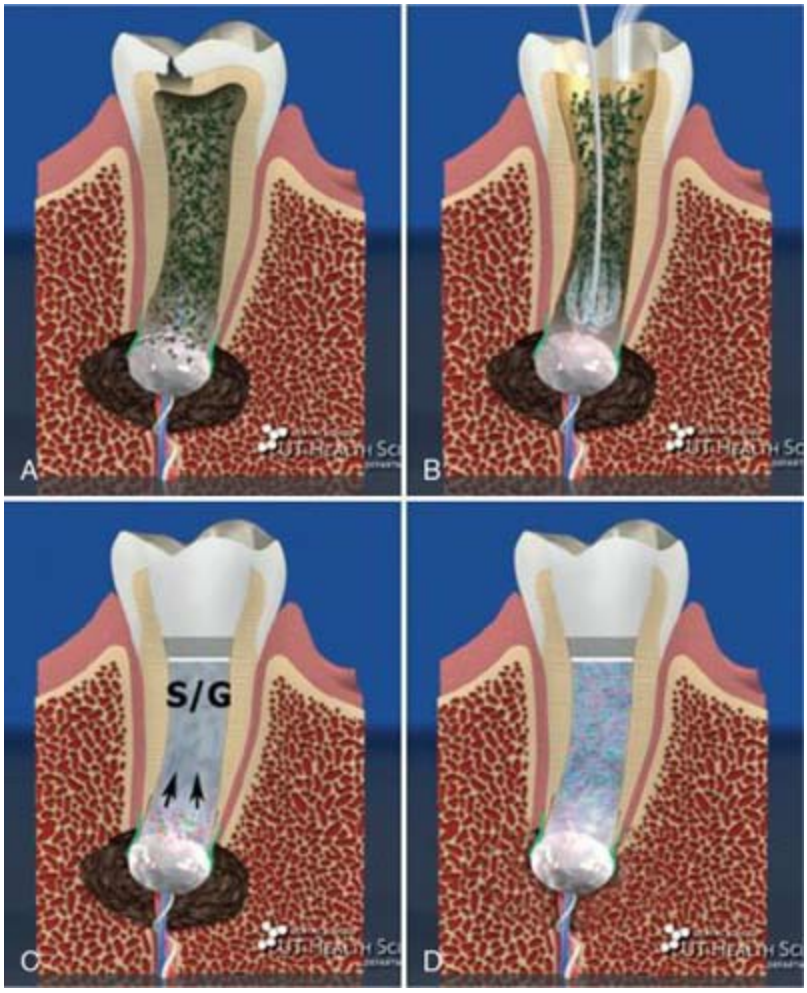

FIG. 12.8 Schematic drawing illustrating bioengineering using a cell

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homing approach. An immature premolar with a necrotic pulp and apical lesion **(A)** is disinfected **(B),** followed by placement of a biodegradable scaffold *(S)* containing growth factors and chemotactic factors *(G)* to allow progressive proliferation and migration of apical stem cells into the canal space **(C)** leading to the population of the canal space with stem cells concomitantly with vascular supply and tissue organization **(D).**

A) Lateral cutaway diagram of root pulp and granuled apical lesion shows a scaffold at the pulp end.

B) Lateral cutaway diagram of root pulp and granuled apical lesion shows a file inserted into an infected canal with a tube exposed to infection for extraction.

C) Lateral cutaway diagram of root pulp and granuled apical lesion shows a scaffold at the pulp end with arrows pointing to the scaffold injected growth and chemotactic factors represented by green and red colored dots.

D) Lateral cutaway diagram of root pulp and granuled apical lesion shows a scaffold at the pulp end with completed process of injecting growth and chemotactic factors, represented by a multicolored canal.

Source: (From Diogenes A, Henry MA, Teixeira FB, Hargreaves KM: An update on clinical regenerative endodontics, *Endod Topics* 28:2–23, 2013.)

Translational studies

Several elegant studies in regenerative endodontics have used various translational methodologies, including evaluation of clinical samples,[59,191] organotype root canal models,[194,300] tooth slice models,[74,120,251] whole tooth culture,[178,286] and animal models.[106,170,212,213,295,296] These studies have been crucial to provide a strong scientific foundation for the field of regenerative endodontics while allowing for clinical treatment protocol optimization, and the development of new treatment strategies such as inclusion of scaffold and growth factors in regenerative procedures.[211,219]

A study demonstrated that new dentin and pulplike tissue could be generated in human root segments implanted subcutaneously in immunocompromised mice.[141] In this study, root segments had one of the openings sealed with MTA to mimic the coronal restoration of regenerative endodontic cases. The canal space was filled with either SCAPs or DPSCs in

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a PLGA-based scaffold. The implants were harvested 3 months later and processed for immunohistochemical analysis. The results indicated that there was a dramatic circumferential apposition of dentinlike material along the dentinal walls. The new mineralized tissue was lined with polarized cells expressing odontoblastic markers. In addition, the dentinlike tissue was largely atubular and displayed cellular inclusions similar to the histologic presentation of osteodentin. Importantly, the cells from the engineered pulp were positive for human mitochondria demonstrating that they were originated from the implanted human stem cells and not from the host (mouse). Lastly, the root segments that were implanted without stem cells had only connective tissue that did not resemble a pulplike tissue nor had mineralized tissues and odontoblast-like cells. Thus a pulp-dentin complex could be engineered in human roots implanted subcutaneously in immunodeficient mice.

Complete pulpal regeneration in dogs has been accomplished by Nakashima and colleagues.[142] In this elegant study, dental pulp was removed via sterile pulpectomy procedure, followed by placement of sorted CD105+ DPSCs in a collagen gel to the midroot. The remaining coronal part of the canal was back-filled with the collagen gel containing the chemotactic factor stromal-derived factor 1 (SDF-1). Subsequent histology demonstrated formation of new pulp tissue with innervation, vascularization, and odontoblast-like cells lining the dentinal walls. In addition, the engineered pulp had protein and RNA expression similar to the native dental pulp.[142]

In another important study, mobilized DPSCs were generated by selecting DPSCs that migrated towards a concentration gradient of granulocyte colony-stimulating factor (G-CSF). These selected cells were implanted in a collagen gel into pulpectomized root canals.[143] Complete pulpal regeneration was observed, with evidence of new dentin formation, blood vessels, and innervation in the engineered tissue (Fig. 12.9). The impressive results with this cell-based approach have laid the foundation for the use of this technology in emerging clinical trials using stem cell transplantation.[215]

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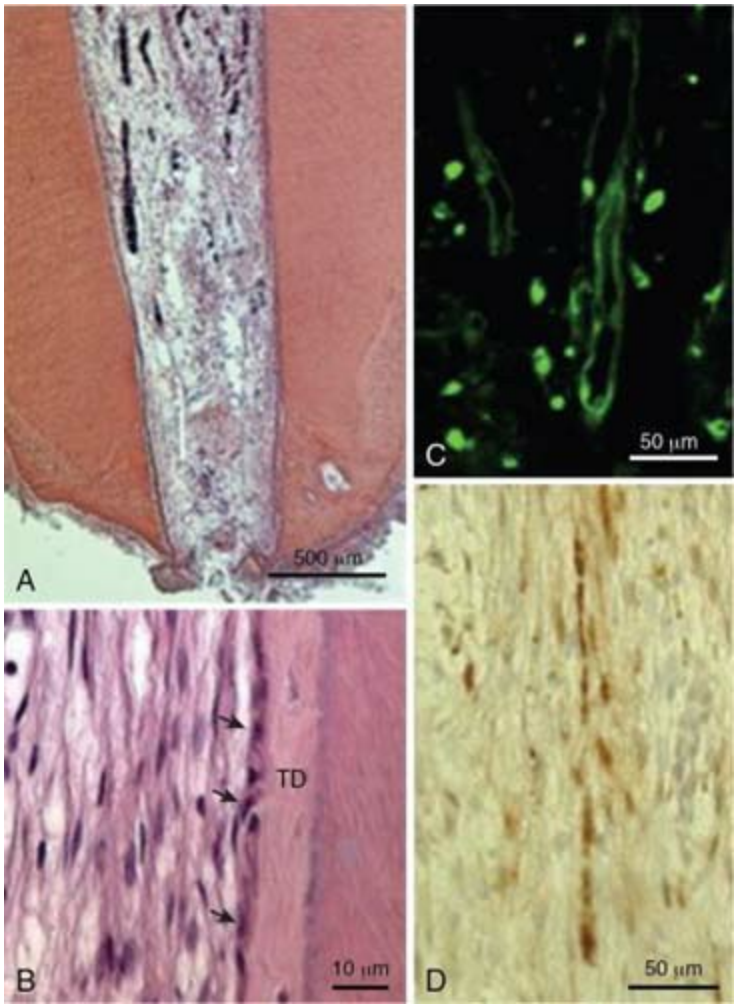

FIG. 12.9 Regeneration of pulp tissue after autologous transplantation of mobilized dental pulp stem cells (MDPSCs) with G-CSF in pulpectomized teeth of young dogs on day 14. (**A** and **B**) Regenerated pulp tissue. (**B**) Odontoblastic cells *(black arrows)* lining to newly formed osteodentin/tubular dentin *(TD)* along with the dentin. (**C**) Immunohistochemical staining of BS-1 lectin. (**D**) Immunohistochemical staining of PGP9.5.

A) Micrograph of canal shows a ruptured pulp end.

B) Micrograph with extended magnification show ruptured pulp tissues with three arrows pointing to black dots representing ruptures.

C) Specimen shows pigments of green stains on a black background.

D) Specimen shows pigments of brown stains on a beige background.

Source: (From Nakashima M, Iohara K: Mobilized dental pulp stem cells for pulp regeneration, *J Endod* 40:S29, 2014.)

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It is noteworthy that all the demonstrations of pulp regeneration in animal models were in root canals without history of infection and pulpal necrosis.[142-144,251] Previously infected root canals must be adequately disinfected to suppress chronic inflammation that is detrimental to regeneration.[62] However, many irrigants and medicaments have detrimental effects on stem cell survival and differentiation.[79] Several studies using organotype root canal models and animal models evaluated the combination and concentrations of irrigants and medicaments that allowed for stem cells proliferation and differentiation.[15,177,194,300,310,312] Furthermore, emerging studies demonstrated that bacterial antigens have a robust effect on the terminal differentiation of stem cells guiding these to a greater osteogenic as opposed to an odontoblastic phenotype.[40,308] Thus adequate disinfection, resolution of inflammation, and the detoxification of residual microbial antigens appear to be limiting factors in complete pulpal regeneration, despite exciting results from current advances in dental pulp tissue engineering.[79]

Summary of basic research on regenerative endodontics

Regeneration of a functional pulp-dentin complex relies on the foundation of tissue engineering and can be viewed as a function of the spatially correct delivery of appropriate stem cells and growth factors embedded within a scaffold. Although considerable research has used in vitro cell culture methods to identify key factors regulating the differentiation of odontoblast-like cells, it should be noted that emerging studies conducted in animal models are quite promising for regeneration of this pulp tissue. Preclinical studies involving surgical placement of a human tooth filled with human stem cell/growth factor/scaffold combination into immunocompromised mice[74,106,251] have permitted histologic analysis of neovascularization as well as the differentiation and mineralization activity of newly formed odontoblasts. The use of human cells in a mouse model permits histologic confirmation that the resulting odontoblast-like cells were of human origin. These novel findings provide strong evidence that either human SCAP or DPSC cell sources, on a PLGA scaffold, were able to regenerate a vascularized tissue that had histologic evidence of odontoblast-like cell differentiation and the spatially appropriate formation of dentinlike material onto the root canal walls. Although no specific growth factors were added to

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this mixture, it is important to note that the root canal walls were treated with 17% EDTA, an irrigant known to expose endogenous growth factor proteins embedded in the dentinal walls.[332] This and other related studies provide strong impetus for clinical translational research evaluating various potential regenerative endodontic therapies.

Clinical studies on regenerative endodontics

To date, most case reports, case series, and retrospective studies published in regenerative endodontics have not fully incorporated the tissue-engineering concepts described. Instead, most of these reports present cases with variations of revascularization techniques.[77] These procedures were initially performed empirically with a strong focus on disinfection and the intentional bleeding into the root canal. However, it became obvious that these procedures were in fact stem cell–based procedures with all the three components of the tissue-engineering triad present: stem cells,[58,191] growth factors,[32] and scaffolds.[27,173,293] Important preclinical studies previously described in this chapter have provided the foundational framework for a paradigm shift. This shift represents a clear departure from the traditional "disinfect the canals at all cost" to "disinfection while creating a microenvironment conducive for tissue engineering." Varied terminologies have been given to these procedures that include revascularization,[27] revitalization,[155,181,186,195,299] and maturogenesis.[5,115,210] Of these terms, the most popularly used has been revascularization. This term is largely based on the trauma literature observation that immature teeth could become "revascularized" after trauma. However, revascularization is a term better used for the reestablishment of the vascularity of an ischemic tissue, such as the dental pulp of an avulsed tooth. From this perspective, a focus on revascularization would ignore the potential importance of growth factors and scaffolds that are required for histologic recapitulation of the pulp-dentin complex. Although we appreciate that angiogenesis and the establishment of a functional blood supply are a key requirement in the maintenance and maturation of a regenerating tissue, it is noteworthy that some of the published cases report positive responses to pulp sensitivity tests such as cold or electric pulp test.[77] In addition, histologic analysis of human teeth treated

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with these procedures revealed the rich presence of innervation that includes nociceptors.[24,179] This is evidence that a space that was previously vacant (débrided root canal) may become populated with an innervated tissue supported by vascularity. Furthermore, studies that performed histologic analyses of the tissue formed following these procedures in patients revealed the presence of ectopic bone formation, periodontal ligament, and cementum in addition to vascularized loose connective tissue.[30] However, odontoblast-like cells and newly formed tissue resembling dentin have also been found in certain cases.[24,234,263] Taken together, the core concepts of tissue engineering distinguish a regenerative treatment philosophy from a revascularization philosophy derived from certain trauma cases (which only occur in a low percentage of replanted teeth). Lastly, there have been a number of reports of procedures performed with intentional manipulation of the principles of tissue engineering such as use of autologous PRP,[9,112,151,237,293] PRF,[206,210] and exogenous growth factors and scaffolds.[173] Thus, instead of using different terms for each variant of these procedures, we will refer to them simply as REPs that include past, present, and future procedures that aim to the functional regeneration of the pulp-dentin complex.

Clinical procedures related to regenerative endodontics

There are several challenges clinicians face when presented with an incompletely formed root in need of endodontic treatment.[77] Because the apex is not fully developed and often has a blunderbuss shape, cleaning and shaping of the apical portion of the root canal system can be difficult. The process is further complicated by the presence of thin, fragile dentinal walls that may be prone to fracture during instrumentation or obturation. In addition, the open apex increases the risk of extruding material into the periradicular tissues. Traditionally an immature tooth with an open apex is treated by apexification, which involves creating an apical barrier to prevent extrusion. In many cases, this entails an involved, long-term treatment with $Ca(OH)_2$, resulting in the formation of a hard-tissue apical barrier.[65,66,99] However, a disadvantage of the traditional apexification procedures is that the short-term or long-term use of $Ca(OH)_2$ has the potential to reduce root strength.[29,80,163,250,269,304,323] This finding is consistent with a large case

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series using the traditional apexification protocol; it showed that a major reason for tooth loss following apexification was root fracture.[65,152] In a retrospective study, the use of calcium hydroxide in apexification procedures resulted in the fracture of 23% of the teeth treated during the follow-up period of up to 18 months.[152] The advent of one-step apexification, by creation of artificial barriers (i.e., apical plugs) using materials such as MTA,[196,197,314,315] has greatly decreased the number of appointments and time to completion. Importantly, the one-step apexification has been shown to have as high success rate as apexification with calcium hydroxide in resolving apical periodontitis (both symptoms and radiographic presentation).[297] However, apexification procedures do not generally result in further root development.[39] A primary advantage of REPs in these cases is the greater likelihood there will be an increase in root length and root wall thickness, in addition to the possible regain of vitality responses. In addition to managing the open apex, REPs have been shown to be beneficial in cases of root resorption promoting the arrestment of the resorptive process and repair of the resorbed root (Fig. 12.10).[164,254,301]

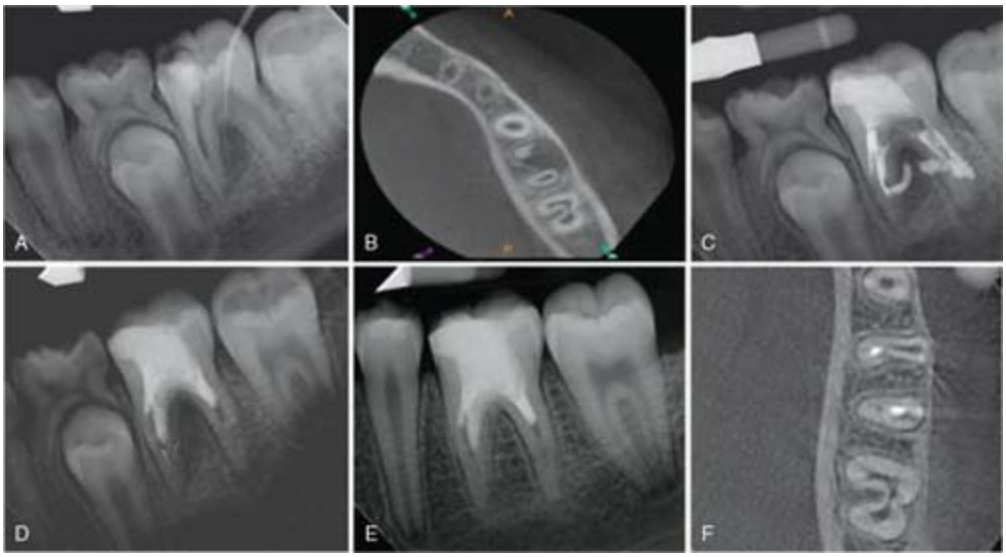

FIG. 12.10 An 11-year-old female was referred for evaluation of tooth #19 diagnosed with pulp necrosis and chronic apical abscess traced with a gutta-percha cone as seen in the periapical (PA) radiograph (**A**). A small-volume CBCT revealed resorption in mesial and distal roots (**B**). The canals were disinfected and medicated with Vitapex (**C**). Four weeks later, the patient was asymptomatic and tooth #19 and the sinus

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tract healed. The tooth was reaccessed, irrigated with 1.5% NaOCl and 17% EDTA, and bleeding evoked in all canals precurved 25 K. MTA was placed over the blood clot followed by glass ionomer and composite restoration (**D**). After 24 months, the patient was asymptomatic, the tooth responded to electric pulp tester (EPT) demonstrated complete healing as seen in PA radiograph (**E**) and small-volume CBCT (**F**).

A) Radiograph of a tooth shows a radiopaque part of crown, radiolucent neck and dark canals. A radiopaque outline of a file is visible.

B) Radiograph of a canal shows presence of radiopaque loops with radiolucent centers.

C) Radiograph of a tooth shows a radiopaque part of crown, radiopaque neck and radiopaque partial canals. A radiopaque outline of a sinus tract is visible.

D) Radiograph of a tooth shows a radiopaque part of crown, radiopaque neck and radiopaque partial canals.

E) Radiograph of a tooth shows a radiopaque part of crown, radiopaque neck and radiopaque partial canals with scaled gingival surface.

F) Radiograph of a canal shows presence of radiopaque loops with radiolucent and radiopaque centers.

Source: (Case courtesy of Dr. Ahmed Alelyani, University of Texas Health Science Center.)

There have been numerous published cases of REPs. Investigators and clinicians have used a variety of medicaments to disinfect the canal space.[77] Between 51% and 80% of the cases included the use of a triple antibiotic paste (TAP) (a 1:1:1 mixture of ciprofloxacin/metronidazole/minocycline), whereas 20% to 37% used $Ca(OH)_2$ as an intracanal medicament.[77,173]

The development of the TAP was led in large part by Hoshino and colleagues.[258,259] They demonstrated the effectiveness of combinations of antibiotics (and in particular the high efficacy of the combination of ciprofloxacin, metronidazole, and minocycline) in eradicating bacteria from the infected dentin of root canals.[258] Astute practitioners realized that the TAP could be a valuable adjunct for revascularization procedures because it could be used to create an environment favorable for the ingrowth of vasculature and regenerative cells by reducing or eradicating bacteria in the canal space of teeth with necrotic pulps and incompletely formed apices. The

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efficacy of the TAP in disinfecting necrotic root canal systems has been demonstrated in a preclinical model.[313] In this dog study, 60 teeth were accessed and infected by sealing dental plaque and sterile saline on a cotton pellet into the pulp chamber for 6 weeks. By the end of this period, each premolar was radiographically confirmed to have apical periodontitis. The canals were then sampled at three time points: before and after irrigation with 1.25% NaOCl, and 2 weeks after the delivery of the TAP into the root canal system using a Lentulo spiral. Before irrigation, all of the teeth had positive cultures for anaerobic bacteria, with a mean colony-forming unit (CFU) count of 1.7×10^8. After irrigation with 1.25% NaOCl, 10% of the teeth sampled cultured bacteria free. The mean CFU count was 1.4×10^4, or an approximate 10,000-fold reduction in viable bacteria. After dressing with the TAP for 2 weeks, 70% of the teeth sampled cultured bacteria free. The mean CFU count was only 26, which is approximately another 1000-fold drop in bacteria. These findings were confirmed in another related dog study.[289] This study provides strong support for the effectiveness of the TAP in disinfection of immature teeth with apical periodontitis.

As stated previously, calcium hydroxide has been the second most used intracanal medicament in published cases. This application represents a new use of a long established intracanal medicament in endodontics. Although $Ca(OH)_2$ appears to be less effective against some intracanal bacterial species than antibiotic paste formulations,[22,249] its use is associated with lower cytotoxicity to stem cells,[15,247] release of important bioactive growth factors from the treated dentin,[128] and greater survival and proliferation of stem cells in presence of the conditioned dentin.[113] In addition, the relatively short-term use of this medicament in regenerative procedures does not appear sufficient to reduce fracture resistance.[323] Another factor to consider when choosing an intracanal medicament is the ability to remove the medicament from the canal space. One study that addressed this question incorporated radioactive tracers in both calcium hydroxide paste (Ultracal; Ultradent, Inc.) and TAP (Champs Pharmacy; San Antonio, TX).[36] The radiolabeled medicaments were placed in extracted teeth with standardized root canals. After 28 days of incubation, canal spaces were irrigated with a standardized protocol using different techniques. Surprisingly, greater than 80% of the TAP could not be removed from the tooth (Fig. 12.11), and it was found not in the canal lumen but

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greater than 350 μm into the dentinal tubules. In contrast, greater than 80% of calcium hydroxide was removed (see Fig. 12.11) with the remaining medicament present in superficially within dentin.[36] This is an important finding, given that drugs remaining within dentin are likely to have an effect on the fate of stem cells in contact with the treated dentin. Another study revealed that TAP, when used at concentrations typically used in case reports (1 g/mL), and removed with standardized irrigation protocol, resulted in no SCAP survival.[15] In contrast, dentin treated with calcium hydroxide promoted SCAP survival and proliferation, and dentin treated with TAP at a 1 mg/mL had no effect on SCAP survival (Fig. 12.12). The ultimate success of REPs relies on maximizing disinfection and detoxification of the root canal system while minimizing detrimental effect on survival and differentiation of stem cells. Therefore it is imperative that the choice of medicament and concentration must also be antimicrobial. The concentration of 1 mg/mL of TAP, approximately 1000 times less concentrated than commonly used in the form as paste, has been tested for its antimicrobial efficacy in a randomized clinical trial using qRT-PCR to detect residual bacteria.[22] In that study, this concentration of TAP resulted in significantly less measurable bacteria than a mixture of calcium hydroxide and chlorhexidine.[22] This clinical study confirmed the findings of many in vitro studies demonstrating that the effective antibacterial concentration of either triple or double antibiotic is 1 to 10 mg/mL.[150,154,177,249,284] It is important to mention that at the concentration of 1 to 10 mg/mL in aqueous medium, the antibiotic formulation is a solution. Despite this solution being effective clinically,[22] its placement and bioavailability over 2 to 4 weeks is more challenging than in a paste form. However, this concentration can be easily used if combined with a macrogol and propylene glycol pasty vehicle as first described by Hoshino and colleagues to facilitate placement and enhance dentinal tubule penetration.[64,138,259] Alternatively, the use of a hydrogel solution has been investigated and methylcellulose found to be a suitable vehicle for delivery of lower concentration of antibiotic mixtures,[284] and it can be combined with a radiopaque agent to allow for radiographic confirmation of its adequate placement.[307] Lastly, resorbable drug delivery systems are being developed to allow for localized and controlled drug release of predefined concentrations of drugs that are known to be

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antibacterial while less detrimental to the microenvironment and cells involved in the regenerative process.[10,42,162] Thus clinicians must carefully evaluate the advantages and disadvantages of each intracanal medicament while observing the ideal therapeutic concentration.[79]

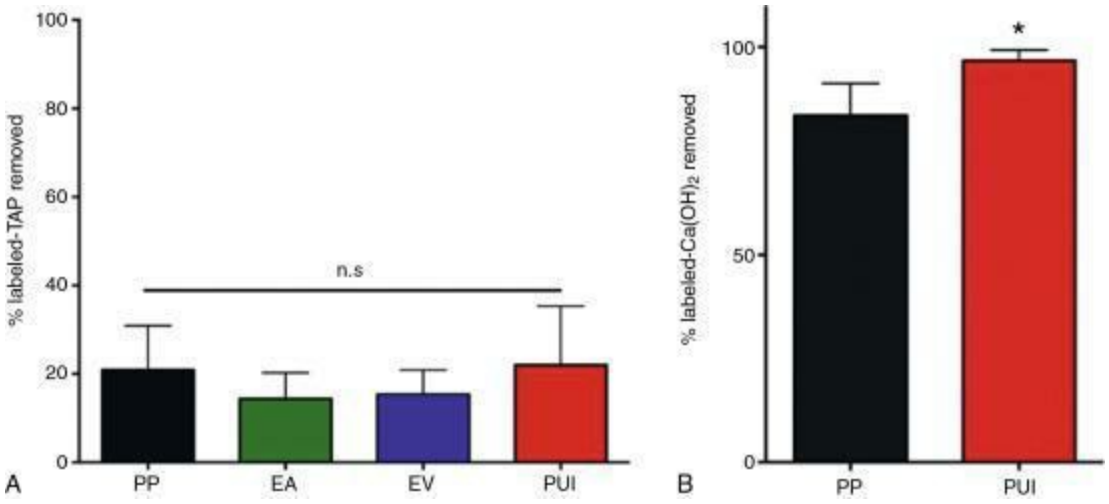

FIG. 12.11 TAP remains in dentin, whereas most $Ca(OH)_2$ is eliminated after endodontic irrigation. Radiolabeled TAP or $Ca(OH)_2$ was placed within canals of standardized root segments and incubated for 28 days at 37°C. The canals were flushed with standardized volumes of EDTA and saline using either positive pressure with a side-vented needle (PP) or positive pressure with ultrasonic activation of irrigants (PUI). There was no difference in labeled TAP removal among groups with only approximately 20% of the medicament being removed by the irrigation protocols (**A**). In contrast, >80% of $Ca(OH)_2$ was removed, with more efficient removal observed in canals irrigated with PUI (**B**). Data are presented as the mean percentage of total radiolabeled medicament removal ± standard error of the mean. *P <.05 tested by the Student t test (n = 12/group).

A) Plot of n.s against percentage labeled tripe antibiotic paste removed. The horizontal axis shows bars for PP, EA, EV and PUI. The vertical axis ranges from 0 to 100 in increments of 20.

The data is as follows:

PP: 20, EA: 12, Ev: 14 and PUI: 24

B) Plot of PP and PUI against percentage labeled calcium hydroxide removed. The horizontal axis shows bars for PP and PUI. The vertical axis ranges from 0 to 100 in increments of 50.

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The data is as follows:

PP: 80 and PUI: 95.

Source: (Modified with permission from Berkhoff JA, Chen PB, Teixeira FB, Diogenes A: Evaluation of triple antibiotic paste removal by different irrigation procedures, *J Endod* 40:1172, 2014.)

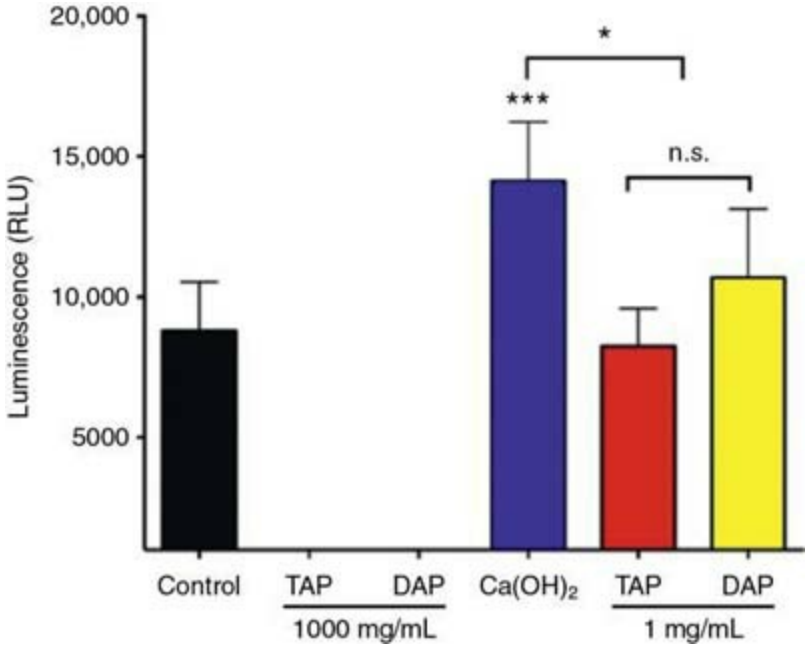

FIG. 12.12 Dentin conditioning for 7 days with medicaments used in REPs has a profound effect on SCAP survival. Standardized dentin disks were treated for 7 days with TAP or double antibiotic paste (DAP) (concentrations of 1000 mg/mL or 1 mg/mL), $Ca(OH)_2$ (Ultracal), or sterile saline (control). SCAP in a Matrigel scaffold (BD Biosciences; Bedford, MA) was seeded into the lumen of the disks after the medicaments were removed and cultured for 7 days. Cell viability (survival) was determined using a luminescent assay. SCAP culture on dentin treated with TAP or DAP at the concentration of 1000 mg/mL resulted in no viable cells. Conversely, dentin conditioning with TAP or DAP at the concentration of 1 mg/mL supported cell viability with no difference from untreated dentin disks (control). Greater survival and proliferation were detected in the group treated with $Ca(OH)_2$. Data are presented as mean ± standard deviation of relative luminescence units (n = 12/group). *P <.05; ***P <.001; n.s., no statistical difference as tested by 1-way analysis of variance.

Plot of control, triple antibiotic paste, double antibiotic paste, calcium hydroxide, triple antibiotic paste and double antibiotic paste against

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luminescence (RLU). The horizontal axis shows bars for control, double antibiotic paste, tripe antibiotic paste, calcium hydroxide, triple antibiotic paste and double antibiotic paste. The vertical axis ranges from 0 to 20000 in increments of 5000.

The data is as follows:

control: 7500, triple antibiotic paste (1000 milligram per milliliter): nil, tripe antibiotic paste (1000 milligram per milliliter): nil, calcium hydroxide: 14000, triple antibiotic paste: 7000 and double antibiotic paste: 11000.

Source: (From Althumairy RI, Teixeira FB, Diogenes A: Effect of dentin conditioning with intracanal medicaments on survival of stem cells of apical papilla, *J Endod* 40:521, 2014.)

Regardless of the intracanal medicament used, REPs have common features. Most of these published procedures reported minimal to no instrumentation.[77] This might be due, at least in part, to the concern of further weakening fragile dentinal walls and the difficulty of mechanically debriding canals of such large diameters. Due to the lack of mechanical debridement, clinicians relied on copious irrigation for maximum antimicrobial and tissue dissolution effects.[77] Canals were then medicated for a period that varied from days to several weeks. At the second visit, if signs and symptoms of diseased had subsided, the medicament was removed, the canal was dried, and intracanal bleeding was evoked in most, but not all, cases.[77] In most cases a coronal plug of MTA was placed over the blood clot or a collagen-based internal matrix, followed by a bonded coronal restoration.[173] Each of these steps will be discussed later in light of the current American Association of Endodontists (AAE) considerations for REPs.

Overview of clinical regenerative endodontic procedures

To date, most published clinical studies on REPs are composed of case reports and case series, and fewer retrospective cohort study[13,60,152,198,265,278] and prospective randomized clinical trials[185,211] on regenerative endodontics treatment are available. Although case series do not provide definitive evidence to support a given treatment modality, they do have the advantage of being conducted in actual patients and thus provide a higher level of evidence than preclinical studies. Although techniques for regenerative endodontics have varied in published case reports and case series,[77] there

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have been some consistent features worth noting. Nearly all reported cases involve patients 8 to 18 years old and teeth with immature apices with exception of published cases performed in mature, fully formed teeth.[132,151,209,252–255] Thus the patient age appears to be an important factor in case selection because some studies suggest that younger patients have a greater healing capacity or stem cell regenerative potential. Another important factor related to age is the stage of root development because the large diameter of the immature (open) apex may foster the ingrowth of tissue into the root canal space and may be indicative of a rich source of mesenchymal stem cells of the apical papilla (SCAP; see Figs. 12.2 and 12.3). These tissues are likely lacerated during the evoked-bleeding step and constitute a likely source of MSCs delivered into the root canal space. Indeed, a clinical study comparing outcomes in REPs found that greater root development was detected in the younger patient group, particularly with apices measuring more than 1 mm on the periapical radiographs.[90] Another consistent finding reported in nearly every case is the lack of instrumentation of the dentinal walls related to concerns about the potential fracture of these thin, incompletely developed roots. The lack of instrumentation would be expected to have the benefit of avoiding generation of a smear layer that could occlude the dentinal walls or tubules. On the other hand, the lack of instrumentation could result in remaining bacterial biofilms within dentinal tubes. This is particularly an issue in immature teeth due to large patent dentinal tubules that are more prone to bacterial penetration.[161] This is an issue that has not been evaluated in many cases, but the lack of canal wall instrumentation (and subsequent identification of bacteria in the apical dentin) has been suggested as a reason for failure of a regeneration case.[187] It is important to emphasize that these procedures are marked by robust disinfection protocols. Sodium hypochlorite, either alone or in combination with other irrigants, has been used to disinfect the canal space in most cases.[173] In a majority of cases, a combination of triple antibiotic (minocycline, metronidazole, and ciprofloxacin) was left in the canal space for a period of days to weeks, so the disinfection protocol was primarily a chemical method rather than the chemomechanical approach used in conventional nonsurgical endodontic therapy.[77] In most cases a blood clot was formed in the canal and might serve as a protein scaffold, permitting

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three-dimensional ingrowth of tissue.[77,173] However, it should be noted that few published cases included the use of PRP or platelet-rich fibrin (PRF)[148] and the use of exogenous scaffolds such as collagen type 1[211] and hydroxyapatite.[219]

Nearly all of these reports noted continued thickening of the root walls and subsequent apical closure. Examples of increased root length and thickening of root walls following REPs are seen in Figs. 12.13–12.16. Because of the lack of histology in most clinical cases, it should be recognized that radiographic findings of continued root wall thickness do not necessarily indicate that dentin was formed. Based on histologic results from preclinical studies, it is possible that the radiographic appearance of increased root wall thickness might be due to ingrowth of cementum, bone, or a dentinlike material. Histologic evidence from human extracted teeth following REPs suggests that regenerated pulp tissue may be in the canal space[263] and that the mineralized tissue along the dentinal walls appear to be cementum-like or osteodentin.[195,199,256] It should also be noted in some of the case reports that, although the teeth were nonresponsive to pulp testing, the authors suggested that vital tissue was identified in the apical portion of the canal space. In these cases, necrotic tissue was removed until bleeding was observed, then the canals were disinfected with antibiotic paste or $Ca(OH)_2$.[77] One could argue that by leaving vital pulp tissue in the apical segment of the canal, the resulting progression of root formation is more similar to apexogenesis than revascularization. Although the biologic process may differ between the revascularization and apexogenesis cases presented in these case reports, the goals of the procedures are similarly advantageous and significant. In both procedures, there is healing of the periradicular tissues and a progression of root development in a tooth that would otherwise have had a progression of pulpal and periradicular pathosis.

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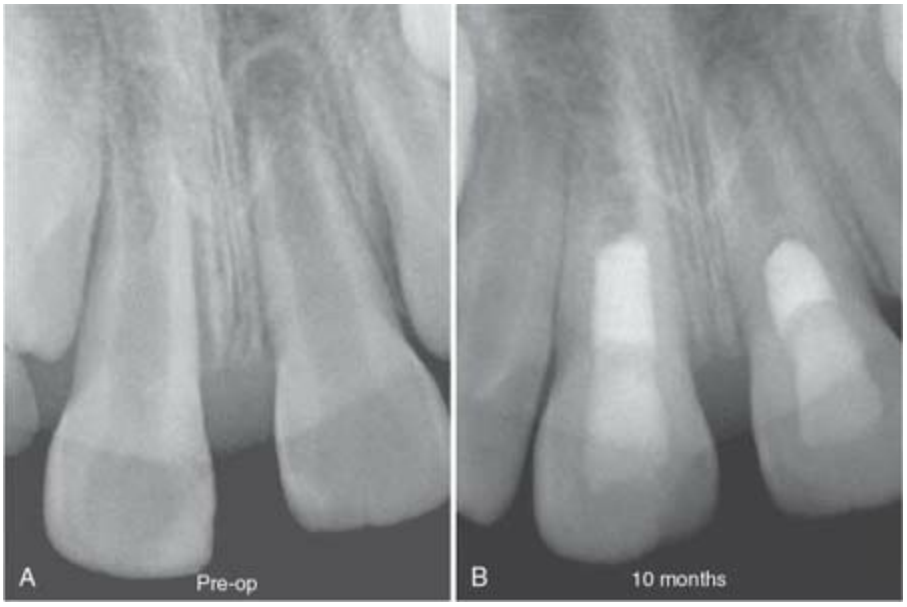

FIG. 12.13 Revascularization case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma, with a class 3 fracture in tooth #8 and a class 2 fracture in tooth #9 (**A**). The patient reported moderate to severe pain in both teeth. The teeth were isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 55 days. Upon recall, the teeth were isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in tooth #9 but not in tooth #8, where CollaCote was placed prior to mineral trioxide aggregate (MTA). The root canal systems were sealed with white MTA and a composite restoration (**B**).

A) Radiograph of a two teeth show dark crowns, necks and canals in each.

B) Radiograph of two teeth show radiolucent crowns, neck and radiopaque partial canals in each.

Source: (Courtesy Dr. Alan Law.)

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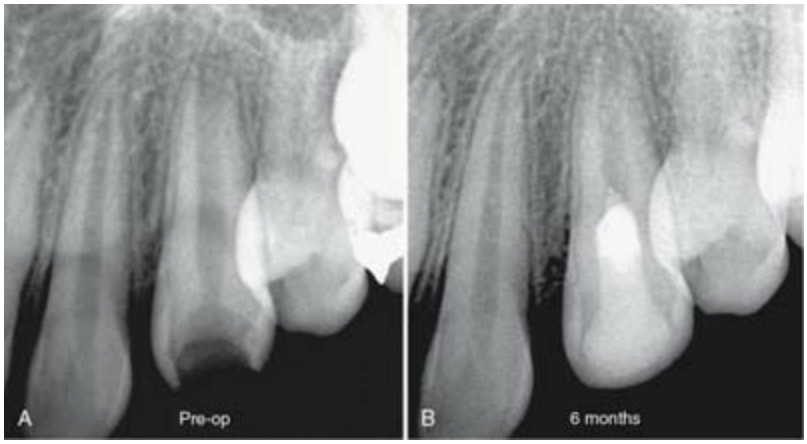

FIG. 12.14 Regenerative endodontic case illustrating treatment delivered to a 13-year-old female patient with a diagnosis of pulpal necrosis secondary to caries, with an unspecified prior history of trauma (**A**). The tooth was isolated, accessed, and irrigated with 5% sodium hypochlorite, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 21 days. Upon recall, the tooth was isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in both teeth, and the root canal system was sealed with white mineral trioxide aggregate and a composite restoration (**B**).

A) Radiograph of a tooth shows a missing crown, dark neck and canal. A radiopaque outline of a part of clamp is visible.

B) Radiograph of a tooth shows a radiolucent crown, and neck with radiopaque partial canal. A semicircular radiopaque patch is visible on the side surface of tooth.

Source: (Courtesy Dr. Alan Law.)

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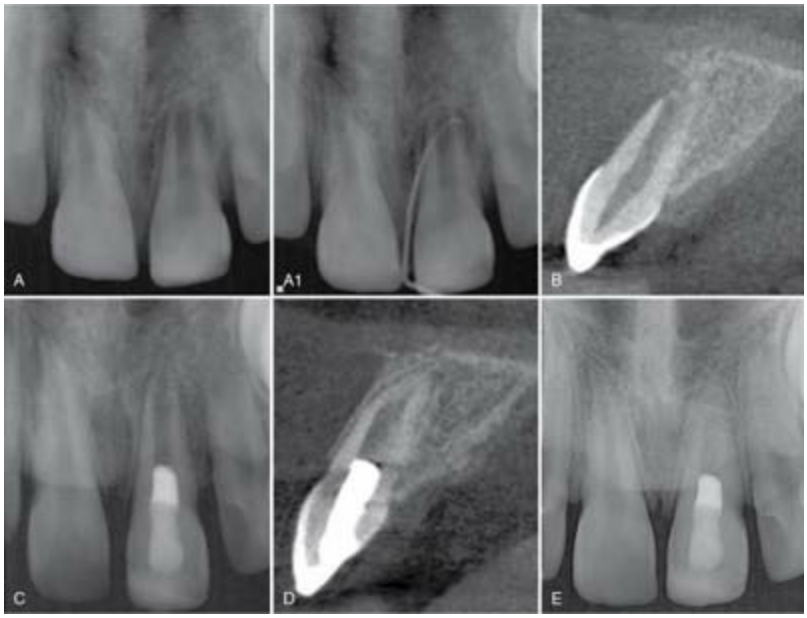

FIG. 12.15 Regenerative endodontic case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma and periradicular chronic apical abscess on tooth #9 seen on preoperative periapical radiograph (**A**) with sinus track tracing with a gutta-percha cone **(A1),** and with CBCT (**B**). The patient was asymptomatic and has draining facial sinus tract. The tooth was treated with a regenerative procedure using double antibiotic paste (DAP), a mixture of ciprofloxacin and metronidazole for 1 month. At the second appointment, the sinus tract had resolved. Then, the tooth was isolated, and the double antibiotic paste was removed by irrigation with 20 mL 17% EDTA. Intracanal bleeding was evoked from the apical tissues using a precurved #25 hand-file extending approximately 2 mm beyond the root apex. A Collaplug barrier was placed at the mid-root level, then covered with 3-mm white MTA. Fuji II LC glass ionomer was used as a coronal seal of the MTA. The access was then restored with a composite resin and polished (**C**). At the 1-year follow-up, the patient was asymptomatic, responsive to electrical pulp test (EPT), periodontal probings were no greater than 3 mm, and the tooth exhibited grade I mobility without coronal discoloration. Importantly there was appreciable root maturation in addition to resolution of apical radiolucency seen on CBCT (**D**) and periapical radiograph (**E**).

A) Radiograph of a tooth shows a radiolucent crown and neck with a dark canal.

A1) Radiograph of a tooth shows a radiolucent crown and neck with a dark canal. A radiolucent outline of a file is visible.

B) Radiograph of a tooth shows a dark crown and neck with a dark canal. A white radiopaque outline of double antibiotic paste around the pulp is visible.

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C) Radiograph of a tooth shows a radiolucent neck with a radiopaque partial canal.

D) Radiograph of a tooth shows a dark crown, neck and partial upper canal. A white radiopaque outline of double antibiotic paste around the pulp and inside the lower partial is visible.

E) Radiograph of a tooth shows a radiolucent neck with a radiopaque upper canal and radiolucent lower canal.

Source: (Courtesy of Dr. Obadah Austah, University of Texas Health Science Center at San Antonio.)

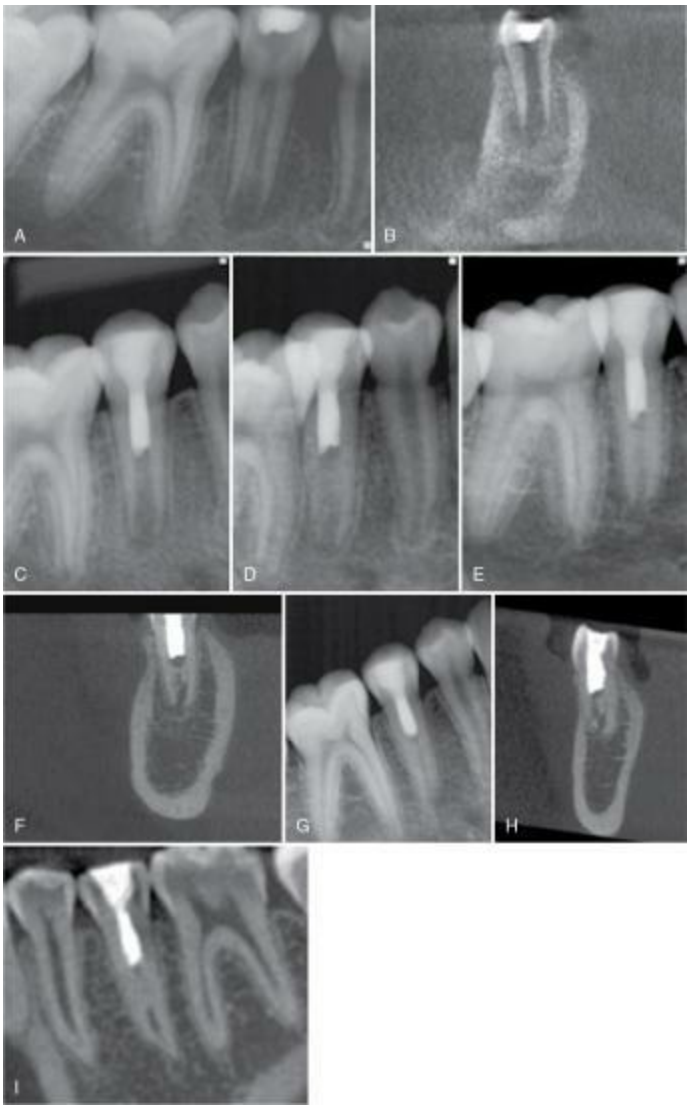

FIG. 12.16 Regenerative endodontic therapy case illustrating

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treatment delivered to a 9-year-old female patient with a diagnosis of previously initiated endodontic therapy (pulpectomy) due to pain from pulpal inflammation on #29. Bilateral examination revealed presence of a talon cusp (dens evaginatus) which may have been the etiology of pulpal inflammation on #29. Radiographic examination revealed large periapical radiolucency on a periapical radiograph (**A**) and cone-beam computed tomography (CBCT) (**B**). The tooth was treated with a regenerative procedure using double antibiotic paste as an intracanal medicament for 48 days. On the second visit, the tooth was isolated, and the double antibiotic paste removed by saline irrigation followed by a final rinse with 17% EDTA. The canal was dried and bleeding was evoked followed by placement of an internal matrix of Collaplug 3–4 mm below the CEJ. Mineral trioxide aggregate (MTA) was then placed over it and the tooth was restored with Fuji II LC and Build It. There was complete resolution of apical radiolucency on 1-month recall (**C**), appreciable root development at 5 months recall (**D**) and complete root maturation seen at 1-year recall on both periapical radiograph and CBCT (**E** and **F**, respectively). Greater root maturation is observed at 2.5 years recall periapical radiograph (**G**) and CBCT (**H** and **I**). In addition, the tooth responded to EPT on the 1- and 2.5-year recall visits.

A) Radiograph of a tooth shows a radiopaque part of crown with dark neck and canal.

B) Radiograph of a tooth shows a white radiopaque patch on the neck with radiolucent canals.

C) Radiograph of a tooth shows a radiopaque part of the crown with a radiopaque upper canal.

D) Radiograph of a tooth shows a radiopaque part of the crown with a radiopaque upper canal.

E) Radiograph of a tooth shows a radiopaque part of the crown with a radiopaque upper canal.

F) Radiograph of a tooth shows a white radiopaque upper canal with a circular scale in the gingival region below it.

G) Radiograph of a tooth shows a radiopaque part of the crown with a radiopaque regular upper canal.

H) Radiograph of a tooth shows a white radiopaque neck and upper canal with a circular scale in the gingival region below it.

H) Radiograph of a tooth shows a white radiopaque part of the crown, neck and upper canal with a dark middle canal and radiolucent lower canal.

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Source: (Courtesy Dr. Nikita Ruparel, University of Texas Health Science Center at San Antonio.)

A retrospective study has compared the radiographic changes in 48 revascularization cases to 40 control cases.[39] Although published revascularization cases have been treated with varying clinical protocols, they can be grouped by method of canal disinfection—namely, TAP, $Ca(OH)_2$ treatment, or formocresol treatment. This study applied a mathematic image-correction procedure that permitted the comparison of nonstandardized radiographs with subsequent statistical analysis of radiographic outcomes. The percent change in root dimensions was first compared in two negative control groups (nonsurgical root canal treatment [NSRCT] and MTA apexification) predicted to have little to no change in root dimensions.[39] This provides an internal test that the mathematic analysis was appropriate. The results indicate that these two negative control groups had minimal measured changes in root width (Fig. 12.17) or root length (Fig. 12.18), with the anticipated finding that instrumentation with files of greater taper resulted in a slight but detectable loss of apical root wall width. The results indicated that revascularization treatment with either the TAP or $Ca(OH)_2$ medicament produced significantly greater increases in root length compared with either the MTA or NSRCT control groups. Treatment with the TAP produced significantly greater increases in root wall thickness compared with the MTA and NSRCT control groups. Treatment with $Ca(OH)_2$ resulted in significantly greater change in root wall thickness compared with the NSRCT group, but no differences were observed between these medicaments and the MTA apexification group. Finally, the TAP produced significantly greater differences in root wall thickness compared with either the $Ca(OH)_2$ or formocresol groups. In general, the formocresol group showed the smallest improvement in root length and wall thickness. Secondary analyses indicated that a 12- to 18-month recall is probably the minimal time to judge radiographic evidence of root development, although later time points (36 months) often demonstrate continued root development. Jeeruphan et al. (2012) used a similar method to measure changes in root length and width following apexification versus revascularization.[152] They reported that teeth treated with revascularization showed a significantly greater percentage

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increase in root length (14.9%) compared with teeth treated by either MTA apexification (6.1%) or calcium hydroxide apexification. They also reported that the revascularization protocol produced significantly greater percentage increases in root width (28.2%) compared with teeth treated by either MTA apexification (0.0%) or calcium hydroxide apexification. In addition to measuring changes in root dimensions, the authors reported a survival (defined as retention of the tooth in the arch at the time of the postoperative recall) rate of 100% at an average of 14 months after revascularization treatment. This compared favorably with survival rates of 95% for MTA apexification and 77% for calcium hydroxide apexification. Although prospective randomized clinical trials with standardized radiographic assessment are clearly required, the results of this retrospective study are consistent with a robust outcome of revascularization procedures, particularly when medicated with either a TAP or a $Ca(OH)_2$ medicament.

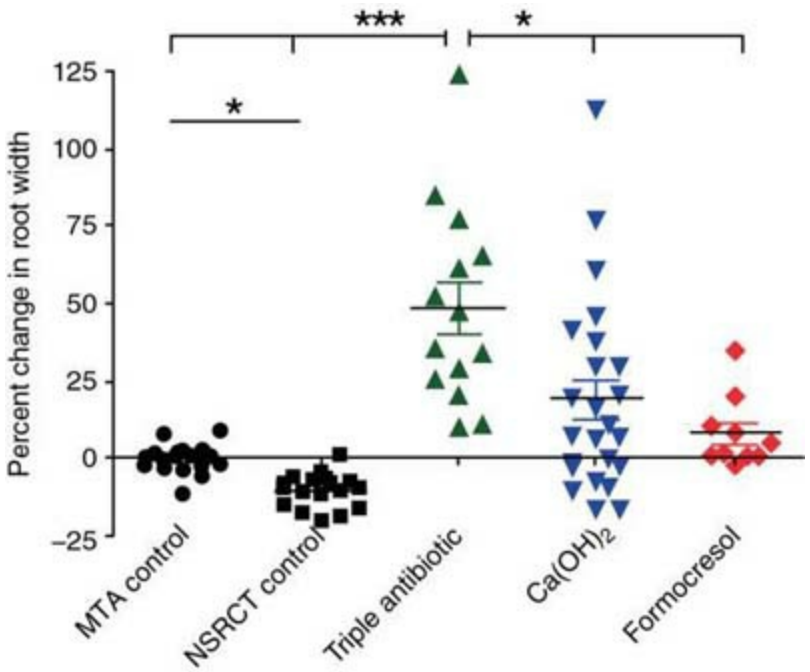

FIG. 12.17 Retrospective analysis of the percentage of change in dentinal wall thickness from preoperative image to postoperative image, measured at the apical third of the root (position of apical third defined in the preoperative image) in 40 control patients and in 48 patients following a revascularization procedure. *, *P* <.05 for each *P* <.05; ***, *P* <.001 versus mineral trioxide aggregate (MTA) apexification control group and NSRCT control group. *P* <.05 versus

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NSRCT control group only. P <.05 versus calcium hydroxide ($Ca[OH]_2$) and formocresol groups. P <.05 versus NSRCT control group only.

Plot of MTA control, NSRCT control, triple antibiotic, calcium hydroxide, formocresol against percent change in root width with a horizontal line marked from 0. The horizontal axis shows points for MTA control, NSRCT control, triple antibiotic, calcium hydroxide, formocresol. The vertical axis ranges from negative 25 to 125 in increments of 25.

The data is as follows:

MTA control: 9 points on the line, 8 points below the line and 2 points above the line.

NSRCT control: 1 point on the line, 16 points below the line and 0 points above the line.

Triple antibiotic: 0 points on the line, 0 points below the line and 15 points above the line.

Calcium hydroxide: 3 points on the line, 5 points below the line and 14 points above the line.

Formocresol: 5 points on the line, 0 points below the line and 4 points above the line.

Source: (From Bose R, Nummikoski P, Hargreaves K: A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures, *J Endod* 35:1343–1349, 2009.)

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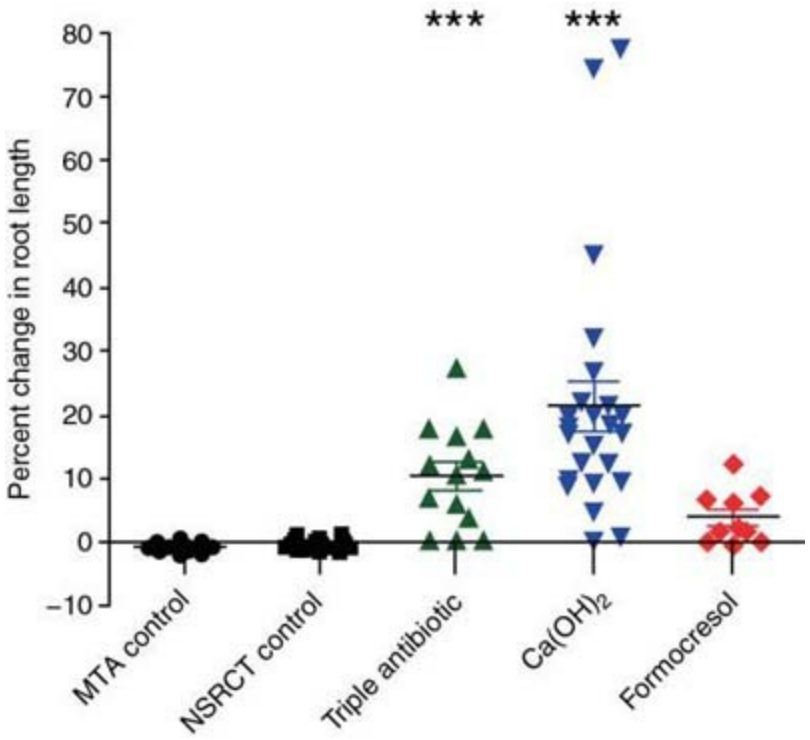

FIG. 12.18 Retrospective analysis of the percentage of change in root length from preoperative image to postoperative image, measured from the cementoenamel junction (CEJ) to the root apex in 40 control patients and in 48 patients following a revascularization procedure. ****P* <.001 versus mineral trioxide aggregate (MTA) apexification control group (n = 20) and NSRCT control group (n = 20). *P* <.05 versus MTA control group only. Median values for each group are depicted by horizontal line, and individual cases are indicated by the corresponding symbol.

Plot of MTA control, NSRCT control, triple antibiotic, calcium hydroxide, formocresol against percent change in root width with a horizontal line marked from 0. The horizontal axis shows points for MTA control, NSRCT control, triple antibiotic, calcium hydroxide, formocresol. The vertical axis ranges from negative 10 to 80 in increments of 10.

The data is as follows:

MTA control: 8 points on the line, 3 points below the line and 0 points above the line.

NSRCT control: 17 points on the line, 0 points below the line and 0 points above the line.

Triple antibiotic: 3 points on the line, 0 points below the line and 11 points above the line.

Calcium hydroxide: 2 points on the line, 0 points below the line and 22 points

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above the line.

Formocresol: 3 points on the line, 0 points below the line and 7 points above the line.

Source: (From Bose R, Nummikoski P, Hargreaves K: A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures, *J Endod* 35:1343–1349, 2009.)

Example of a revascularization protocol

Based on current research, several considerations can be reviewed when considering a protocol for revascularization treatment. The first issue is case selection; the best available evidence indicates that this treatment should be considered for the incompletely developed permanent tooth that has an open apex and is negative to pulpal responsiveness testing. Although the ultimate goal of this approach may include a tissue engineering–based method of pulpal regeneration in the fully developed permanent tooth, it should be recognized that current revascularization protocols have not been developed or evaluated for these more challenging cases. Informed consent should include the number of appointments (at least two), the possible adverse effects (primarily potential staining of the crown), the potential lack of response to treatment and alternative treatments, and possible posttreatment symptoms. Because the canal space will not be accessible following revascularization, teeth requiring retention in the canal space for the restoration are not good candidates for REPs. One reason for clinical staining of the crown and any root structure above the gingival margin appears to be due to the presence of minocycline.[169,235,241] This can be minimized by using a delivery system that restricts the drug below the cementoenamel junction (CEJ)[241] or the use of medication that does not contain minocycline such as double antibiotic mixture (ciprofloxacin and metronidazole) or calcium hydroxide. The use of MTA (ProRoot, Dentsply Tulsa Dental, Tulsa, OK), in both gray- and white-form variants,[33,43,48] may also cause tooth discoloration, which can also be reduced but not completely eliminated by a walking bleach technique.[43] Importantly, newer tricalcium silicate materials such as Biodentine and Endosequence BC Root Repair Material-Putty (ES) have

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substantially less discoloration potential, while promoting greater proliferation of SCAPs and differentiation into odontoblastic-like cells than MTA in an in vitro regenerative endodontics model.[200] This is particularly important because these nonstaining materials can be placed above the CEJ, favoring the formation of mineralized tissues in the cervical region of teeth, where ferrule and resistance to fracture are important factors. Importantly, because MTA promoted greater osteoblastic potential than the other tested biomaterials, it is recognized as the best-suited material to be used in MTA plugs and endodontic microsurgeries when bone formation and repair is the desired outcome.[200] Alternative treatments that should be discussed with the patient and guardian would include MTA apexification, no treatment, or extraction.

At the first appointment (Fig. 12.19, *A–E*), the treatment alternatives, risks, and potential benefits should be described to the patient and guardian after collecting clinical information and establishing pulpal and apical diagnoses. Following informed consent, the tooth is anesthetized, isolated, and accessed. Minimal instrumentation should be accomplished perhaps with the use of Hedstrom files circumferentially "brushing" the canal walls without major dentin removal. In addition, an appropriately sized file should be placed in the root canal system and radiographed to determine working length is important. Next, the root canal system should be copiously and slowly irrigated. Because of its proven efficacy as a canal disinfectant and a tissue dissolution agent, sodium hypochlorite (NaOCl) has been the irrigant of choice in most the revascularization case reports.[77] However, studies have demonstrated that treatment of dentin with NaOCl results in an indirect cytotoxic effect on stem cells.[194,300] Importantly, minimal deleterious effect was seen with the use of 1.5% NaOCl followed by 17% EDTA. Thus lower concentration (1.5%) of NaOCl followed by EDTA should be considered as the standard irrigation for REPs.[194] Moreover, the use of chlorhexidine should be limited or avoided because it does not have tissue dissolution capability and has also been shown to be cytotoxic to stem cells. However, if chlorhexidine is used, its cytotoxicity can be largely avoided by adding an inactivation step with a solution of L-alpha-lecithin followed by 17% EDTA.[310] Because canal disinfection relies considerably on chemical irrigants, it is important to place the needle into the apical third and irrigate

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using needles with a closed end and side-port vents (e.g., Max-I-Probe needles), together with a slow rate of infusion, to help to reduce any irrigants passing through the open apex. There is evidence that negative pressure can be beneficial and it should be considered in these procedures.[70] In addition, ultrasonic activation of solutions has been shown to promote greater tissue dissolution in addition to accelerating the release of growth factors from the dentinal walls.[312] Thus ultrasonic activation should be considered, particularly in the last visit for both sodium hypochlorite and the final EDTA irrigation.

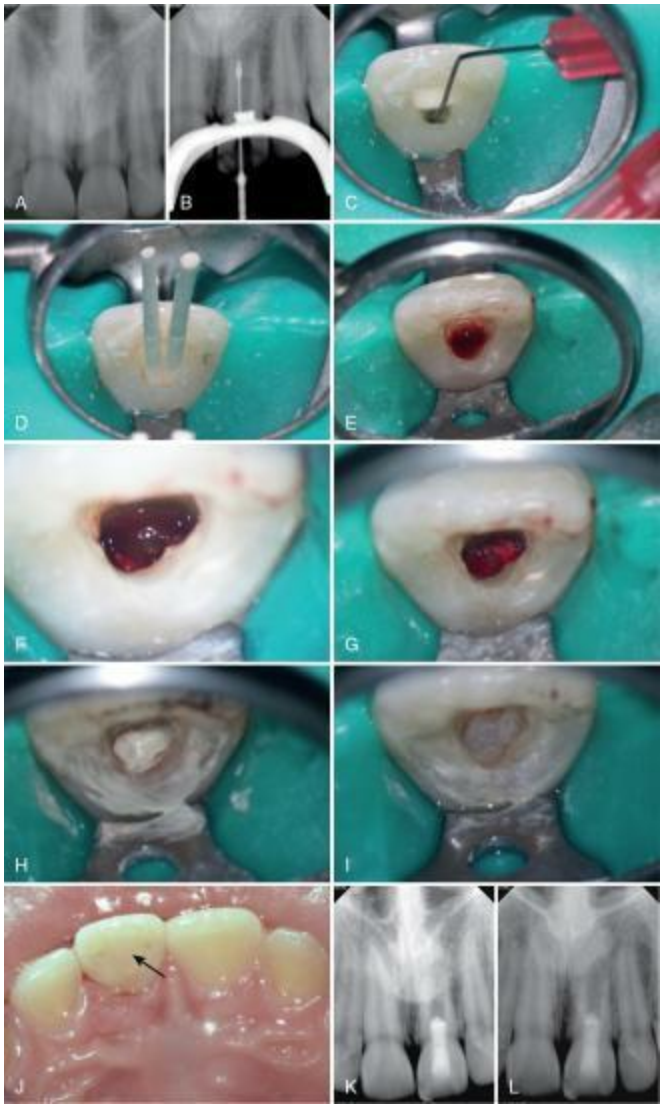

FIG. 12.19 Example of a revascularization case. Treatment was delivered to a 12-year-old boy who suffered trauma on tooth#9 2 years

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prior to the appointment. First appointment: (**A**), Clinical examination revealed a pain upon percussion and palpation. A working length file was placed into the root canal system (**B**). The tooth was slowly irrigated with 20 mL of 1.5% sodium hypochlorite (NaOCl) followed by 20 mL of saline using a Max-I-Probe needle inserted to the apical third (**C**). The canals were dried and medicated with calcium hydroxide paste (Ultracal) (**D**). The patient returned asymptomatic 1 month later. The tooth was isolated, accessed, and the medicament removed by slow irrigation with 1.5% NaOCl followed by 17% EDTA. After the canal was adequately dried, intracanal bleeding was achieved by lacerating the apical tissues (**E**). Blood clotting was observed after approximately 10 minutes **(F).** CollaPlug was placed below the CEJ (**G**) to serve as a matrix to position the white mineral trioxide aggregate coronal to the blood clot (**H**). The tooth was then sealed with a layer of Fuji IX (**I**), etched and restored with a composite (**J**). A final radiograph was taken (**K**). The tooth responded to electrical pulp tested at the 1-year recall, and demonstrated apical closure and moderate dentinal wall thickening (**L**).

A) Radiograph of a tooth shows a radiolucent crown with a dark canal.

B) Radiograph of a tooth shows an outline of a working file inserted inside the root canal. A radiopaque outline of a clamp is visible.

C) Close up of a tooth exposed over a paper dam held in a clamp shows an open crown with an angled file inserted inside it.

D) Close up of a tooth exposed over a paper dam held in a clamp shows two canal drying instruments inserted into the open crown.

E) Close up of a tooth held in a clamp shows bleeding clot inside the open crown.

F) Close up of a tooth shows blood clot inside the open crown.

G) Close up of a tooth shows a dried blood clot inside the open crown.

H) Close up of a tooth held in a clamp shows a layer of core build up paste inside the open crown.

I) Close up of a tooth held in a clamp shows a layer of core build up paste inside the open crown.

J) Close up of the posterior set of teeth shows an arrow pointing to the surface of the central incisor.

K) Radiograph of a tooth shows a radiolucent crown with radiopaque neck and upper canal.

L) Radiograph of a tooth shows a dark crown with radiolucent neck and upper

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canal.

Source: (Courtesy Dr. Anibal Diogenes, University of Texas Health Science Center at San Antonio.)

The root canal system is then dried with sterile paper points, and the antimicrobial medicament is delivered into the root canal space. The best available evidence would support the use of either an antibiotic mixture (TAP or double antibiotic paste [DAP]) at the concentration of 1 to 10 mg/mL or $Ca(OH)_2$. TAP and $Ca(OH)_2$ have been shown to be effective (see Figs. 12.13 and 12.14). The TAP has the advantage of being a very effective antibiotic combination against endodontic microorganisms; its efficacy is supported by its use in many published cases.[77,173] However, this combination is not approved by the FDA and carries a potential for minocycline staining of the crown. Thus, when performing REPs on teeth in esthetic areas, practitioners should consider the use of calcium hydroxide or eliminating minocycline from the antibiotic paste, sealing the coronal dentin with a dentin-bonding agent or composite. After antimicrobial medicament is placed, the tooth is then sealed with a sterile sponge and a temporary filling (e.g., Cavit), and the patient is discharged for 3 to 4 weeks.

At the second appointment (see Fig. 12.19, *F–M*), the patient is evaluated for resolution of any signs or symptoms of an acute infection (e.g., swelling, sinus tract pain) that may have been present at the first appointment. The antimicrobial treatment is repeated if resolution has not occurred. In most reported cases, the acute signs and symptoms resolved after treatment with the intracanal medicament.[77] Because revascularization-induced bleeding will be evoked at this appointment, the tooth should not be anesthetized with a local anesthetic containing a vasoconstrictor. Instead, 3% mepivacaine can be used, which will facilitate the ability to trigger bleeding into the root canal system. Following isolation and reestablishment of coronal access, the tooth should be copiously and slowly irrigated, possibly together with gentle agitation with a small hand file to remove the antimicrobial medicament, followed by ultrasonic activation of the irrigant with the tip 2 to 3 mm away from the apical foramen. When choosing an irrigant at the second appointment, it is worth considering irrigants such as NaOCl and chlorhexidine may be cytotoxic to stem cells either directly[89] or indirectly

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after dentin has been exposed to the irrigants.[194,300,310] It has been demonstrated that exposure of dentin to 5% to 6% sodium hypochlorite leads to decreased stem cell survival and odontoblastic differentiation. This indirect effect is likely related to various deleterious effects of sodium hypochlorite on the dentin matrix leading to reduced cell attachment[242] and a decrease in dentin matrix-derived growth factors such as TGF-β1.[270,332] Thus it is prudent to continue to use sodium hypochlorite at the concentration of 1.5% on the second visit. Lastly, irrigation with 17% EDTA may be advantageous because it has been shown to release growth factors from dentin and promote stem cell survival and differentiation.[312,332] In addition, Galler et al. (2011), using dentin cylinders transplanted into immunocompromised mice, showed that DPSCs seeded onto EDTA-conditioned dentin differentiated into odontoblast-like cells and had cellular processes extending into the dentin.[106] On the other hand, stem cells differentiated into osteoclasts/odontoclasts, causing resorption on the dentinal walls if dentin was previously treated with 5% sodium hypochlorite. Thus the use of 17% EDTA as the last irrigant promotes attachment, proliferation, and odontoblastic differentiation of stem cells that could further increase with the use of passive ultrasonic irrigation.[312]

After drying the canal system with sterile paper points, a file is placed a few millimeters beyond the apical foramen, and the apical tissue is lacerated with bleeding preferably coronal to the CEJ. A small piece of Colla-Plug (Zimmer Dental, Carlsbad, CA) may be placed over the clot to serve as an internal matrix and prevent the restorative materials from being displaced into the canal space upon placement. Approximately 2 to 3 mm of a bioactive hydraulic cement such as Biodentine, ES, or MTA should be placed as coronal as possible while leaving enough chamber space for a glass ionomer and composite restoration. Although MTA has been used in many of the case reports and may have many desirable properties,[230,231,294,298] it is associated with severe discoloration, making it unsuitable to be used in these procedures.[160,172,174,180] Alternatively, Biodentine or ES putty could be used because there have been encouraging studies demonstrating that they induce stem cell proliferation and odontoblastic differentiation.[178,192,200,328] Indeed, Biodentine[11,282,293] and ES putty[45] have been successfully used in REPs and appear better suited materials due to the reduced risk of discoloration.

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In addition to follow-up visits in the first several months after REPs to assess for signs of healing, a 12- to 18-month recall should be considered as a reasonable time point to radiographically assess apical healing and root development.[39]

The following box summarizes the steps in a REP. Importantly, these considerations are based on the best available evidence and are likely to change as the field progresses (Box 12.1).

Box 12.1

Treatment Procedures for Regenerative Endodontics

First treatment visit for regenerative endodontics:

1. Informed consent, including explanation of risks and alternative treatments or no treatment.
2. After ascertaining adequate local anesthesia rubber dam isolation is obtained.
3. The root canal systems are accessed and working length is determined (radiograph of a file loosely positioned at 1 mm from root end).
4. The root canal systems are slowly irrigated first with 1.5% NaOCl (20 mL/canal, 5 min) and then irrigated with saline (20 mL/canal, 5 min), with irrigating needle positioned approximately 1 mm from root end.
5. Canals are dried with paper points.
6. Calcium hydroxide or an Antibiotic Paste or solution (combined total of 1–10 mg/mL) is delivered to canal system
7. Access is temporarily restored

Final (second) treatment visit for regenerative endodontics (Typically 2–4 weeks after the first visit):

1. A clinical exam is first performed to ensure that that there is no moderate to severe sensitivity to palpation and percussion. If such sensitivity is observed, or a sinus tract or swelling is noted, then the treatment provided at the first visit is repeated.
2. After ascertaining adequate local anesthesia with 3% mepivacaine (no

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epinephrine) rubber dam isolation is obtained.
3. The root canal systems are accessed; the intracanal medicament is removed by irrigating with 17% ethylenediaminetetraacetic acid (EDTA) (30 mL/canal, 5 min) and then a final flush with saline (5 mL/canal, 1 min).
4. The canals are dried with paper points.
5. Bleeding is induced by rotating a precurved K-file size #25 at 2 mm past the apical foramen with the goal of having the whole canal filled with blood at least to the level of the cementoenamel junction, but preferably coronal to it.
6. Once a blood clot is formed, a premeasured piece of Collaplug (Zimmer Dental Inc., Warsaw, IN) is carefully placed on top of the blood clot, preferably above the cementoenamel junction, to serve as an internal matrix for the placement of approximately 2 mm of a tricalcium silicate biomaterial such as Biodentine (Septodont) or Endosequence BC Root Repair Material-Putty (ES) (Brasseler).
7. A (3 mm) layer of glass ionomer layer (e.g., Fuji IX, GC America, Alsip, IL; or other) is flowed gently over the bioactive coronal barrier and light cured for 40 s.
8. A bonded reinforced composite resin restoration is placed over the glass ionomer.
9. The case needs to be followed-up at 3 months, 6 months, and yearly after that for a total of 4 years.

Clinical measures of treatment outcomes

The goal of conventional endodontic therapy is to maintain or restore the health of periradicular tissues by preventing or healing apical periodontitis. The goals of regenerative procedures extend beyond the goals of conventional endodontic therapy and include continued root development and reestablishment of pulpal vitality while aiming to achieve the functional recapitulation of a healthy pulp-dentin complex. As mentioned earlier, contemporary REPs are being modified and evolving towards accomplishing these additional objectives that are not achievable with conventional nonsurgical root canal therapy. Accordingly, the definition of success for REPs includes multilevel goals that are not mutually exclusive. This

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multilevel approach should be focused on three levels of outcomes: patient-centered, clinician-centered, and scientist-centered outcomes (Fig. 12.20).

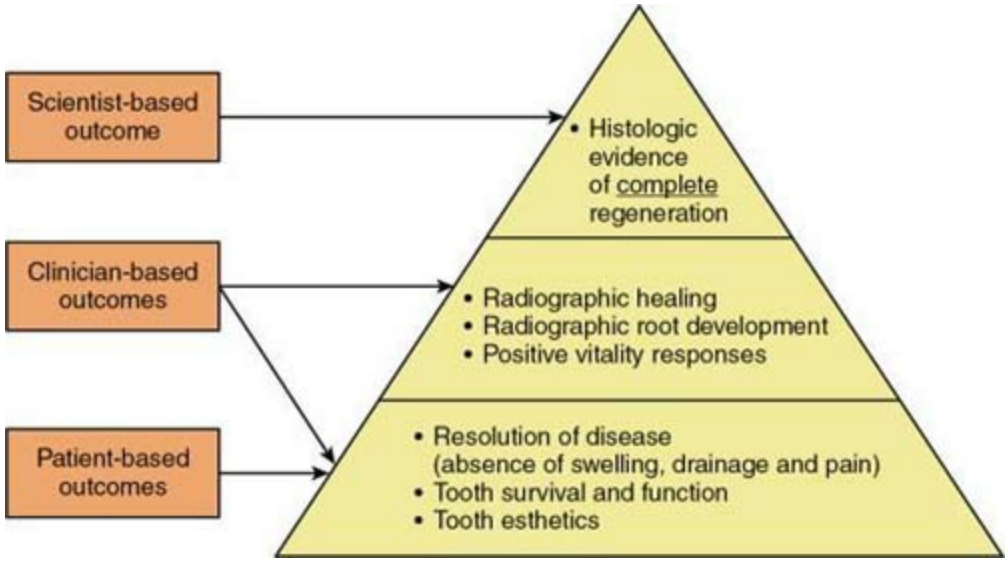

FIG. 12.20 Trilevel outcome assessment pyramid. Regenerative endodontic procedures must be assessed and evaluated in a systematic manner, acknowledging that there are different levels of possible outcomes that carry different meanings and value for patients, clinicians, and scientists. The patient-centered outcomes represent the base of this pyramid, symbolizing the fundamental importance of achieving healing, reestablishment of function, and patient satisfaction and overall well-being.

A three-level pyramidal diagram shows different levels of possible outcomes. Scientist-based outcome leads to histologic evidence of complete regeneration, clinician-based outcomes lead to radiographic healing, radiographic root development and positive vitality responses; and patient-based outcomes lead to resolution of disease (absence of swelling, drainage and pain), tooth survival and function and tooth esthetics.

Source: (Modified from Diogenes A, Ruparel NB, Shiloah Y, Hargreaves KM: Regenerative endodontics: a way forward, *JADA* 147(5):372–380, 2016.)

Patient-centered outcomes

It is well established that the primary goal of endodontics is the prevention and healing of pulpal and periradicular inflammation (i.e., resolution of signs and symptoms of disease). Unfortunately, there has been too much emphasis

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on radiographic assessment while often ignoring the benefit and improvement in the quality of life given to the patient. Clinical studies focusing on patient-centered outcomes in endodontics report that the greatest benefit of endodontic therapy was alleviation of pain and psychological discomfort[202] and improvement in quality of life.[129] Unfortunately, a tooth could be considered a failed treatment if any radiographic finding is detected by a clinician despite the benefit that the treatment has brought to the patient. Not surprising, the definition of clinical success has been a controversial topic in endodontics throughout the history of the specialty, but it has been largely based on lack of symptoms and a strict radiographic criteria,[34,35,102,281] or even dependent on the absence of cultivable microorganisms.[47] This radiographic criteria of success have been present in endodontics for decades and exemplify the disconnect between the expectations of patients, clinicians, and scientists. This disconnect is not unique to endodontics but found in all fields of dentistry and medicine. Patients are the primary stakeholders of their own well-being status, providing feedback on treatment effectiveness, and thus advancing the development of therapies that promote the overall population health. For patients undergoing regenerative procedures, their primary expectation is that teeth are disease free (i.e., no symptoms) and functional (i.e., survival) and have acceptable esthetics.[78]

A recent metanalysis of published REPs and apexification procedures revealed that the success defined as healing of apical periodontitis was found in approximately 91% and 95% for REPs and apexification procedures without statistically significant difference between the two treatment modalities.[297] Likewise, there was no difference in survival rate between REPs and apexification procedures, 98% and 97%, respectively. As pointed out in this metanalysis, most included studies were case series and retrospective studies. Additional randomized clinical trials with large sample sizes and adequate follow-up (>2 years) and recall rates are required to improve the quality of the pooled data. It is important to highlight that this lack of higher-quality studies is not restricted to REPs, but it is also true for apexification procedures. Therefore so far there have not been studies that demonstrated a meaningful difference between REPs and apexification in healing of apical periodontitis and survival rates, with the latter impacted by the often-found short follow-up periods in the current published studies.

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Collectively, currently published studies support that both procedures demonstrate equally acceptable clinical outcomes for healing and survival addressing two of the three patient-centered outcomes.

Esthetics is considered a patient outcome because it is a personal perception. Tooth discoloration has been identified as an adverse event associated with many published REPs.[160,218,235] Tooth discoloration has two primary sources in these procedures: intracanal medicament and coronal barrier material. As discussed previously in this chapter, the undesirable staining that has marked the initial generations of these procedures can be greatly minimized, or completely eliminated, with the use of medicaments that do not contain minocycline and alternative tricalcium silicate biomaterial with lesser staining risk than MTA, such as Biodentine or ES, in esthetic zones.[49,167] In addition, the use of a dentin-bonding agent to seal the dentinal tubules in the pulp chamber is beneficial also to minimizing any possible discoloration from the coronal plug material.[6,169] Therefore a clinician's choice of medicaments and materials has the potential to greatly impact this important patient-centered outcome.

Clinician-centered outcomes

Because the well-being of our patient is the paramount goal of every clinician, we must first ascertain that the patient-centered outcomes (i.e., no symptoms, functional survival, and acceptable esthetics). In addition, clinicians use objective and subjective clinical testing to assess radiographic evidence of healing and continued root development and responses to sensibility tests (i.e., vitality testing).

Continued root development is another desirable outcome of REPs. This is normally evaluated by the clinician upon radiographic evaluation. A total of four possible radiographic outcomes regarding root development can be found (Fig. 12.21). These outcomes range from: type 4—no change; type 3—closing of apical opening without change in elongation/thickening; type 2—root elongation/thickening without closure of the apical opening; and type 1—elongation/thickening and closure of the apex (most desirable outcome). Many published case reports and case series provided initial subjective evidence that REPs promoted radiographic healing and continued root development, until a retrospective study used a quantitative methodology to

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assess root development, comparing to apexification procedures. In this initial study, REPs were found to promote significantly greater root elongation and thickening compared with no changes seen with apexification procedures.[39] The methodology used in this study was adopted by other studies finding similar results.[13,153,159,211] Furthermore, this methodology was later revised and modified to assess changes in radiographic root area.[97] Despite being designed to digitally align radiographs taken in a nonstandardized manner to allow for quantitative measurements, there are instances in which the discrepancy in angulation and acquired image dimension ratios exceed the capacity of the software to perform adequate correction.[159] As a viable alternative, volumetric analysis of acquired cone-beam computed tomography (CBCT) images has been proposed[91] as a method to three-dimensionally quantify changes in root development. This methodology has been used in two studies, both demonstrating that REPs promoted an increase in total root volume.[24,198] A recent randomized prospective study used acquired CBCT volumes to quantify and characterize the type of development detected in cases treated with REPs (n = 69) and apexification procedures (n = 34). In this study, REPs promoted the average root growth of greater than 0.24 mm in approximately 82.6% of the teeth, whereas teeth treated with apexification had no changes in root development. Interestingly, teeth with dental anomaly (i.e., dens invaginatus) as etiology of disease showed more desirable outcome patterns (type 1 and type 2; see Fig. 12.21), whereas trauma was associated with less development and greater incidence of the less desirable type 4 and type 3 outcome patterns (see Fig. 12.21). These data agree with previous case series and retrospective studies that included trauma as the main etiology for less desirable outcomes[13,257] compared with other studies that included less trauma cases.[153] Therefore these studies suggest that trauma negatively affects root development following REPs. Even though more studies are required to establish the correlation of trauma with less desirable outcomes in REPs, these observations agree with different lines of evidence demonstrating that development can be halted if trauma disrupts the interaction between the apical papilla and the HERS.

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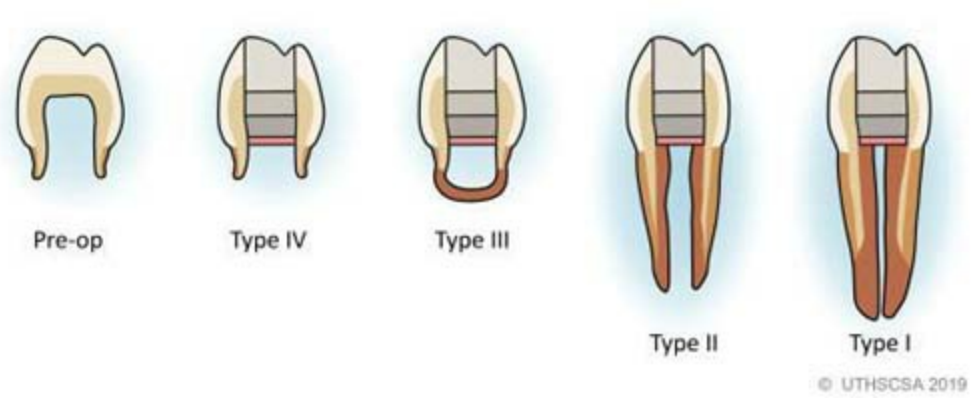

FIG. 12.21 Schematic illustrating the different possible outcomes in root development following regenerative endodontic procedures. Type I shows root development and narrowing of the apical foramen. Type II shows root development but failure of apex closure. Type III shows no root development but closure of the apical foramen. Type IV shows no developments and failure to apex closure.

Lateral diagrams of tooth show five stages of root development as labeled from pre-operative stage to type one.

Vitality responses have been reported in approximately 50% of the published cases.[77] Responses to electric pulp tester (EPT) are more commonly reported than cold responses. These responses to vitality testing (with either cold or EPT), as well as the lack of signs and symptoms of pathosis, suggest the presence of functioning tissue in the canal space. Vitality responses, in addition to continued root development, are a very desirable outcome because "normal" nociception is protective and suggests immune competence of the vascularized tissue. However, the lack of response to sensitivity tests in absence of signs and symptoms of disease should not be interpreted as failure, because there have been several reports of cases demonstrating healing of apical periodontitis and appreciable root development in the absence of a positive response to vitality testing. Histologic examination of teeth treated with REPs has revealed the presence of nerve fibers in a well-vascularized loose connective tissue.[179,199] A recent histologic study of a tooth treated successfully with REPs revealed that this innervation includes nociceptors, thus lending evidence that functional free nerve endings are regenerated following these procedures.[24] These free nerve endings are attracted to the "regenerated" pulp space by a process called axonal targeting that is likely mediated by cellular and noncellular

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processes.[25,72,88]

The significance of reinnervation is the newly formed tissues within the canal space likely transcend the expected physiologic responses to noxious stimuli and infections. It is known that several species of amphibians including salamanders have the capacity of full limb regeneration. Interesting, this amazing regenerative capacity, which has been studied by researchers for decades, is completely lost if the limb is denervated.[92] This concurrent loss of innervation and regenerative potential suggests that peripheral nerves have a pivotal role in regeneration. Further research is required to establish regenerative role in the pulp-dentin complex. Nonetheless, the appropriate innervation of the newly formed pulplike tissue must represent a major goal of these procedures. Lastly, the presence of reinnervation may not always be detected with sensitivity tests that are prone to false-positive and false-negative responses[182] in addition to variations in the thickness and apical extent of coronal barrier and restorations.

Scientist-based outcomes: Regeneration or repair?

Scientific outcomes that are not directly related to the well-being of the patient or the clinical success according to clinician-based outcome measures can be of high importance. These outcomes require scientific methodology to be evaluated and focus on important scientific questions, promoting further research and bring advancements to the field. A good example of a scientist-based outcome is the nature of the tissue formed, including the identification of cellular components, formed following REPs. Undoubtedly, there is considerable debate on whether REPs promote true regeneration or repair.[268] This has strong scientific merit, but it is directly related to the clinical success of a case. Histologic evaluation of teeth that were previously treated with REPs but later extracted due to recurring trauma and fractures suggests that the newly formed tissue does not resemble the lost dental pulp, and formed tissues include ectopic bone, cementum, and periodontal ligament, in addition to commonly found components of the dental pulp complex such as fibroblasts, blood vessels, and nerves.[30,195,199,264]

A recent histologic study evaluated the scientist-based outcomes of two REP cases with different clinical outcomes.[24] One case was considered a clinical failure due to recurrent caries and resorption, despite the noticed

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continued root development resulting in extraction and histologic examination, which revealed the presence of inflammatory infiltrate absence of odontoblast-like cells and the predominant presence of cementum and bone as newly mineralized tissues. The other case was considered a clinical success for 6 years and was extracted only for orthodontic reasons. In addition to the typical ectopic mineralized tissues, odontoblast-like cells were found and newly formed tubular dentin (Fig. 12.22).[24] In addition, this study characterized the innervation found in the tissue as containing peptidergic nociceptors (i.e., expressing calcitonin gene related peptide) (Fig. 12.23). This is the first study that used laser confocal microscopy to identify cellular components and mineralized matrix in teeth treated with REPs.[24] The results of this study are surprising and suggest that, when sophisticated methods are used, further characterization can be achieved, revealing that at least a partial regeneration of components of the pulp-dentin complex is possible. It has been suggested that the current scientist-based status of REPs could be described as guided endodontic repair (GER) because the tissues are not fully regenerated but demonstrate the result of an intentional and directed repair that was not previously thought possible.

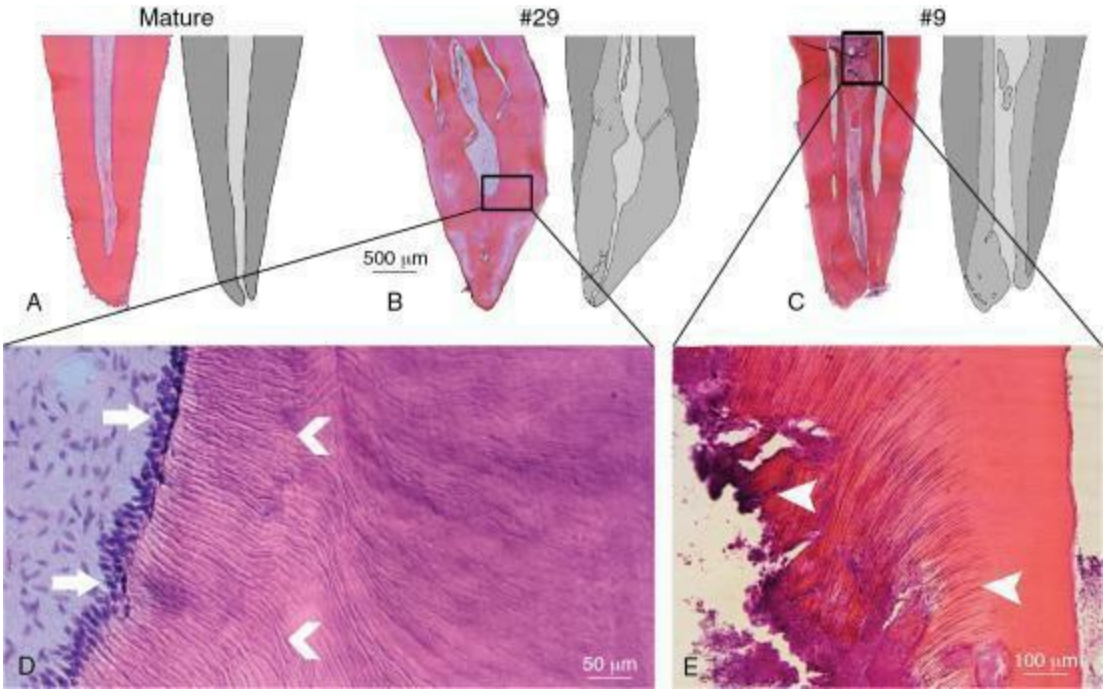

FIG. 12.22 (**A–C**) Representative hematoxylin-eosin staining of longitudinal sections of a healthy mature tooth #28, tooth #29, and

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tooth #9. (**D**) An enlarged section from **B** demonstrating mature, secretory odontoblasts lining newly formed tubular dentin *(arrows)*. (**E**) An enlarged section from **C** showing the presence of biofilm colonies attached and penetrated into dentinal tubules of tooth #9 *(arrows)*.

A) Colored specimen of mature tooth number 28 show a red pulp with pink canal. Black and white specimen shows a dark gray pulp with light gray canal.

B) Specimen of tooth number 29 shows a red irregular pulp with deformed pink canal with pigments of sea green color. Black and white specimen shows dark gray outlines of tooth with a light gray pulp and lighter gray deformed canal.

C) Specimen of tooth number 9 shows a red irregular pulp with outlines of grey and deformed pink canal with pigments of black color. Black and white specimen shows dark gray outlines of tooth with a light gray pulp with enlarged end and lighter gray canal with pigments of light gray.

D) Micrograph shows regions of scatter odontoblasts and lateral textured dentin. Two arrows point out to the odontoblasts clustered along the line of boundary.

D) Micrograph shows bacteria visibly clustered along the boundary line of lateral textured dentinal tubule with two arrows pointing to the bacteria entering the tubules.

Source: (From Austah O, Joon R, Fath WM, et al: Comprehensive characterization of 2 immature teeth treated with regenerative endodontic procedures, *J Endod* 44(12):1802–1811, 2018.)

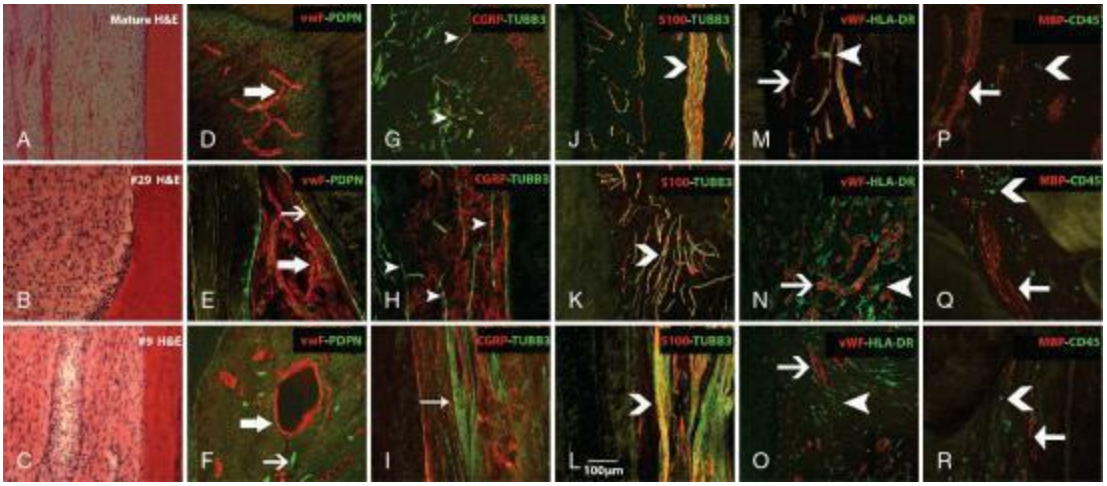

FIG. 12.23 (**A–C**) Representative hematoxylin-eosin staining of sections of a healthy mature tooth #28, tooth #29, and tooth #9. (**D–F**)

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Immunohistochemical staining showing von Willebrand factor and podoplanin (PDPN) immunoreactivity in a healthy mature tooth #28, tooth #29 and tooth #9. (**G–I**) Immunohistochemical staining showing colocalization of calcitonin gene–related peptide (CGRP) and class III beta-tubulin (TUBB3) in a healthy mature tooth #28 and tooth #29. (I) Tooth #9 shows TUBB3 immunoreactivity but no colocalization with calcitonin gene–related peptide. (**J–L**) Immunohistochemical staining demonstrating colocalization of Schwan cell marker (S100) with TUBB3 in a healthy mature tooth #28, tooth #29, and tooth #9. (**M–O**) Immunohistochemical staining showing close proximity of blood vessels stained with von Willebrand factor and a marker for antigen-presenting cells (human leukocyte antigen–antigen D related; HLA-DR)) in a healthy mature tooth #28, tooth #29, and tooth #9. (**P–R**) Immunohistochemical staining demonstrating close proximity of blood vessels stained with von Willebrand factor and a pan-leukocytic marker (CD45) in a healthy mature tooth, tooth #29, and tooth #9. *Arrows* indicate positive expression of the various proteins.

A) Mature H and E: Micrograph shows laterally divided plain and pigmented region. Pigments of black color are visible in the left region with lateral transopaque patches.

B) Number 29 H and E: Micrograph shows curvature division between plain and pigmented region. Pigments of black color are visible in the left region with cluster of pigment lining the boundary.

C) Number 9 H and E: Micrograph shows laterally divided region with partial pigmentation of black color and completely pigmented region. Pigments of black color are visible in the left region with lateral transopaque rod-shaped patch.

D) vwF PDPN: Colocalized specimen shows a pigmented surface with arrow pointing to pigmented red rod-like calcitonin gene–related peptide (podoplanin (PDPN)).

E) vWF-PDPN: Colocalized specimen shows a pigmented A-shaped surface with arrows pointing to the outlines of green pigment and circular disk of red pigment inside it.

F) vWF-PDPN: Colocalized specimen shows a pigmented surface with arrows pointing to the pigments of green and circular disk of red pigment with black center.

G) CGRP-TUBB3: Colocalized specimen shows a green pigmented surface with arrow pointing to the red and green pigments.

H) CGRP-TUBB3: Colocalized specimen shows a pigmented surface with arrow pointing to the red pigments and vertically sectioned green pigments.

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I) CGRP-TUBB3: Colocalized specimen shows a pigmented surface with arrow pointing to the vertical sections of red and green pigments.

J) S100-TUBB3: Colocalized specimen shows pigmented surface with arrow pointing to the multicolored vertical section of red, green and yellow pigments.

K) S100-TUBB3: Colocalized specimen shows pigmented surface with arrow pointing to the vertical lines of multicolored section of red, green and yellow pigments.

L) S100-TUBB3: Colocalized specimen shows pigmented surface with arrow pointing to the vertical multicolored sections of red, green and yellow pigments.

M) vWF-HLA-DR: Colocalized specimen shows pigmented surface and sections of multicolored pigments with arrow pointing to the red and green pigments.

N) vWF-HLA-DR: Colocalized specimen shows pigmented surface with arrow pointing to the red and green pigments.

O) vWF-HLA-DR: Colocalized specimen shows pigmented surface with arrow pointing to the ellipsoidal outline of red pigments and vertical segments of green pigments.

P) MBP-CD45: Colocalized specimen shows pigmented surface with arrow pointing to the curved section of red pigments and scattered green pigments.

Q) MBP-CD45: Colocalized specimen shows pigmented surface with arrow pointing to the vertical segments of red pigments and scattered segments of green pigments.

R) MBP-CD45: Colocalized specimen shows pigmented surface with arrow pointing to the scattered green and red segments.

Source: (From Austah O, Joon R, Fath WM, et al: Comprehensive characterization of 2 immature teeth treated with regenerative endodontic procedures, *J Endod* 44(12):1802–1811, 2018.)

Summary

The field of regenerative endodontics is rapidly advancing. This progress is based on the principles of tissue engineering, namely the spatial delivery of appropriate cells, scaffolds, and growth factors. Similar to most rapidly developing fields, the preclinical area of research has outpaced translational

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clinical studies. Preclinical studies have identified several MSC sources capable of differentiating into odontoblast-like cells, as well as candidate scaffolds and growth factors capable of guiding this development. The initial preclinical animal studies indicate that using all three components of the tissue-engineering triad (stem cells, growth factors, and scaffolds) can result in complete regeneration of the pulp-dentin complex. There is a delicate balance between disinfection of the root canal system and the interplay of these three components that warrants more research. It is likely that this combined approach of in vitro and in vivo preclinical research will greatly advance our understanding of the conditions necessary to regenerate a functional pulp-dentin complex.

The translational nature of regenerative endodontic research is allowing for changes to take place in clinic practice in a relatively short time. This crosstalk between basic and clinical sciences is largely fueled by the realization that all three components of the tissue-engineering triad are already present in revascularization procedures: stem cells, scaffold (blood clot), and growth factors (from dentin and blood). Preclinical studies that evaluated the effect of irrigants[194,300,310] and medicaments[15,248] on the survival of stem cells, release of growth factors from dentin,[270] and odontoblastic differentiation[52,106] are shaping the future generations of regenerative procedures. Furthermore, the incorporation of other scaffolds such as PRP, PRF, and gelatin sponges have been used in the clinic practice with encouraging results.[173] Discussion about the requirements of an appropriate scaffold and growth factor was begun in the 1960s by Nygaard-Østby,[221] who had no access to our contemporary instruments, materials, and the entire knowledge base of tissue engineering. Although clinical revascularization procedures do not constitute the ideal regenerative treatment, it is important to note that they do generate a scaffold (fibrin) and growth factors (from platelets and access to proteins embedded in the dentinal walls), and the clinical outcome results in continued radiographic root development of the immature permanent tooth with a diagnosis of pulpal necrosis. Thus REPs provide a treatment of high clinical value in cases with an otherwise poor prognosis. Histologic analyses of REPs conducted in patients or animals suggest that the increase in root dimensions is often due to deposition of cementum-like material, osteodentin, dentin, or bone.

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Future clinical research will likely focus on further translating basic research findings into improved regenerative procedures. For example, the formation of a cementum-like material on the dentinal walls may lead to studies evaluating benefits of REPs for overall tooth resistance to fracture. In addition, it is clear that the multipotent nature of many MSC types could contribute to the finding of cementum deposition. Controlled differentiation of stem cells into odontoblasts is an important area of research and amenable to tissue-engineering concepts. The development of delivery systems that permit structural reinforcement of the cervical area (or ideally, the pulp chamber) might provide clinical opportunities to regenerate lost tooth structure, thereby permitting natural teeth to be retained instead of possible fracture or extraction. Finally, the ultimate and long-term goal for REPs should be to treat the fully formed permanent tooth. Although this situation is more complex than the immature tooth with an open apex and a ready source of stem cells, it provides the unique potential of saving the natural dentition while restoring the sensory, immunologic, and defensive properties of the pulp-dentin complex.

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References

1. About I. *Dentin regeneration in vitro: the pivotal role of supportive cells*, Adv Dent Res 2011;23: 320.
2. About I, Bottero MJ, de Denato P, et al: *Human dentin production in vitro*, Exp Cell Res 2000;258: 33.
3. About I, Laurent-Maquin D, Lendahl U, et al: *Nestin expression in embryonic and adult human teeth under normal and pathological conditions*, Am J Pathol 2000;157: 287.
4. Agata H, Kagami H, Watanabe N, et al: *Effect of ischemic culture conditions on the survival and differentiation of porcine dental pulp-derived cells*, Differentiation 2008;76: 981.
5. Aggarwal V, Miglani S, Singla M. *Conventional apexification and revascularization induced maturogenesis of two non-vital, immature teeth in same patient: 24 months follow up of a case*, J Conserv Dent 2012;15: 68.
6. Akbari M, Rouhani A, Samiee S, et al: *Effect of dentin bonding agent on the prevention of tooth discoloration produced by mineral trioxide aggregate*, Int J Dent 2012;2012: 563203.
7. Al-Hezaimi K, Al-Tayar BA, Bajuaifer YS, et al: *A hybrid approach to direct pulp capping by using emdogain with a capping material*, J Endod 2011;37: 667.
8. Al-Hezaimi K, Salameh Z, Al-Fouzan K, et al: *Histomorphometric and micro-computed tomography analysis of pulpal response to three different pulp capping materials*, J Endod 2011;37: 507.
9. Alagl A, Bedi S, Hassan K, et al: *Use of platelet-rich plasma for regeneration in non-vital immature permanent*

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www.konkur.in

teeth: Clinical and cone-beam computed tomography evaluation, J Int Med Res 2017;45: 583.
10. Albuquerque MT, Valera MC, Moreira CS, et al: *Effects of ciprofloxacin-containing scaffolds on enterococcus faecalis biofilms*, J Endod 2015;41: 710.
11. Aldakak MM, Capar ID, Rekab MS, et al: *Single-visit pulp revascularization of a nonvital immature permanent tooth using biodentine*, Iran Endod J 2016;11: 246.
12. Aldekhayel SA, Sinno H, Gilardino MS. *Acellular dermal matrix in cleft palate repair: an evidence-based review*, Plast Reconstr Surg 2012;130: 177.
13. Alobaid AS, Cortes LM, Lo J, et al: *Radiographic and clinical outcomes of the treatment of immature permanent teeth by revascularization or apexification: a pilot retrospective cohort study*, J Endod 2014;40: 1063.
14. Alqahtani Q, Zaky SH, Patil A, et al: *Decellularized swine dental pulp tissue for regenerative root canal therapy*, J Dent Res 2018;97: 1460.
15. Althumairy RI, Teixeira FB, Diogenes A. *Effect of dentin conditioning with intracanal medicaments on survival of stem cells of apical papilla*, J Endod 2014;40: 521.
16. Ando Y, Honda MJ, Ohshima H, et al: *The induction of dentin bridge-like structures by constructs of subcultured dental pulp-derived cells and porous HA/TCP in porcine teeth*, Nagoya J Med Sci 2009;71: 51.
17. Andreasen JO. *Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth*, Scand J Dent Res 1970;78: 273.
18. Andreasen JO, Borum MK, Andreasen FM. *Replantation of 400 avulsed permanent incisors. 3. Factors related to root growth*, Endod Dent Traumatol 1995;11: 69.
19. Andreasen JO, Borum MK, Jacobsen HL, et al:

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Replantation of 400 avulsed permanent incisors. 2. Factors related to pulpal healing, Endod Dent Traumatol 1995;11: 59.

20. Anitua E, Andia I, Ardanza B, et al: *Autologous platelets as a source of proteins for healing and tissue regeneration*, Thromb Haemost 2004;91: 4.
21. Aranha AM, Zhang Z, Neiva KG, et al: *Hypoxia enhances the angiogenic potential of human dental pulp cells*, J Endod 2010;36: 1633.
22. Arruda MEF, Neves MAS, Diogenes A, et al: *Infection control in teeth with apical periodontitis using a triple antibiotic solution or calcium hydroxide with chlorhexidine: a randomized clinical trial*, J Endod 2018;44: 1474.
23. Atilla G. *A rare find in Anatolia—a tooth implant (mid-sixth century B.C.)*, J Oral Implantol 1993;19: 54.
24. Austah O, Joon R, Fath WM, et al: *Comprehensive characterization of 2 immature teeth treated with regenerative endodontic procedures*, J Endod 2018;44: 1802.
25. Austah O, Widbiller M, Tomson PL, et al: *Expression of neurotrophic factors in human dentin and their regulation of trigeminal neurite outgrowth*, J Endod 2019.
26. Baksh D, Song L, Tuan RS. *Adult mesenchymal stem cells: characterization, differentiation, and application in cell and gene therapy*, J Cell Mol Med 2004;8: 301.
27. Banchs F, Trope M. *Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol?*, J Endod 2004;30: 196.
28. Bang G, Nordenram A, Anneroth G. *Allogenic demineralized dentin implants in jaw defects of Java monkeys*, Int J Oral Surg 1972;1: 126.

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29. Batur YB, Erdemir U, Sancakli HS. *The long-term effect of calcium hydroxide application on dentin fracture strength of endodontically treated teeth*, Dent Traumatol 2013;29: 461.
30. Becerra P, Ricucci D, Loghin S, et al: *Histologic study of a human immature permanent premolar with chronic apical abscess after revascularization/revitalization*, J Endod 2014;40: 133.
31. Begue-Kirn C, Smith AJ, Loriot M, et al: *Comparative analysis of TGF beta s, BMPs, IGF1, msxs, fibronectin, osteonectin and bone sialoprotein gene expression during normal and in vitro-induced odontoblast differentiation*, Int J Dev Biol 1994;38: 405.
32. Begue-Kirn C, Smith AJ, Ruch JV, et al: *Effects of dentin proteins, transforming growth factor beta 1 (TGF beta 1) and bone morphogenetic protein 2 (BMP2) on the differentiation of odontoblast in vitro*, Int J Dev Biol 1992;36: 491.
33. Belobrov I, Parashos P. *Treatment of tooth discoloration after the use of white mineral trioxide aggregate*, J Endod 2011;37: 1017.
34. Bender IB, Seltzer S, Soltanoff W. *Endodontic success—a reappraisal of criteria. 1*, Oral Surg Oral Med Oral Pathol 1966;22: 780.
35. Bender IB, Seltzer S, Soltanoff W. *Endodontic success—a reappraisal of criteria. II*, Oral Surg Oral Med Oral Pathol 1966;22: 790.
36. Berkhoff JA, Chen PB, Teixeira FB, et al: *Evaluation of triple antibiotic paste removal by different irrigation procedures*, J Endod 2014;40: 1172.
37. Bidder M, Latifi T, Towler DA. *Reciprocal temporospatial patterns of Msx2 and Osteocalcin gene*

Telegram: @dental_k

www.konkur.in

expression during murine odontogenesis, J Bone Miner Res 1998;13: 609.

38. Blin-Wakkach C, Lezot F, Ghoul-Mazgar S, et al: *Endogenous Msx1 antisense transcript: in vivo and in vitro evidences, structure, and potential involvement in skeleton development in mammals*, Proc Natl Acad Sci U S A 2001;98: 7336.
39. Bose R, Nummikoski P, Hargreaves K. *A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures*, J Endod 2009;35: 1343.
40. Botero TM, Shelburne CE, Holland GR, et al: *TLR4 mediates LPS-induced VEGF expression in odontoblasts*, J Endod 2006;32: 951.
41. Botero TM, Son JS, Vodopyanov D, et al: *MAPK signaling is required for LPS-induced VEGF in pulp stem cells*, J Dent Res 2010;89: 264.
42. Bottino MC, Kamocki K, Yassen GH, et al: *Bioactive nanofibrous scaffolds for regenerative endodontics*, J Dent Res 2013;92: 963.
43. Boutsioukis C, Noula G, Lambrianidis T. *Ex vivo study of the efficiency of two techniques for the removal of mineral trioxide aggregate used as a root canal filling material*, J Endod 2008;34: 1239.
44. Boyce ST. *Skin substitutes from cultured cells and collagen-GAG polymers*, Med Biol Eng Comput 1998;36: 791.
45. Bukhari S, Kohli MR, Setzer F, et al: *Outcome of revascularization procedure: a retrospective case series*, J Endod 2016;42: 1752.
46. Butler WT. *Dentin matrix proteins*, Eur J Oral Sci Suppl 1, 1998;106: 204.

Telegram: @dental_k

www.konkur.in

47. Bystrom A, Happonen RP, Sjogren U, et al: *Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis*, Endod Dent Traumatol 1987;3: 58.
48. Camilleri J. *Color stability of white mineral trioxide aggregate in contact with hypochlorite solution*, J Endod 2014;40: 436.
49. Camilleri J. *Staining potential of Neo MTA Plus, MTA Plus, and Biodentine used for pulpotomy procedures*, J Endod 2015.
50. Caplan AI. *Mesenchymal stem cells*, J Orthop Res 1991;9: 641.
51. Caplan AI. *Mesenchymal stem cells: time to change the name!*, Stem Cells Transl Med 2017;6: 1445.
52. Casagrande L, Demarco FF, Zhang Z, et al: *Dentin-derived BMP-2 and odontoblast differentiation*, J Dent Res 2010;89: 603.
53. Cassidy N, Fahey M, Prime SS, et al: *Comparative analysis of transforming growth factor-beta isoforms 1-3 in human and rabbit dentine matrices*, Arch Oral Biol 1997;42: 219.
54. Cavalcanti BN, Zeitlin BD, Nor JE. *A hydrogel scaffold that maintains viability and supports differentiation of dental pulp stem cells*, Dent Mater 2013;29: 97.
55. Chang SW, Lee SY, Ann HJ, et al: *Effects of calcium silicate endodontic cements on biocompatibility and mineralization-inducing potentials in human dental pulp cells*, J Endod 2014;40: 1194.
56. Chiapasco M, Romeo E, Casentini P, et al: *Alveolar distraction osteogenesis vs. vertical guided bone regeneration for the correction of vertically deficient edentulous ridges: a 1-3-year prospective study on*

Telegram: @dental_k

www.konkur.in

humans, Clin Oral Implants Res 2004;15: 82.

57. Chrepa V, Austah O, Diogenes A. *Evaluation of a commercially available hyaluronic acid hydrogel (restylane) as injectable scaffold for dental pulp regeneration: an in vitro evaluation*, J Endod 2017;43: 257.
58. Chrepa V, Henry MA, Daniel BJ, et al: *Delivery of apical mesenchymal stem cells into root canals of mature teeth*, J Dent Res 2015.
59. Chrepa V, Pitcher B, Henry MA, et al: *Survival of the apical papilla and its resident stem cells in a case of advanced pulpal necrosis and apical periodontitis*, J Endod 2017;43: 561.
60. Chueh LH, Ho YC, Kuo TC, et al: *Regenerative endodontic treatment for necrotic immature permanent teeth*, J Endod 2009;35: 160.
61. Cochran DL, Schenk R, Buser D, et al: *Recombinant human bone morphogenetic protein-2 stimulation of bone formation around endosseous dental implants*, J Periodontol 1999;70: 139.
62. Cooper PR, Takahashi Y, Graham LW, et al: *Inflammation-regeneration interplay in the dentine-pulp complex*, J Dent 2010;38: 687.
63. Coudert AE, Pibouin L, Vi-Fane B, et al: *Expression and regulation of the Msx1 natural antisense transcript during development*, Nucleic Acids Res 2005;33: 5208.
64. Cruz EV, Kota K, Huque J, et al: *Penetration of propylene glycol into dentine*, Int Endod J 2002;35: 330.
65. Cvek M. *Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. A retrospective clinical study*, Endod Dent Traumatol 1992;8: 45.

Telegram: @dental_k

www.konkur.in

66. Cvek M. *Treatment of non-vital permanent incisors with calcium hydroxide. IV. Periodontal healing and closure of the root canal in the coronal fragment of teeth with intra-alveolar fracture and vital apical fragment. A follow-up*, Odontol Revy 1974;25: 239.
67. Cvek M, Tsilingaridis G, Andreasen JO. *Survival of 534 incisors after intra-alveolar root fracture in patients aged 7-17 years*, Dent Traumatol 2008;24: 379.
68. d'Aquino De Rosa R, A, Laino G, et al: *Human dental pulp stem cells: from biology to clinical applications*, J Exp Zool Part B: Molecular and Developmental Evolution 2009;312B: 408.
69. D'Souza RN, Happonen RP, Ritter NM, et al: *Temporal and spatial patterns of transforming growth factor-beta 1 expression in developing rat molars*, Arch Oral Biol 1990;35: 957.
70. da Silva Nelson-Filho LA, P, da Silva RA, et al: *Revascularization and periapical repair after endodontic treatment using apical negative pressure irrigation versus conventional irrigation plus triantibiotic intracanal dressing in dogs' teeth with apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: 779.
71. Dammaschke T. *The history of direct pulp capping*, J Hist Dent 2008;56: 9.
72. de Almeida Chen JF, P, Henry MA, et al: *Stem cells of the apical papilla regulate trigeminal neurite outgrowth and targeting through a BDNF-dependent mechanism*, Tissue Eng Part A 2014;20: 3089.
73. De Barros S, Dehez S, Arnaud E, et al: *Aging-related decrease of human ASC angiogenic potential is reversed by hypoxia preconditioning through ROS production*, Mol Ther 2013;21: 399.

Telegram: @dental_k

www.konkur.in

74. Demarco FF, Casagrande L, Zhang Z, et al: *Effects of morphogen and scaffold porogen on the differentiation of dental pulp stem cells*, J Endod 2010;36: 1805.
75. Diderich KE, Nicolaije C, Priemel M, et al: *Bone fragility and decline in stem cells in prematurely aging DNA repair deficient trichothiodystrophy mice*, Age (Dordr) 2012;34: 845.
76. Diogenes A, Ferraz CC, Akopian AN, et al: *LPS sensitizes TRPV1 via activation of TLR4 in trigeminal sensory neurons*, J Dent Res 2011;90: 759.
77. Diogenes A, Henry MA, Teixeira FB, et al: *An update on clinical regenerative endodontics*, Endo Top 2013;28: 2.
78. Diogenes A, Ruparel NB, Shiloah Y, et al: *Regenerative endodontics: A way forward*, J Am Dent Assoc 2016;147: 372.
79. Diogenes AR, Ruparel NB, Teixeira FB, et al: *Translational science in disinfection for regenerative endodontics*, J Endod 2014;40: S52.
80. Doyon GE, Dumsha T, von Fraunhofer JA. *Fracture resistance of human root dentin exposed to intracanal calcium hydroxide*, J Endod 2005;31: 895.
81. Duque C, Hebling J, Smith AJ, et al: *Reactionary dentinogenesis after applying restorative materials and bioactive dentin matrix molecules as liners in deep cavities prepared in nonhuman primate teeth*, J Oral Rehabil 2006;33: 452.
82. Durand SH, Flacher V, Romeas A, et al: *Lipoteichoic acid increases TLR and functional chemokine expression while reducing dentin formation in in vitro differentiated human odontoblasts*, J Immunol 2006;176: 2880.
83. Egusa H, Sonoyama W, Nishimura M, et al: *Stem cells in dentistry—part I: stem cell sources*, J Prosthodont Res

Telegram: @dental_k

www.konkur.in

2012;56: 151.
84. Ejtehadifar M, Shamsasenjan K, Movassaghpour A, et al: *The effect of hypoxia on mesenchymal stem cell biology*, Adv Pharm Bull 2015;5: 141.
85. El Karim IA, Linden GJ, Curtis TM, et al: *Human odontoblasts express functional thermo-sensitive TRP channels: implications for dentin sensitivity*, Pain 2011;152: 2211.
86. el-Ansary MM. *History of pain relief by ancient Egyptians*, Middle East J Anaesthesiol 1989;10: 99.
87. El-Backly RM, Massoud AG, El-Badry AM, et al: *Regeneration of dentine/pulp-like tissue using a dental pulp stem cell/poly(lactic-co-glycolic) acid scaffold construct in New Zealand white rabbits*, Aust Endod J 2008;34: 52.
88. Eskander MA, Takimoto K, Diogenes A. *Evaluation of mesenchymal stem cell modulation of trigeminal neuronal responses to cold*, Neuroscience 2017.
89. Essner MD, Javed A, Eleazer PD. *Effect of sodium hypochlorite on human pulp cells: an in vitro study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 662.
90. Estefan El Batouty BS, KM, Nagy MM, et al: *Influence of age and apical diameter on the success of endodontic regeneration procedures*, J Endod 2016;42: 1620.
91. EzEldeen Van Dessel M, Van Gorp G, J, et al: *3-dimensional analysis of regenerative endodontic treatment outcome*, J Endod 2015;41: 317.
92. Farkas JE, Monaghan JR. *A brief history of the study of nerve dependent regeneration*, Neurogenesis (Austin) 2017;4: e1302216.
93. Ferracane JL, Cooper PR, Smith AJ. *Can interaction of*

Telegram: @dental_k

www.konkur.in

materials with the dentin-pulp complex contribute to dentin regeneration?, Odontology 2010;98: 2.

94. Fichman G, Gazit E. *Self-assembly of short peptides to form hydrogels: design of building blocks, physical properties and technological applications*, Acta Biomater 2014;10: 1671.
95. Finkelman RD, Mohan S, Jennings JC, et al: *Quantitation of growth factors IGF-I, SGF/IGF-II, and TGF-beta in human dentin*, J Bone Miner Res 1990;5: 717.
96. Fitzgerald M, Chiego DJ Jr, Heys DR. *Autoradiographic analysis of odontoblast replacement following pulp exposure in primate teeth*, Arch Oral Biol 1990;35: 707.
97. Flake NM, Gibbs JL, Diogenes A, et al: *A standardized novel method to measure radiographic root changes after endodontic therapy in immature teeth*, J Endod 2014;40: 46.
98. Francke OC. *Capping of the living pulp: from Philip Pfaff to John Wessler*, Bull Hist Dent 1971;19: 17.
99. Frank AL. *[Apexification: therapy for the divergent pulpless tooth]*, Shikai Tenbo 1983;61: 729.
100. Friedenstein AJ, Chailakhyan RK, Latsinik NV, et al: *Stromal cells responsible for transferring the microenvironment of the hemopoietic tissues. Cloning in vitro and retransplantation in vivo*, Transplantation 1974;17: 331.
101. Friedenstein AJ, Deriglasova UF, Kulagina NN, et al: *Precursors for fibroblasts in different populations of hematopoietic cells as detected by the in vitro colony assay method*, Exp Hematol 1974;2: 83.
102. Friedman S, Mor C. *The success of endodontic therapy—healing and functionality*, J Calif Dent Assoc 2004;32: 493.

Telegram: @dental_k

www.konkur.in

103. Galler KM, Brandl FP, Kirchhof S, et al: *Suitability of different natural and synthetic biomaterials for dental pulp tissue engineering*, Tissue Eng Part A 2018;24: 234.
104. Galler KM, Buchalla W, Hiller KA, et al: *Influence of root canal disinfectants on growth factor release from dentin*, J Endod 2015;41: 363.
105. Galler KM, D'Souza RN, Federlin M, et al: *Dentin conditioning codetermines cell fate in regenerative endodontics*, J Endod 2011;37: 1536.
106. Galler KM, D'Souza RN, Federlin M, et al: *Dentin conditioning codetermines cell fate in regenerative endodontics*, J Endod 2011;37: 1536.
107. Galler KM, D'Souza RN, Hartgerink JD, et al: *Scaffolds for dental pulp tissue engineering*, Adv Dent Res 2011;23: 333.
108. Galler KM, Eidt A, Schmalz G. *Cell-free approaches for dental pulp tissue engineering*, J Endod 2014;40: S41.
109. Galler KM, Hartgerink JD, Cavender AC, et al: *A customized self-assembling peptide hydrogel for dental pulp tissue engineering*, Tissue Eng Part A 2012;18: 176.
110. Galler KM, Widbiller M, Buchalla W, et al: *EDTA conditioning of dentine promotes adhesion, migration and differentiation of dental pulp stem cells*, Int Endod J 2016;49: 581.
111. Garcia-Irigoyen O, Carotti S, Latasa MU, et al: *Matrix metalloproteinase-10 expression is induced during hepatic injury and plays a fundamental role in liver tissue repair*, Liver Int 2013.
112. JF Gavino Orduna, Caviedes-Bucheli J, MC Manzanares Cespedes, et al: *Use of platelet-rich plasma in endodontic procedures in adults: regeneration or repair? A report of 3 cases with 5 years of follow-up*, J Endod 2017;43: 1294.

Telegram: @dental_k

www.konkur.in

113. Gelbier MJ. *Pulp capping and pulpotomy: 1750 - 2008*, Dent Hist 2010;58:.
114. Gher ME, Quintero G, Assad D, et al: *Bone grafting and guided bone regeneration for immediate dental implants in humans*, J Periodontol 1994;65: 881.
115. Ghorbanzadeh A, Ghorbanzadeh A. *Maturogenesis of two maxillary central incisors: a case report with 10 years of follow up*, J Dent (Tehran) 2015;12: 306.
116. Gilbert TW, Sellaro TL, Badylak SF. *Decellularization of tissues and organs*, Biomaterials 2006;27: 3675.
117. Goldberg M, Six N, Decup F, et al: *[Mineralization of the dental pulp: contributions of tissue engineering to tomorrow's therapeutics in odontology]*, Pathol Biol (Paris) 2002;50: 194.
118. Goldberg M, Six N, Decup F, et al: *Application of bioactive molecules in pulp-capping situations*, Adv Dent Res 2001;15: 91.
119. Goldberg M, Smith AJ. *Cells and extracellular matrices of dentin and pulp: a biological basis for repair and tissue engineering*, Crit Rev Oral Biol Med 2004;15: 13.
120. Goncalves SB, Dong Z, Bramante CM, et al: *Tooth slice-based models for the study of human dental pulp angiogenesis*, J Endod 2007;33: 811.
121. Gonzalez-Ramos M, Calleros L, Lopez-Ongil S, et al: *HSP70 increases extracellular matrix production by human vascular smooth muscle through TGF-beta1 up-regulation*, Int J Biochem Cell Biol 2013;45: 232.
122. Gould TR. *Ultrastructural characteristics of progenitor cell populations in the periodontal ligament*, J Dent Res 1983;62: 873.
123. Graham L, Cooper PR, Cassidy N, et al: *The effect of calcium hydroxide on solubilisation of bio-active dentine*

Telegram: @dental_k

www.konkur.in

matrix components, Biomaterials 2006;27: 2865.
124. Gronthos S, Mankani M, Brahim J, et al: *Postnatal human dental pulp stem cells (DPSCs) in vitro and in vivo*, Proc Natl Acad Sci U S A 2000;97: 13625.
125. Grossman LI. *Endodontics 1776-1976: a bicentennial history against the background of general dentistry*, J Am Dent Assoc 1976;93: 78.
126. Gu K, Chang S, Ritchie HH, et al: *Molecular cloning of a human dentin sialophosphoprotein gene*, Eur J Oral Sci 2000;108: 35.
127. Guo W, He Y, Zhang X, Lu W, Wang C, Yu H, Liu Y, Li Y, Zhou Y, Zhou J, Zhang M, Deng Z, Jin Y. *The use of dentin matrix scaffold and dental follicle cells for dentin regeneration*, Biomaterials 2009;30: 6708.
128. Habich C, Baumgart K, Kolb H, et al: *The receptor for heat shock protein 60 on macrophages is saturable, specific, and distinct from receptors for other heat shock proteins*, J Immunol 2002;168: 569.
129. Hamasha AA, Hatiwsh A. *Quality of life and satisfaction of patients after nonsurgical primary root canal treatment provided by undergraduate students, graduate students and endodontic specialists*, Int Endod J 2013;46: 1131.
130. Hargreaves KM, Diogenes A, Teixeira FB. *Treatment options: biological basis of regenerative endodontic procedures*, Pediatr Dent 2013;35: 129.
131. Hargreaves KM, Giesler T, Henry M, et al: *Regeneration potential of the young permanent tooth: what does the future hold?*, J Endod 2008;34: S51.
132. He L, Kim SG, Gong Q, et al: *Regenerative endodontics for adult patients*, J Endod 2017;43: S57.
133. He W, Qu T, Yu Q, et al: *LPS induces IL-8 expression through TLR4, MyD88, NF-kappaB and MAPK pathways*

Telegram: @dental_k

www.konkur.in

in human dental pulp stem cells, Int Endod J 2013;46: 128.

134. He W, Wang Z, Luo Z, et al: *LPS promote the odontoblastic differentiation of human dental pulp stem cells via MAPK signaling pathway*, J Cell Physiol 2015;230: 554.
135. He W, Wang Z, Zhou Z, et al: *Lipopolysaccharide enhances Wnt5a expression through toll-like receptor 4, myeloid differentiating factor 88, phosphatidylinositol 3-OH kinase/AKT and nuclear factor kappa B pathways in human dental pulp stem cells*, J Endod 2014;40: 69.
136. Helmlinger G, Yuan F, Dellian M, et al: *Interstitial pH and pO2 gradients in solid tumors in vivo: high-resolution measurements reveal a lack of correlation*, Nat Med 1997;3: 177.
137. Hilton TJ, Ferracane JL, Mancl L. *Northwest practice-based research collaborative in evidence-based D: comparison of CaOH with MTA for direct pulp capping: a PBRN randomized clinical trial*, J Dent Res 2013;92: 16S.
138. Hoshino E, Kurihara-Ando N, Sato I, et al: *In-vitro antibacterial susceptibility of bacteria taken from infected root dentine to a mixture of ciprofloxacin, metronidazole and minocycline*, Int Endod J 1996;29: 125.
139. Huang GT, Shagramanova K, Chan SW. *Formation of odontoblast-like cells from cultured human dental pulp cells on dentin in vitro*, J Endod 2006;32: 1066.
140. Huang GT, Sonoyama W, Liu Y, et al: *The hidden treasure in apical papilla: the potential role in pulp/dentin regeneration and bioroot engineering*, J Endod 2008;34: 645.
141. Huang GT, Yamaza T, Shea LD, et al: *Stem/progenitor cell-mediated de novo regeneration of dental pulp with newly deposited continuous layer of dentin in an in vivo*

Telegram: @dental_k

www.konkur.in

model, Tissue Eng Part A 2010;16: 605.

142. Iohara K, Imabayashi K, Ishizaka R, et al: *Complete pulp regeneration after pulpectomy by transplantation of CD1051 stem cells with stromal cell-derived factor-1*, Tissue Eng Part A 2011;17: 1911.
143. Iohara K, Murakami M, Takeuchi N, et al: *A novel combinatorial therapy with pulp stem cells and granulocyte colony-stimulating factor for total pulp regeneration*, Stem Cells Transl Med 2013;2: 521.
144. Iohara K, Zheng L, Ito M, et al: *Regeneration of dental pulp after pulpotomy by transplantation of CD31(-)/CD146(-) side population cells from a canine tooth*, Regen Med 2009;4: 377.
145. Irish JD. *A 5,500 year old artificial human tooth from Egypt: a historical note*, Int J Oral Maxillofac Implants 2004;19: 645.
146. Ishizaka R, Hayashi Y, Iohara K, et al: *Stimulation of angiogenesis, neurogenesis and regeneration by side population cells from dental pulp*, Biomaterials 2013;34: 1888.
147. Ishizaka R, Iohara K, Murakami M, et al: *Regeneration of dental pulp following pulpectomy by fractionated stem/progenitor cells from bone marrow and adipose tissue*, Biomaterials 2012;33: 2109.
148. Ito K, Yamada Y, Nagasaka T, et al: *Osteogenic potential of injectable tissue-engineered bone: a comparison among autogenous bone, bone substitute (Bio-oss), platelet-rich plasma, and tissue-engineered bone with respect to their mechanical properties and histological findings*, J Biomed Mater Res A 2005;73: 63.
149. Iwaya S-i, Ikawa M, Kubota M. *Revascularization of an immature permanent tooth with apical periodontitis and*

Telegram: @dental_k

www.konkur.in

sinus tract, Dent Traumatol 2001;17: 185.

150. Jacobs JC, Troxel A, Ehrlich Y, et al: *Antibacterial effects of antimicrobials used in regenerative endodontics against biofilm bacteria obtained from mature and immature teeth with necrotic pulps*, J Endod 2017;43: 575.
151. Jadhav GR, Shah N, Logani A. *Platelet-rich plasma supplemented revascularization of an immature tooth associated with a periapical lesion in a 40-year-old man*, Case Rep Dent 2014;2014: 479584.
152. Jeeruphan T, Jantarat J, Yanpiset K, et al: *Mahidol study 1: comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: a retrospective study*, J Endod 2012;38: 1330.
153. Jeeruphan T, Jantarat J, Yanpiset K, et al: *Mahidol study 1: comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: a retrospective study*, J Endod 2012;38: 1330.
154. Jenks DB, Ehrlich Y, Spolnik K, et al: *Residual antibiofilm effects of various concentrations of double antibiotic paste used during regenerative endodontics after different application times*, Arch Oral Biol 2016;70: 88.
155. Johns DA, Vidyanath S. *Revitalization of tooth with necrotic pulp and open apex by using platelet-rich plasma: a case report*, J Endod 2011;37: 743- author reply 743.
156. Jones KB, Klein OD. *Oral epithelial stem cells in tissue maintenance and disease: the first steps in a long journey*, Int J Oral Sci 2013;5: 121.
157. Joseph BK, Savage NW, Daley TJ, et al: *In situ hybridization evidence for a paracrine/autocrine role for insulin-like growth factor-I in tooth development*, Growth

Telegram: @dental_k

www.konkur.in

Factors 1996;13: 11.
158. Judd PL, Casas MJ. *Psychosocial perceptions of premature tooth loss in children*, Ont Dent 1995;72: 16.
159. Kahler B, Mistry S, Moule A, et al: *Revascularization outcomes: a prospective analysis of 16 consecutive cases*, J Endod 2014;40: 333.
160. Kahler B, Rossi-Fedele G. *A review of tooth discoloration after regenerative endodontic therapy*, J Endod 2016;42: 563.
161. Kakoli P, Nandakumar R, Romberg E, et al: *The effect of age on bacterial penetration of radicular dentin*, J Endod 2009;35: 78.
162. Kamocki K, Nor JE, Bottino MC. *Dental pulp stem cell responses to novel antibiotic-containing scaffolds for regenerative endodontics*, Int Endod J 2015;48: 1147.
163. Karapinar-Kazandag Tom-Kun Yamagishi M, Basrani B, V, et al: *Fracture resistance of simulated immature tooth roots reinforced with MTA or restorative materials*, Dent Traumatol 2016;32: 146.
164. Kaval ME, Guneri P, Caliskan MK. *Regenerative endodontic treatment of perforated internal root resorption: a case report*, Int Endod J 2018;51: 128.
165. Kavyasudha C, Macrin D, ArulJothi KN, et al: *Clinical applications of induced pluripotent stem cells - stato attuale*, Adv Exp Med Biol 2018;1079: 127.
166. Kellner M, Steindorff MM, Strempel JF, et al: *Differences of isolated dental stem cells dependent on donor age and consequences for autologous tooth replacement*, Arch Oral Biol 2014;59: 559.
167. Keskin C, Demiryurek EO, Ozyurek T. *Color stabilities of calcium silicate-based materials in contact with different irrigation solutions*, J Endod 2015;41: 409.

Telegram: @dental_k

www.konkur.in

168. Kim J, Song YS, Min KS, et al: *Evaluation of reparative dentin formation of ProRoot MTA, Biodentine and BioAggregate using micro-CT and immunohistochemistry*, Restor Dent Endod 2016;41: 29.
169. Kim JH, Kim Y, Shin SJ, et al: *Tooth discoloration of immature permanent incisor associated with triple antibiotic therapy: a case report*, J Endod 2010;36: 1086.
170. Kim JY, Xin X, Moioli EK, et al: *Regeneration of dental-pulp-like tissue by chemotaxis-induced cell homing*, Tissue Eng Part A 2010;16: 3023.
171. Kitamura M, Nakashima K, Kowashi Y, et al: *Periodontal tissue regeneration using fibroblast growth factor-2: randomized controlled phase II clinical trial*, PLoS One 2008;3: e2611.
172. Kohli MR, Yamaguchi M, Setzer FC, et al: *Spectrophotometric analysis of coronal tooth discoloration induced by various bioceramic cements and other endodontic materials*, J Endod 2015;41: 1862.
173. Kontakiotis EG, Filippatos CG, Tzanetakis GN, et al: *Regenerative endodontic therapy: a data analysis of clinical protocols*, J Endod 2015;41: 146.
174. Krastl G, Allgayer N, Lenherr P, et al: *Tooth discoloration induced by endodontic materials: a literature review*, Dent Traumatol 2013;29: 2.
175. Kuroda R, Matsumoto T, Niikura T, et al: *Local transplantation of granulocyte colony stimulating factor-mobilized CD341 cells for patients with femoral and tibial nonunion: pilot clinical trial*, Stem Cells Transl Med 2014;3: 128.
176. Langer R, Vacanti JP. *Tissue engineering*, Science 1993;260: 920.
177. Latham J, Fong H, Jewett A, et al: *Disinfection efficacy of*

Telegram: @dental_k

www.konkur.in

current regenerative endodontic protocols in simulated necrotic immature permanent teeth, J Endod 2016;42: 1218.

178. Laurent P, Camps J, About I. *Biodentine(TM) induces TGF-beta1 release from human pulp cells and early dental pulp mineralization*, Int Endod J 2012;45: 439.
179. Lei L, Chen Y, Zhou R, et al: *Histologic and immunohistochemical findings of a human immature permanent tooth with apical periodontitis after regenerative endodontic treatment*, J Endod 2015;41: 1172.
180. Lenherr P, Allgayer N, Weiger R, et al: *Tooth discoloration induced by endodontic materials: a laboratory study*, Int Endod J 2012;45: 942.
181. Lenzi R, Trope M. *Revitalization procedures in two traumatized incisors with different biological outcomes*, J Endod 2012;38: 411.
182. Levin LG, Law AS, Holland GR, et al: *Identify and define all diagnostic terms for pulpal health and disease states*, J Endod 2009;35: 1645.
183. Li Z, Jiang CM, An S, et al: *Immunomodulatory properties of dental tissue-derived mesenchymal stem cells*, Oral Dis 2014;20: 25.
184. Liao Al Shahrani J, M, Al-Habib M, et al: *Cells isolated from inflamed periapical tissue express mesenchymal stem cell markers and are highly osteogenic*, J Endod 2011;37: 1217.
185. Lin J, Zeng Q, Wei X, et al: *Regenerative endodontics versus apexification in immature permanent teeth with apical periodontitis: a prospective randomized controlled study*, J Endod 2017;43: 1821.
186. Lin LM, Ricucci D, Huang GT. *Regeneration of the*

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www.konkur.in

dentine-pulp complex with revitalization/revascularization therapy: challenges and hopes, Int Endod J 2014;47: 713.

187. Lin LM, Shimizu E, Gibbs JL, et al: *Histologic and histobacteriologic observations of failed revascularization/revitalization therapy: a case report*, J Endod 2014;40: 291.
188. Linsuwanont P, Wimonsutthikul K, Pothimoke U, et al: *Treatment outcomes of mineral trioxide aggregate pulpotomy in vital permanent teeth with carious pulp exposure: the retrospective study*, J Endod 2017;43: 225.
189. Liu J, Jin T, Chang S, et al: *Matrix and TGF-beta-related gene expression during human dental pulp stem cell (DPSC) mineralization*, In Vitro Cell Dev Biol Anim 2007;43: 120.
190. Liu Y, Gao Y, Zhan X, et al: *TLR4 Activation by ipopolysaccharide and Streptococcus mutans induces differential regulation of proliferation and migration in human dental pulp stem cells*, J Endod 2014;40: 1375.
191. Lovelace TW, Henry MA, Hargreaves KM, et al: *Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure*, J Endod 2011;37: 133.
192. Luo Z, Li D, Kohli MR, et al: *Effect of Biodentine on the proliferation, migration and adhesion of human dental pulp stem cells*, J Dent 2014;42: 490.
193. Marion LR. *Dentistry of ancient Egypt*, J Hist Dent 1996;44: 15.
194. Martin De Almeida DE, JF, Henry MA, et al: *Concentration-dependent effect of sodium hypochlorite on stem cells of apical papilla survival and differentiation*, J Endod 2014;40: 51.

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www.konkur.in

195. Martin G, Ricucci D, Gibbs JL, et al: *Histological findings of revascularized/revitalized immature permanent molar with apical periodontitis using platelet-rich plasma*, J Endod 2013;39: 138.
196. Mente J, Hage N, Pfefferle T, et al: *Mineral trioxide aggregate apical plugs in teeth with open apical foramina: a retrospective analysis of treatment outcome*, J Endod 2009;35: 1354.
197. Mente J, Leo M, Panagidis D, et al: *Treatment outcome of mineral trioxide aggregate in open apex teeth*, J Endod 2013;39: 20.
198. Meschi Torres Garcia N, EzEldeen M, AE, et al: *A Retrospective case series in regenerative endodontics: trend analysis based on clinical evaluation and 2- and 3-dimensional radiology*, J Endod 2018;44: 1517.
199. Meschi N, Hilkens P, Lambrichts I, et al: *Regenerative endodontic procedure of an infected immature permanent human tooth: an immunohistological study*, Clin Oral Investig 2016;20: 807.
200. Miller AA, Takimoto K, Wealleans J, et al: *Effect of 3 bioceramic materials on stem cells of the apical papilla proliferation and differentiation using a dentin disk model*, J Endod 2018;44: 599.
201. Miura M, Gronthos S, Zhao M, et al: *SHED: stem cells from human exfoliated deciduous teeth*, Proc Natl Acad Sci U S A 2003;100: 5807.
202. Montero J, Lorenzo B, Barrios R, et al: *Patient-centered outcomes of root canal treatment: a cohort follow-up study*, J Endod 2015;41: 1456.
203. Moore KA, Lemischka IR. *Stem cells and their niches*, Science 2006;311: 1880.
204. Moorrees CF, Fanning EA, Hunt EE Jr. *Age variation of*

Telegram: @dental_k

www.konkur.in

formation stages for ten permanent teeth, J Dent Res 1963;42: 1490.

205. Morsczeck C, Gotz W, Schierholz J, et al: *Isolation of precursor cells (PCs) from human dental follicle of wisdom teeth*, Matrix Biol 2005;24: 155.

206. Murray PE. *Platelet-rich plasma and platelet-rich fibrin can induce apical closure more frequently than blood-clot revascularization for the regeneration of immature permanent teeth: a meta-analysis of clinical efficacy*, Front Bioeng Biotechnol 2018;6: 139.

207. Murray PE, About I, Lumley PJ, et al: *Human odontoblast cell numbers after dental injury*, J Dent 2000;28: 277.

208. Murray PE, Garcia-Godoy F, Hargreaves KM. *Regenerative endodontics: a review of current status and a call for action*, J Endod 2007;33: 377.

208a Musson DS, McLachlan JL, Sloan AJ, et al: *Adrenomedullin is expressed during rodent dental tissue development and promotes cell growth and mineralization*, Biol Cell 2010;102: 145.

209. Nagas E, Uyanik MO, Cehreli ZC. *Revitalization of necrotic mature permanent incisors with apical periodontitis: a case report*, Restor Dent Endod 2018;43: e31.

210. Nagaveni NB, Pathak S, Poornima P, et al: *Revascularization induced maturogenesis of non-vital immature permanent tooth using platelet-rich-fibrin: a case report*, J Clin Pediatr Dent 2016;40: 26.

211. Nagy MM, Tawfik HE, Hashem AA, et al: *Regenerative potential of immature permanent teeth with necrotic pulps after different regenerative protocols*, J Endod 2014;40: 192.

Telegram: @dental_k

www.konkur.in

212. Nakashima M. *Dentin induction by implants of autolyzed antigen-extracted allogeneic dentin on amputated pulps of dogs*, Endod Dent Traumatol 1989;5: 279.
213. Nakashima M. *Induction of dentin formation on canine amputated pulp by recombinant human bone morphogenetic proteins (BMP)-2 and -4*, J Dent Res 1994;73: 1515.
214. Nakashima M, Akamine A. *The application of tissue engineering to regeneration of pulp and dentin in endodontics*, J Endod 2005;31: 711.
215. Nakashima M, Iohara K. *Mobilized dental pulp stem cells for pulp regeneration: initiation of clinical trial*, J Endod 2014;40: S26.
216. Nasstrom K. *Dentin formation after corticosteroid treatment. A clinical study and an experimental study on rats*, Swed Dent J Suppl 1996;115: 1.
217. Nasstrom K, Forsberg B, Petersson A, et al: *Narrowing of the dental pulp chamber in patients with renal diseases*, Oral Surg Oral Med Oral Pathol 1985;59: 242.
218. Nosrat A, Homayounfar N, Oloomi K. *Drawbacks and unfavorable outcomes of regenerative endodontic treatments of necrotic immature teeth: a literature review and report of a case*, J Endod 2012;38: 1428.
219. Nosrat A, Kolahdouzan A, Khatibi AH, et al: *Clinical, radiographic, and histologic outcome of regenerative endodontic treatment in human teeth using a novel collagen-hydroxyapatite scaffold*, J Endod 2019;45: 136.
220. Nygaard-Ostby B. *[Mortal or vital treatment of the inflamed pulp?]*, SSO Schweiz Monatsschr Zahnheilkd 1966;76: 545.
221. Nygaard-Ostby B, Hjortdal O. *Tissue formation in the root canal following pulp removal*, Scand J Dent Res

Telegram: @dental_k

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1971;79: 333.
222. Oberpenning F, Meng J, Yoo JJ, et al: *De novo reconstitution of a functional mammalian urinary bladder by tissue engineering*, Nat Biotechnol 1999;17: 149.
223. Ogino Y, Ayukawa Y, Kukita T, et al: *The contribution of platelet-derived growth factor, transforming growth factor-beta1, and insulin-like growth factor-I in platelet-rich plasma to the proliferation of osteoblast-like cells*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 724.
224. Okamoto Y, Sonoyama W, Ono M, et al: *Simvastatin induces the odontogenic differentiation of human dental pulp stem cells in vitro and in vivo*, J Endod 2009;35: 367.
225. Okita K, Ichisaka T, Yamanaka S. *Generation of germline-competent induced pluripotent stem cells*, Nature 2007;448: 313.
226. Ostby BN. *The role of the blood clot in endodontic therapy. An experimental histologic study*, Acta Odontol Scand 1961;19: 324.
227. Paakkonen V, Bleicher F, Carrouel F, et al: *General expression profiles of human native odontoblasts and pulp-derived cultured odontoblast-like cells are similar but reveal differential neuropeptide expression levels*, Arch Oral Biol 2009;54: 55.
228. Paakkonen V, Vuoristo JT, Salo T, et al: *Comparative gene expression profile analysis between native human odontoblasts and pulp tissue*, Int Endod J 2008;41: 117.
229. Palma PJ, Ramos JC, Martins JB, et al: *Histologic evaluation of regenerative endodontic procedures with the use of chitosan scaffolds in immature dog teeth with apical periodontitis*, J Endod 2017;43: 1279.
230. Parirokh M, Torabinejad M. *Mineral trioxide aggregate:*

Telegram: @dental_k

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a comprehensive literature review—Part I: chemical, physical, and antibacterial properties, J Endod 2010;36: 16.

231. Parirokh M, Torabinejad M. *Mineral trioxide aggregate: a comprehensive literature review—Part III: Clinical applications, drawbacks, and mechanism of action*, J Endod 2010;36: 400.
232. Paryani K, Kim SG. *Regenerative endodontic treatment of permanent teeth after completion of root development: a report of 2 cases*, J Endod 2013;39: 929.
233. Paula-Silva FW, Ghosh A, Silva LA, et al: *TNF-alpha promotes an odontoblastic phenotype in dental pulp cells*, J Dent Res 2009;88: 339.
234. Peng C, Zhao Y, Wang W, et al: *Histologic findings of a human immature revascularized/regenerated tooth with symptomatic irreversible pulpitis*, J Endod 2017;43: 905.
235. Petrino JA, Boda KK, Shambarger S, et al: *Challenges in regenerative endodontics: a case series*, J Endod 2010;36: 536.
236. Pettiette Endod MT, Zhong S, Moretti AJ, J. *Potential correlation between statins and pulp chamber calcification*, J Endod 2013;39: 1119.
237. Priya MH, Tambakad PB, Naidu J. *Pulp and periodontal regeneration of an avulsed permanent mature incisor using platelet-rich plasma after delayed replantation: a 12-month clinical case study*, J Endod 2016;42: 66.
238. Ravindran S, George A. *Multifunctional ECM proteins in bone and teeth*, Exp Cell Res 2014;325: 148.
239. Ravindran S, Huang CC, George A. *Extracellular matrix of dental pulp stem cells: applications in pulp tissue engineering using somatic MSCs*, Front Physiol 2014;4: 395.

Telegram: @dental_k

www.konkur.in

240. Reddi AH. *Role of morphogenetic proteins in skeletal tissue engineering and regeneration*, Nat Biotechnol 1998;16: 247.
241. Reynolds K, Johnson JD, Cohenca N. *Pulp revascularization of necrotic bilateral bicuspids using a modified novel technique to eliminate potential coronal discolouration: a case report*, Int Endod J 2009;42: 84.
242. Ring KC, Murray PE, Namerow KN, et al: *The comparison of the effect of endodontic irrigation on cell adherence to root canal dentin*, J Endod 2008;34: 1474.
242a Roberts-Clark DJ, Smith AJ. *Angiogenic growth factors in human dentine matrix*, Arch Oral Biol 2000;45: 1013.
243. Robertson A, Andreasen FM, Andreasen JO, et al: *Long-term prognosis of crown-fractured permanent incisors. The effect of stage of root development and associated luxation injury*, Int J Paediatr Dent 2000;10: 191.
244. Robertson A, Andreasen FM, Bergenholtz G, et al: *Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors*, J Endod 1996;22: 557.
245. Rule DC, Winter GB. *Root growth and apical repair subsequent to pulpal necrosis in children*, Br Dent J 1966;120: 586.
246. Ruparel Henry NB, JF de Almeida, MA, et al: *Characterization of a stem cell of apical papilla cell line: effect of passage on cellular phenotype*, J Endod 2013;39: 357.
247. Ruparel NB, Teixeira FB, Ferraz CC, et al: *Direct effect of intracanal medicaments on survival of stem cells of the apical papilla*, J Endod 2012;38: 1372.
248. Ruparel NB, Teixeira FB, Ferraz CC, et al: *Direct effect of intracanal medicaments on survival of stem cells of the*

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www.konkur.in

apical papilla, J Endod 2012;38: 1372.

249. Sabrah AH, Yassen GH, Gregory RL. *Effectiveness of antibiotic medicaments against biofilm formation of Enterococcus faecalis and Porphyromonas gingivalis*, J Endod 2013;39: 1385.
250. Said F, Moskovitz M. *Effect of calcium hydroxide as a root canal dressing material on dentin fracture strength in primary teeth- in vitro study*, J Clin P ediatr Dent 2018;42: 146.
251. Sakai VT, Zhang Z, Dong Z, et al: *SHED differentiate into functional odontoblasts and endothelium*, J Dent Res 2010;89: 791.
252. Saoud TM, Huang GT, Gibbs JL, et al: *Management of teeth with persistent apical periodontitis after root canal treatment using regenerative endodontic therapy*, J Endod 2015;41: 1743.
253. Saoud TM, Martin G, Chen YH, et al: *Treatment of mature permanent teeth with necrotic pulps and apical periodontitis using regenerative endodontic procedures: a case series*, J Endod 2016;42: 57.
254. Saoud TM, Mistry S, Kahler B, et al: *Regenerative Endodontic procedures for traumatized teeth after horizontal root fracture, avulsion, and perforating root resorption*, J Endod 2016;42: 1476.
255. Saoud TM, Sigurdsson A, Rosenberg PA, et al: *Treatment of a large cystlike inflammatory periapical lesion associated with mature necrotic teeth using regenerative endodontic therapy*, J Endod 2014;40: 2081.
256. Saoud TM, Zaazou A, Nabil A, et al: *Histological observations of pulpal replacement tissue in immature dog teeth after revascularization of infected pulps*, Dent Traumatol 2015;31: 243.

Telegram: @dental_k

www.konkur.in

257. Saoud TM, Zaazou A, Nabil A, et al: *Clinical and radiographic outcomes of traumatized immature permanent necrotic teeth after revascularization/revitalization therapy*, J Endod 2014;40: 1946.
258. Sato I, Ando-Kurihara N, Kota K, et al: *Sterilization of infected root-canal dentine by topical application of a mixture of ciprofloxacin, metronidazole and minocycline in situ*, Int Endod J 1996;29: 118.
259. Sato T, Hoshino E, Uematsu H, et al: *In vitro antimicrobial susceptibility to combinations of drugs on bacteria from carious and endodontic lesions of human deciduous teeth*, Oral Microbiol Immunol 1993;8: 172.
260. Schwarz S, Rotter N. *Human salivary gland stem cells: isolation, propagation, and characterization*, Methods Mol Biol 2012;879: 403.
261. Seo BM, Miura M, Gronthos S, et al: *Investigation of multipotent postnatal stem cells from human periodontal ligament*, Lancet 2004;364: 149.
262. Shi S, Gronthos S. *Perivascular niche of postnatal mesenchymal stem cells in human bone marrow and dental pulp*, J Bone Miner Res 2003;18: 696.
263. Shimizu E, Jong G, Partridge N, et al: *Histologic observation of a human immature permanent tooth with irreversible pulpitis after revascularization/regeneration procedure*, J Endod 2012;38: 1293.
264. Shimizu E, Ricucci D, Albert J, et al: *Clinical, radiographic, and histological observation of a human immature permanent tooth with chronic apical abscess after revitalization treatment*, J Endod 2013;39: 1078.
265. Silujjai J, Linsuwanont P. *Treatment outcomes of apexification or revascularization in nonvital immature*

Telegram: @dental_k

www.konkur.in

permanent teeth: A Retrospective Study, J Endod 2017;43: 238.

266. Simon S, Perard M, Zanini M, et al: *Should pulp chamber pulpotomy be seen as a permanent treatment? Some preliminary thoughts*, Int Endod J 2013;46: 79.
267. Simon S, Smith AJ, Berdal A, et al: *The MAP kinase pathway is involved in odontoblast stimulation via p38 phosphorylation*, J Endod 2010;36: 256.
268. Simon SR, Tomson PL, Berdal A. *Regenerative endodontics: regeneration or repair?*, J Endod 2014;40: S70.
269. Sireesha A, Jayasree R, Vidhya S, et al: *Comparative evaluation of micron- and nano-sized intracanal medicaments on penetration and fracture resistance of root dentin - an in vitro study*, Int J Biol Macromol 2017;104: 1866.
270. Smith AJ, Duncan HF, Diogenes A, et al: *Exploiting the bioactive properties of the dentin-pulp complex in regenerative endodontics*, J Endod 2016;42: 47.
271. Smith AJ, Lesot H. *Induction and regulation of crown dentinogenesis: embryonic events as a template for dental tissue repair?*, Crit Rev Oral Biol Med 2001;12: 425.
272. Smith AJ, Lumley PJ, Tomson PL, et al: *Dental regeneration and materials: a partnership*, Clin Oral Investig 2008;12: 103.
273. Smith AJ, Matthews JB, Hall RC. *Transforming growth factor-beta1 (TGF-beta1) in dentine matrix. Ligand activation and receptor expression*, Eur J Oral Sci Suppl 1, 1998;106: 179.
274. Smith AJ, Tobias RS, Plant CG, et al: *In vivo morphogenetic activity of dentine matrix proteins*, J Biol Buccale 1990;18: 123.

Telegram: @dental_k

www.konkur.in

275. Smith AJ, Tobias RS, Plant CG, et al: *Morphogenetic proteins from dentine extracellular matrix and cell-matrix interactions*, Biochem Soc Trans 1991;19: 187S.
276. Smith AJ, Tobias RS, Plant CG, et al: *Preliminary studies on the in vivo morphogenetic properties of dentine matrix proteins*, Biomaterials 1990;11: 22.
277. Song JS, Takimoto K, Jeon M, et al: *Decellularized human dental pulp as a scaffold for regenerative endodontics*, J Dent Res 2017;96: 640.
278. Song M, Cao Y, Shin SJ, et al: *Revascularization-associated intracanal calcification: assessment of prevalence and contributing factors*, J Endod 2017;43: 2025.
279. Sonoyama W, Liu Y, Fang D, et al: *Mesenchymal stem cell-mediated functional tooth regeneration in swine*, PLoS One 2006;1: e79.
280. Staquet MJ, Durand SH, Colomb E, et al: *Different roles of odontoblasts and fibroblasts in immunity*, J Dent Res 2008;87: 256.
281. Strindberg LZ. *The dependence of the results of pulp therapy on certain factors: an analytic study based on radiographic and clinical follow-up examinations*, Acta Odontol Scand Suppl 1956; 1.
282. Subash D, Shoba K, Aman S, et al: *Revitalization of an immature permanent mandibular molar with a necrotic pulp using platelet-rich fibrin: a case report*, J Clin Diagn Res 2016;10: ZD21.
283. Symons AL, Symons DJ. *Pulpal obliteration related to long-term glucocorticosteroid medication*, Spec Care Dentist 1994;14: 103.
284. Tagelsir A, Yassen GH, Gomez GF, et al: *Effect of antimicrobials used in regenerative endodontic procedures*

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www.konkur.in

on 3-week-old Enterococcus faecalis biofilm, J Endod 2016;42: 258.

285. Takeda T, Tezuka Y, Horiuchi M, et al: *Characterization of dental pulp stem cells of human tooth germs*, J Dent Res 2008;87: 676.
286. Tecles O, Laurent P, Aubut V, et al: *Human tooth culture: a study model for reparative dentinogenesis and direct pulp capping materials biocompatibility*, J Biomed Mater Res B Appl Biomater 2008;85: 180.
287. Thelen DS, Trovik TA, Bardsen A. *Impact of traumatic dental injuries with unmet treatment need on daily life among Albanian adolescents: a case-control study*, Dent Traumatol 2011;27: 88.
288. Thesleff I, Vaahtokari A. *The role of growth factors in determination and differentiation of the odontoblastic cell lineage*, Proc Finn Dent Soc Suppl 1, 1992;88: 357.

288a Thibodeau B, Teixeira F, Yamauchi M, et al: *Pulp revascularization of immature dog teeth with apical periodontitis*, J Endod 2007;33: 680.

289. Thomadakis G, Ramoshebi LN, Crooks J, et al: *Immunolocalization of bone morphogenetic protein-2 and -3 and osteogenic protein-1 during murine tooth root morphogenesis and in other craniofacial structures*, Eur J Oral Sci 1999;107: 368.
290. Tobias Duarte PC, Gomes-Filho JE, Ervolino E, et al: *Histopathological condition of the remaining tissues after endodontic infection of rat immature teeth*, J Endod 2014;40: 538.
291. Tomic S, Djokic J, Vasilijic S, et al: *Immunomodulatory properties of mesenchymal stem cells derived from dental pulp and dental follicle are susceptible to activation by toll-like receptor agonists*, Stem Cells Dev 2011;20: 695.

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www.konkur.in

292. Tomson PL, Grover LM, Lumley PJ, et al: *Dissolution of bio-active dentine matrix components by mineral trioxide aggregate*, J Dent 2007;35: 636.

292a Tomson PL, Lumley PJ, Alexander MY, et al: *Hepatocyte growth factor is sequestered in dentine matrix and promotes regeneration-associated events in dental pulp cells*, Cytokine 2013;61: 622.

293. Topcuoglu G, Topcuoglu HS. *Regenerative endodontic therapy in a single visit using platelet-rich plasma and biodentine in necrotic and asymptomatic immature molar teeth: a report of 3 cases*, J Endod 2016;42: 1344.

294. Torabinejad M, Chivian N. *Clinical applications of mineral trioxide aggregate*, J Endod 1999;25: 197.

295. Torabinejad M, Corr R, Buhrley M, et al: *An animal model to study regenerative endodontics*, J Endod 2011;37: 197.

296. Torabinejad M, Faras H, Corr R, et al: *Histologic examinations of teeth treated with 2 scaffolds: a pilot animal investigation*, J Endod 2014;40: 515.

297. Torabinejad M, Nosrat A, Verma P, et al: *Regenerative endodontic treatment or mineral trioxide aggregate apical plug in teeth with necrotic pulps and open apices: a systematic review and meta-analysis*, J Endod 2017;43: 1806.

298. Torabinejad M, Parirokh M. *Mineral trioxide aggregate: a comprehensive literature review—part II: leakage and biocompatibility investigations*, J Endod 2010;36: 190.

299. Torabinejad M, Turman M. *Revitalization of tooth with necrotic pulp and open apex by using platelet-rich plasma: a case report*, J Endod 2011;37: 265.

300. Trevino EG, Patwardhan AN, Henry MA, et al: *Effect of irrigants on the survival of human stem cells of the apical*

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papilla in a platelet-rich plasma scaffold in human root tips, J Endod 2011;37: 1109.

301. Tzanetakis GN. *Management of intruded immature maxillary central incisor with pulp necrosis and severe external resorption by regenerative approach*, J Endod 2018;44: 245.
302. Tziafas D, Kodonas K. *Dentinogenic specificity in the preclinical evaluation of vital pulp treatment strategies: a critical review*, Dent J (Basel) 2015;3: 133.
303. Vainio S, Karavanova I, Jowett A, et al: *Identification of BMP-4 as a signal mediating secondary induction between epithelial and mesenchymal tissues during early tooth development*, Cell 1993;75: 45.
304. Valera MC, Albuquerque MT, Yamasaki MC, et al: *Fracture resistance of weakened bovine teeth after long-term use of calcium hydroxide*, Dent Traumatol 2015;31: 385.
305. Vanacker De Berdt J, Viswanath A, P, et al: *Hypoxia modulates the differentiation potential of stem cells of the apical papilla*, J Endod 2014;40: 1410.
306. Vangsness CT Jr, Farr J II, Boyd J, et al: *Adult human mesenchymal stem cells delivered via intra-articular injection to the knee following partial medial meniscectomy: a randomized, double-blind, controlled study*, J Bone Joint Surg Am 2014;96: 90.
307. Verma R, Fischer BI, Gregory RL, et al: *The radiopacity and antimicrobial properties of different radiopaque double antibiotic pastes used in regenerative endodontics*, J Endod 2018;44: 1376.
308. Vishwanat L, Duong R, Takimoto K, et al: *Effect of bacterial biofilm on the osteogenic differentiation of stem cells of apical papilla*, J Endod 2017;43: 916.

Telegram: @dental_k

www.konkur.in

309. Wei X, Ling J, Wu L, et al: *Expression of mineralization markers in dental pulp cells*, J Endod 2007;33: 703.
310. Widbiller M, Althumairy RI, Diogenes A. *Direct and indirect effect of chlorhexidine on survival of stem cells from the apical papilla and its neutralization*, J Endod 2019;45: 156.
311. Widbiller M, Driesen RB, Eidt A, et al: *Cell homing for pulp tissue engineering with endogenous dentin matrix proteins*, J Endod 2018;44: 956.
312. Widbiller M, Eidt A, Hiller KA, et al: *Ultrasonic activation of irrigants increases growth factor release from human dentine*, Clin Oral Investig 2017;21: 879.
313. Windley W III, Teixeira F, Levin L, et al: *Disinfection of immature teeth with a triple antibiotic paste*, J Endod 2005;31: 439.
314. Witherspoon DE, Ham K. *One-visit apexification: technique for inducing root-end barrier formation in apical closures*, Pract Proced Aesthet Dent 2001;13: 455.
315. Witherspoon DE, Small JC, Regan JD, et al: *Retrospective analysis of open apex teeth obturated with mineral trioxide aggregate*, J Endod 2008;34: 1171.
316. Wong ML, Griffiths LG. *Immunogenicity in xenogeneic scaffold generation: antigen removal vs. decellularization*, Acta Biomater 2014;10: 1806.
317. Xu L, Tang L, Jin F, et al: *The apical region of developing tooth root constitutes a complex and maintains the ability to generate root and periodontium-like tissues*, J Periodon Res 2009;44: 275.
318. Xu Q, Li Z. *Regenerative endodontic treatment of a maxillary mature premolar*, Case Rep Dent 2018;2018: 5234136.
319. Xu X, Jha AK, Harrington DA, et al: *Hyaluronic acid-*

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www.konkur.in

based hydrogels: from a natural polysaccharide to complex networks, Soft Matter 2012;8: 3280.

320. Xu Y, MacEntee MI. *The roots of dentistry in ancient China*, J Can Dent Assoc 1994;60: 613.
321. Yamamura T. *Differentiation of pulpal cells and inductive influences of various matrices with reference to pulpal wound healing*, J Dent Res Spec No, 1985;64: 530.
322. Yang X, Yang F, Walboomers XF, et al: *The performance of dental pulp stem cells on nanofibrous PCL/gelatin/nHA scaffolds*, J Biomed Mater Res A 2010;93: 247.
323. Yassen GH, Vail MM, Chu TG, et al: *The effect of medicaments used in endodontic regeneration on root fracture and microhardness of radicular dentine*, Int Endod J 2013;46: 688.
324. Yildirim S, Can A, Arican M, et al: *Characterization of dental pulp defect and repair in a canine model*, Am J Dent 2011;24: 331.
325. Yip WK. *The prevalence of dens evaginatus*, Oral Surg Oral Med Oral Pathol 1974;38: 80.
326. Yoshimura H, Muneta T, Nimura A, et al: *Comparison of rat mesenchymal stem cells derived from bone marrow, synovium, periosteum, adipose tissue, and muscle*, Cell Tissue Res 2007;327: 449.
327. Young CS, Terada S, Vacanti JP, et al: *Tissue engineering of complex tooth structures on biodegradable polymer scaffolds*, J Dent Res 2002;81: 695.
328. Zanini M, Sautier JM, Berdal A, et al: *Biodentine induces immortalized murine pulp cell differentiation into odontoblast-like cells and stimulates biomineralization*, J Endod 2012;38: 1220.
329. Zhang CZ, Li H, Young WG, et al: *Evidence for a local*

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action of growth hormone in embryonic tooth development in the rat, Growth Factors 1997;14: 131.

330. Zhang J, Zhang Y, Lv H, et al: *Human stem cells from the apical papilla response to bacterial lipopolysaccharide exposure and anti-inflammatory effects of nuclear factor I C*, J Endod 2013;39: 1416.
331. Zhao JH, Tsai CH, Chang YC. *Management of radicular cysts using platelet-rich fibrin and bioactive glass: a report of two cases*, J Formos Med Assoc 2014;113: 470.
332. Zhao S, Sloan AJ, Murray PE, et al: *Ultrastructural localisation of TGF-beta exposure in dentine by chemical treatment*, Histochem J 2000;32: 489.

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PART II
Advanced Science Topics

OUTLINE

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13: Structure and functions of the dentin-pulp complex

Inge Fristad, Ellen Berggreen*

CHAPTER OUTLINE

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Morphologic zones of the pulp

The pulp-dentin complex

The dental pulp and dentin function as a unit and the odontoblasts represent a crucial element in this system. The odontoblasts are located in the periphery of the pulp tissue, with extensions into the inner part of dentin. Dentin would not exist unless produced by odontoblasts, and the dental pulp is dependent upon the protection provided by the dentin and enamel. Likewise, integrated dynamics of the pulp-dentin complex imply that impacts on dentin may affect the pulpal components and that disturbances in the dental pulp will in turn affect the quantity and quality of the dentin produced.

Odontoblast layer

The outermost stratum of cells of the healthy pulp is the odontoblast layer (Figs. 13.1 and 13.2). This layer is located immediately subjacent to the predentin. The odontoblast processes, however, pass on through the predentin into the inner part of dentin. Consequently, the odontoblast layer is actually composed of the cell bodies of odontoblasts. In addition, capillaries, nerve fibers, and dendritic cells may be found among the odontoblasts.

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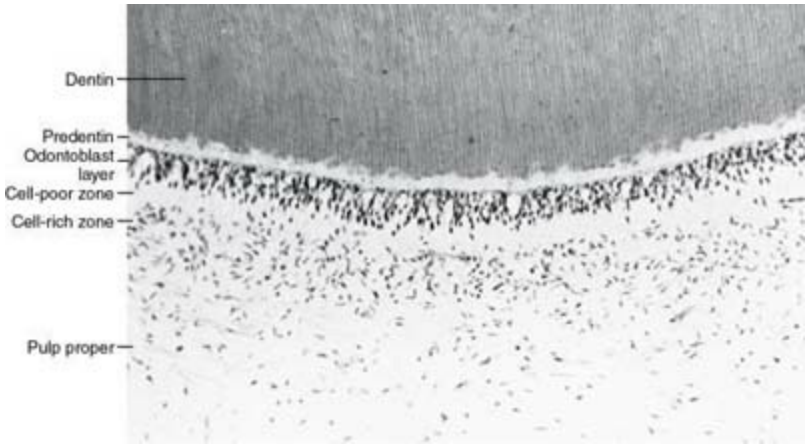

FIG. 13.1 Morphologic zones of the mature pulp.

Micrograph of the mature pulp in tooth with labels as follows: Dentin, predentin, odontoblast layer, cell-poor zone, cell-rich zone, and pulp proper.

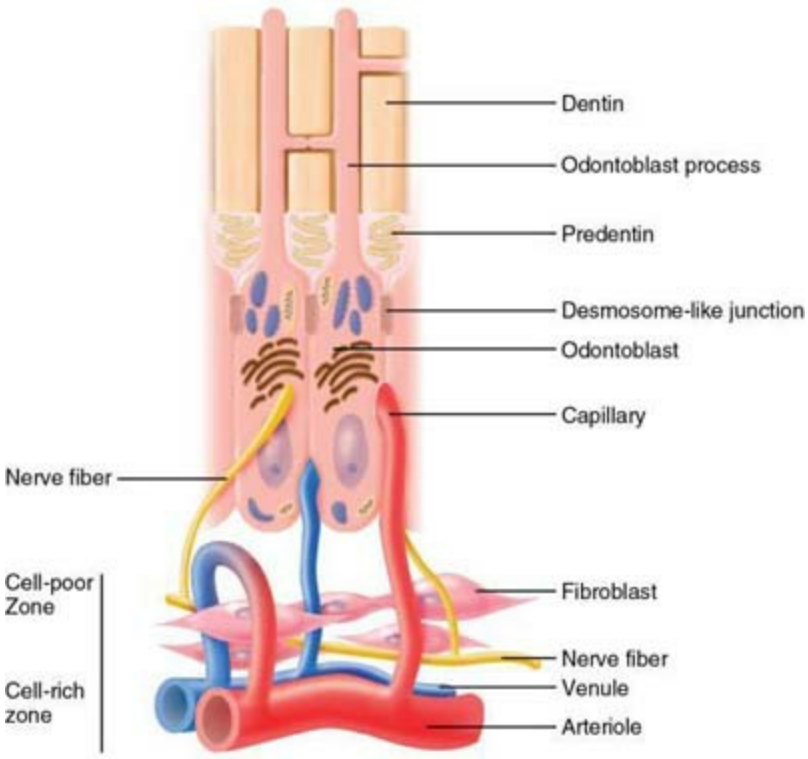

FIG. 13.2 Diagrammatic representation of the odontoblast layer and subodontoblastic region of the pulp.

Pulp with labels as follows: Dentin, odontoblast process, predentin, desmosome-like junction, odontoblast, capillary, fibroblast, nerve fiber, venule, arteriole, cell-rich zone, cell-poor zone, and nerve fiber.

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In the coronal portion of a young pulp that is actively secreting collagen, the odontoblasts assume a tall columnar form.[63] The odontoblasts vary in height; consequently, their nuclei are not all at the same level and are aligned in a staggered array, often described as a palisade appearance. This organization makes the layers appear to be three to five cells in thickness even though there is only one actual layer of odontoblasts. Between adjacent odontoblasts there are small intercellular spaces approximately 30 to 40 nm in width. Odontoblast cell bodies are connected by tight and gap junctional complexes.[29 63,162] Gap junctions are formed by connexin proteins[115] that permit cell-to-cell passage of signal molecules.

The odontoblast layer in the coronal pulp contains more cells per unit area than in the radicular pulp.[245] Whereas the odontoblasts of the mature coronal pulp are usually columnar, those in the midportion of the radicular pulp are more cuboidal.[63] Near the apical foramen, the odontoblasts appear as a squamous layer of flattened cells. Because fewer dentinal tubules per unit area are present in the root than in the crown of the tooth, the odontoblast cell bodies are less crowded and are able to spread out laterally.[245] During maturation and aging, there is a continued ongoing crowding in the odontoblast layer, particularly in the coronal pulp, due to narrowing of the pulp space. Apoptosis of odontoblasts seems to adjust for this limited space during development.[257]

There is a series of specialized cell-to-cell junctions (i.e., junctional complexes), including desmosomes (i.e., zonula adherens), gap junctions (i.e., nexuses), and tight junctions (i.e., zonula occludens) that connect adjacent odontoblasts. Spot desmosomes located in the apical part of odontoblast cell bodies mechanically join odontoblasts together. The numerous gap junctions provide permeable pathways through which signal molecules can pass between cells (Fig. 13.3) to synchronize secretory activity that produces relatively uniform predentin layers (see Fig. 13.2). These junctions are most numerous during the formation of primary dentin. Gap junctions and desmosomes have also been observed joining odontoblasts to the processes of fibroblasts in the subodontoblastic area. Tight junctions are found mainly in the apical part of odontoblasts in young teeth. These structures consist of linear ridges and grooves that close off the intercellular

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space. However, tracer studies suggest direct passage of small elements from subodontoblastic capillaries to predentin and dentin between the odontoblasts.[359] It appears that tight junctions determine the permeability of the odontoblast layer when dentin is covered by enamel or cementum by restricting the passage of molecules, ions, and fluid between the extracellular compartments of the pulp and predentin.[29] During cavity preparation, these junctions are disrupted, thereby increasing dentin permeability.[380,381]

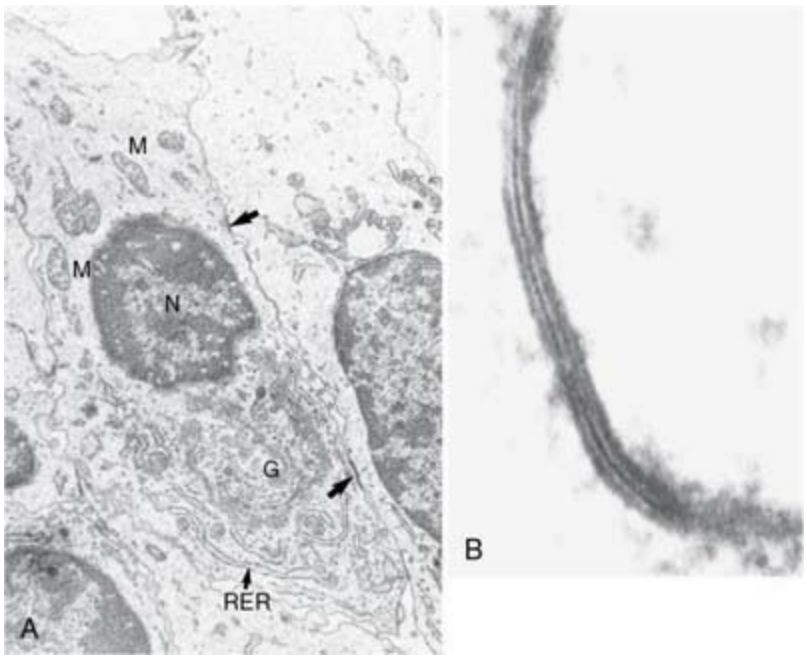

FIG. 13.3 **A,** Electron micrograph of a mouse molar odontoblast demonstrating gap junctions *(arrows),* nucleus *(N)*, mitochondria *(M)*, Golgi complex *(G)*, and rough endoplasmic reticulum *(RER)*. **B,** High magnification of a section fixed and stained with lanthanum nitrate to demonstrate a typical gap junction.

A) Electron micrograph shows an oblique junction with a large dark patch of irregular structure, marked N (nucleus) on the left, which is surrounded by M (mitochondria), G (Golgi apparatus), and RER (rough endoplasmic reticulum).

B) Magnified view of junction has three layers.

Source: (Courtesy Dr. Charles F. Cox, School of Dentistry, University of Alabama.)

Cell-poor zone

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Immediately subjacent to the odontoblast layer in the coronal pulp, there is often a narrow zone approximately 40 μm in width that is relatively free of cells (see Fig. 13.1) and hence is called the *cell-free layer of Weil*. It is traversed by blood capillaries, unmyelinated nerve fibers, and the slender cytoplasmic processes of fibroblasts (see Fig. 13.2). The presence or absence of the cell-poor zone depends on the functional status of the pulp.[63] It may not be apparent in young pulps, where dentin forms rapidly, or in older pulps, where reparative dentin is being produced.

Cell-rich zone

In the subodontoblastic area, there is a stratum containing a relatively high proportion of fibroblasts compared with the more central region of the pulp (see Fig. 13.1). It is much more prominent in the coronal pulp than in the radicular pulp. Besides fibroblasts, the cell-rich zone may include a variable number of immune cells like macrophages and dendritic cells but also undifferentiated mesenchymal stem cells (MSCs).

On the basis of evidence obtained in rat molar teeth, it has been suggested[128] that the cell-rich zone forms as a result of peripheral migration of cells populating the central regions of the pulp, commencing at about the time of tooth eruption. Migration of immunocompetent cells out of and into the cell-rich zone has been demonstrated as a result of antigenic challenge.[428] Although cell division within the cell-rich zone is rare in normal pulps, the death of the odontoblasts triggers a great increase in the rate of mitosis. Because odontoblasts are postmitotic cells, irreversibly injured odontoblasts are replaced by cells that migrate from the cell-rich zone onto the inner surface of the dentin.[100] This mitotic activity is probably the first step in the formation of a new odontoblast layer.[74,261,263,345] Studies implicate stem cells as a source for these replacement odontoblasts.[353]

Pulp proper

The pulp proper is the central mass of the pulp (see Fig. 13.1). It consists of loose connective tissue and contains the larger blood vessels and nerves. The most prominent cell in this zone is the fibroblast.

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Cells of the pulp

Odontoblast

Because odontoblasts are responsible for dentinogenesis, both during tooth development and aging, the odontoblast is the most characteristic and specialized cell of the dentin-pulp complex. During dentinogenesis, the odontoblasts form dentin and the dentinal tubules, and their presence within the tubules makes dentin a living responsive tissue.

Dentinogenesis, osteogenesis, and cementogenesis are in many respects quite similar, and odontoblasts, osteoblasts, and cementoblasts have many characteristics in common. Each of these cells produces a matrix composed of collagen fibrils, noncollagenous proteins, and proteoglycans that are capable of undergoing mineralization. The ultrastructural characteristics of odontoblasts, osteoblasts, and cementoblasts are likewise similar in that each exhibits a highly ordered rough endoplasmic reticulum (RER), a prominent Golgi complex, secretory granules, and numerous mitochondria. In addition, these cells are rich in RNA, and their nuclei contain one or more prominent nucleoli. These are the general characteristics of protein-secreting cells.

The most significant differences among odontoblasts, osteoblasts, and cementoblasts are their morphologic characteristics and the anatomic relationship between the cells and the mineralized structures they produce. Whereas osteoblasts and cementoblasts are polygonal to cuboidal in form, the fully developed odontoblast of the coronal pulp is a tall columnar cell.[63,245] In bone and cementum, some of the osteoblasts and cementoblasts become entrapped in the matrix as osteocytes or cementocytes, respectively. The odontoblast, on the other hand, leaves behind a cellular process to form the dentinal tubule, and the cell body resides outside the mineralized tissue. Lateral branches between the major odontoblast processes interconnect[180,258] through canaliculi, just as osteocytes and cementocytes are linked through the canaliculi in bone and cementum. This provides a pathway for intercellular communication and circulation of fluid and metabolites through the mineralized matrix.

The ultrastructural features of the odontoblast have been the subject of numerous investigations. The cell body of the active odontoblast has a large nucleus that may contain up to four nucleoli (Fig. 13.4). The nucleus is

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situated at the basal end of the cell and is contained within a nuclear envelope. A well-developed Golgi complex, centrally located in the supranuclear cytoplasm, consists of an assembly of smooth-walled vesicles and cisternae. Numerous mitochondria are evenly distributed throughout the cell body. RER is particularly prominent, consisting of closely stacked cisternae forming parallel arrays that are dispersed diffusely within the cytoplasm. Numerous ribosomes closely associated with the membranes of the cisternae mark the sites of protein synthesis. Within the lumen of the cisternae, filamentous material (probably representing newly synthesized protein) can be observed.

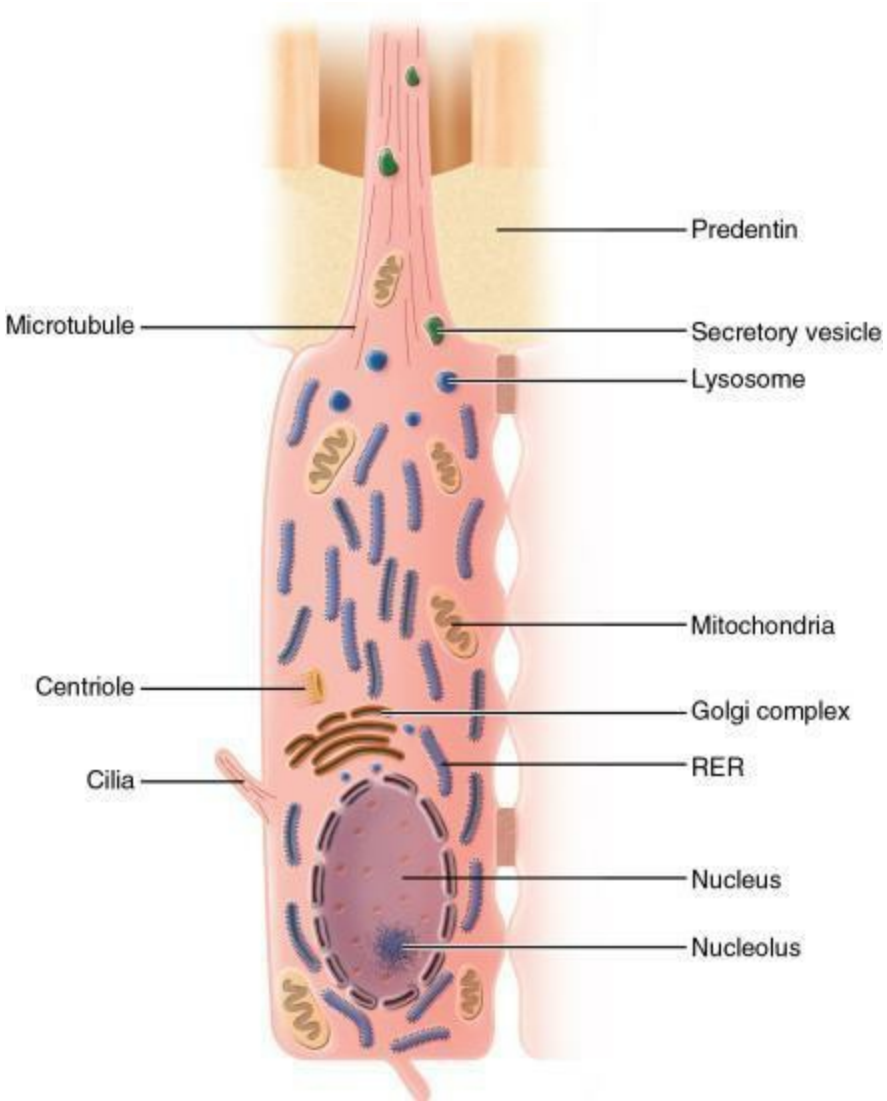

FIG. 13.4 Diagram of a fully differentiated odontoblast. *RER,* Rough

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endoplasmic reticulum.

Differentiated odontoblast with labels as follows: Predentin, secretory vesicle, lysosome, mitochondria, Golgi complex, RER, nucleus, nucleolus, cilia, centriole, and microtubule.

The odontoblast appears to synthesize mainly type I collagen,[212,408] although small amounts of type V collagen have been found in the extracellular matrix (ECM). In addition to proteoglycans[39,85,125] and collagen,[212,224] the odontoblast secretes dentin sialoprotein[41] and phosphophoryn,[41,70] a highly phosphorylated phosphoprotein involved in extracellular mineralization.[41,78] Phosphophoryn is unique to dentin and is not found in any other mesenchymal cell types.[78] The odontoblast also secretes both acid phosphatase and alkaline phosphatase. The latter enzyme is closely linked to mineralization, but the precise role of alkaline phosphatase in dentinogenesis is not completely understood. Acid phosphatase, a lysosomal enzyme, may be involved in digesting resorbed material from the predentin matrix.[89]

In contrast to the active odontoblast, the resting or inactive odontoblast has a decreased number of organelles and may become progressively shorter.[63,245] These changes can begin with the completion of root development and eruption when dentin production shifts from primary to secondary dentin.

Direct actions of odontoblasts on dental nerves and vice versa have been proposed based on the excitability of odontoblasts, the differential expression of receptors for neuropeptides on odontoblasts (Fig. 13.5), the demonstration of the thermosensitive transient receptor potential (TRP) ion channels, and the finding that all nine voltage-gated sodium channels are variably expressed on odontoblasts in developing, mature, and aging rat teeth.[53,72,84,109,236,237] In addition, a possible function of odontoblast in immune regulation has been proposed by the finding of innate immune components in the odontoblast layer.[387] Hence the odontoblasts should be capable of recognizing and differentially responding to bacterial components, thereby serving immune and pulp-dentine barrier functions.

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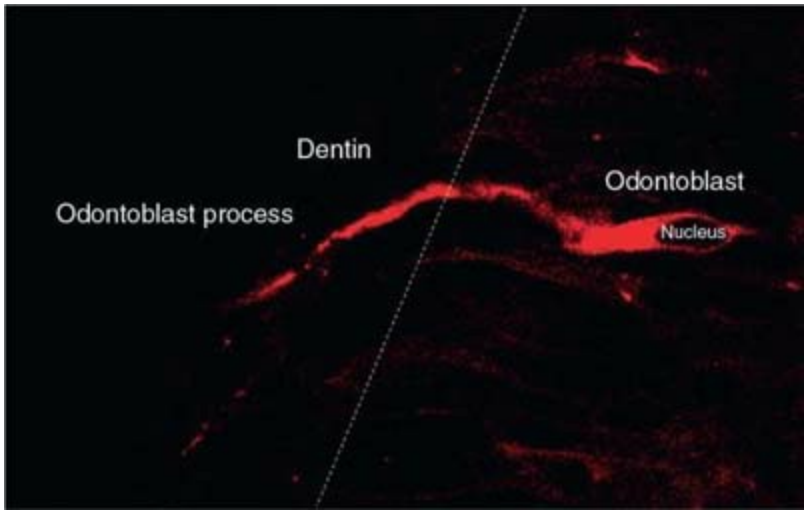

FIG. 13.5 Confocal microscopic image showing an odontoblast with its process expressing neurokinin 2 receptor. Neurokinin 2 has affinity to all the neuropeptides of the neurokinin family. The dotted line represents the border of the predentin.

Microscopic image of pulp with labels as follows: Odontoblast process, dentin, odontoblast, and nucleus.

Source: (From Fristad I, Vandevska-Radunovic V, Fjeld K, et al: NK1, NK2, NK3 and CGRP1 receptors identified in rat oral soft tissues, and in bone and dental hard tissue cells, *Cell Tissue Res* 311:383–391, 2003.)

Odontoblast process

A dentinal tubule forms around each of the major odontoblastic processes. The odontoblast process occupies most of the space within the tubule and coordinates the formation of peritubular dentin.

Microtubules and microfilaments are the principal ultrastructural components of the odontoblast process and its lateral branches.[112,160] Microtubules extend from the cell body out into the process.[112] These straight structures follow a course that is parallel with the long axis of the cell and impart the impression of rigidity. Although their precise role is unknown, theories as to their functional significance suggest that they may be involved in cytoplasmic extension, transport of materials, or the provision of a structural framework. Occasionally, mitochondria can be found in the process where it passes through the predentin.

The plasma membrane of the odontoblast process closely approximates the

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wall of the dentinal tubule. Localized constrictions in the process occasionally produce relatively large spaces between the tubule wall and the process. Such spaces may contain collagen fibrils and fine granular material that presumably represents ground substance (see also The Pulpal Interstitium and Ground Substance in this chapter). The peritubular dentin matrix within the tubule is lined by an electron-dense limiting membrane called the *lamina limitans*.[265,361,405] A narrow space separates the limiting membrane from the plasma membrane of the odontoblast process, except for the areas where the process is constricted.

In restoring a tooth, the removal of enamel and dentin often disrupts odontoblasts.[42,46,74,81,206,262] It would be of considerable clinical importance to establish the extent of the odontoblast processes in human teeth, because with this knowledge the clinician would be in a better position to estimate the impact of the restorative procedure on the underlying odontoblasts. However, the extent to which the process penetrates into dentin has been a matter of considerable controversy. It has long been thought that the process is present throughout the full thickness of dentin. Although ultrastructural studies using transmission electron microscopy have described the process as being limited to the inner third of the dentin,[49,113,361,405] it should be noted that this could possibly be the result of shrinkage occurring during fixation and dehydration. Other studies employing scanning electron microscopy have described the process extending further into the tubule, often as far as the dentoenamel junction (DEJ),[132,181,343,419] but it has been suggested that what has been observed in scanning electron micrographs is actually the lamina limitans.[361,362,405]

In an attempt to resolve this issue, monoclonal antibodies directed against microtubules were used to demonstrate tubulin in the microtubules of the process. Immunoreactivity was observed throughout the dentinal tubule, suggesting that the process extends throughout the thickness of dentin.[343] However, a study employing confocal microscopy found that odontoblast processes in rat molars do not extend to the outer dentin or DEJ, except during the early stages of tooth development.[49] It is likely that the walls of tubules contain many proteins originally derived from odontoblasts that no longer remain at that site. Because dentin matrix has no turnover, these antigens remain fixed in place. From a clinical perspective, it is important to

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remember that these processes in the tubules represent appendages from living odontoblasts in the pulp, which explains why the dentin must be considered a vital tissue, the destruction of which will affect the pulp.

The odontoblast is considered to be a fixed postmitotic cell in that once it has fully differentiated, it apparently cannot undergo further cell division. If this is indeed the case, the life span of the odontoblast coincides with the life span of the viable pulp. However, its metabolic activity can be dynamically altered (described under the heading Pulpal Repair).

Relationship of odontoblast structure to secretory function

Studies using radiolabeled chemicals have shed light on the functional significance of the cytoplasmic organelles of the active odontoblast.[407,408] In experimental animals, intraperitoneal injection of a collagen precursor (e.g., ^{3}H-proline) was followed by autoradiographic labeling of the odontoblasts and predentin matrix (Fig. 13.6).[408] Rapid incorporation of the isotope in the RER soon lead to labeling of the Golgi complex in the area where the procollagen was packed and concentrated into secretory vesicles. Radiolabeled vesicles passed along their migration pathway until they reach the base of the odontoblast process. Here they fused with the cell membrane and released their tropocollagen molecules into the predentin matrix by the process of exocytosis.

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FIG. 13.6 Autoradiograph demonstrating odontoblasts and predentin in a developing rat molar 1 hour after intraperitoneal injection of ^{3}H-proline.

Autoradiograph shows odontoblast cells extending toward dentin. Four horizontal layers with different patterns have scattered tiny dots throughout.

It is now known that collagen fibrils precipitate from a solution of secreted tropocollagen and that the aggregation of fibrils occurs on the outer surface of the odontoblast plasma membrane. Fibrils are released into the predentin and increase in thickness as they approach the mineralized matrix. Whereas fibrils at the base of the odontoblast process are approximately 15 nm in diameter, fibrils in the region of the calcification front have attained a diameter of about 50 nm.

Similar tracer studies[407] have elucidated the pathway of synthesis, transport, and secretion of the predentin proteoglycans. The protein moiety of these molecules is synthesized by the RER of the odontoblast, whereas sulfation and addition of the glycosaminoglycan (GAG) moieties to the protein molecules take place in the Golgi complex. Secretory vesicles then transport the proteoglycans to the base of the odontoblast process, where they are secreted into the predentin matrix. Proteoglycans, principally chondroitin sulfate, accumulate near the calcification front. The role of the proteoglycans is speculative, but mounting evidence suggests that they act as inhibitors of calcification by binding calcium. It appears that just before calcification, the

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proteoglycans are removed, probably by lysosomal enzymes secreted by the odontoblasts.[86]

Pulp fibroblast

Fibroblasts are the most numerous cells of the pulp. They appear to be tissue-specific cells capable of giving rise to cells that are committed to differentiation (e.g., odontoblast-like cells) if given the proper signal. These cells synthesize type I and III collagen, as well as proteoglycans and GAGs. Thus they produce and maintain the matrix proteins of the ECM. Because they are also able to phagocytose and digest collagen, fibroblasts are responsible for collagen turnover in the pulp.

Although distributed throughout the pulp, fibroblasts are particularly abundant in the cell-rich zone. The early differentiating fibroblasts are polygonal and appear to be widely separated and evenly distributed within the ground substance. Cell-to-cell contacts are established between the multiple processes that extend out from each of the cells. Many of these contacts take the form of gap junctions that provide for electronic coupling or chemical signaling from one cell to another. In terms of ultrastructure, the organelles of the immature fibroblasts are generally in a rudimentary stage of development, with an inconspicuous Golgi complex, numerous free ribosomes, and sparse RER. As they mature, the cells become stellate in form, and the Golgi complex enlarges, the RER proliferates, secretory vesicles appear, and the fibroblasts take on the characteristic appearance of protein-secreting cells. In addition, collagen fibrils accumulate along the outer surface of the cell body. With an increase in the number of blood vessels, nerves, and collagen fibers, there is a relative decrease in the number of fibroblasts in the pulp.

Many fibroblasts of the pulp are characterized by being relatively undifferentiated. A more modern term for these undifferentiated cells is MSC. MSC derived from different tissues appear to be tissue specific and are responsible for maintenance of specific tissue functions. However, MSC share some common characteristics, as they are able to differentiate into other cell lineages when given the proper signals. Like MSC from bone and adipose tissues, pulpal MSC proliferate well in culture (Fig. 13.7), and when stimulated they have neurogenic, adipogenic, chondrogenic, and osteogenic

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differentiation potential.[282] Many pulpal cells seem to remain in a relatively undifferentiated modality, compared with fibroblasts of most other connective tissues.[138] This perception has been supported by the observation of large numbers of reticulin-like fibers in the pulp. Reticulin fibers have an affinity for silver stains and are similar to the argyrophilic fibers of the pulp. However, in a careful review, it appears that actual reticulin fibers may not be present in the pulp; instead the previously described fibers are actually *argyrophilic collagen fibers*.[15] The fibers apparently acquire a GAG sheath, and it is this sheath that is impregnated by silver stains. In the young pulp, the nonargyrophilic collagen fibers are sparse, but they progressively increase in number as the pulp ages.

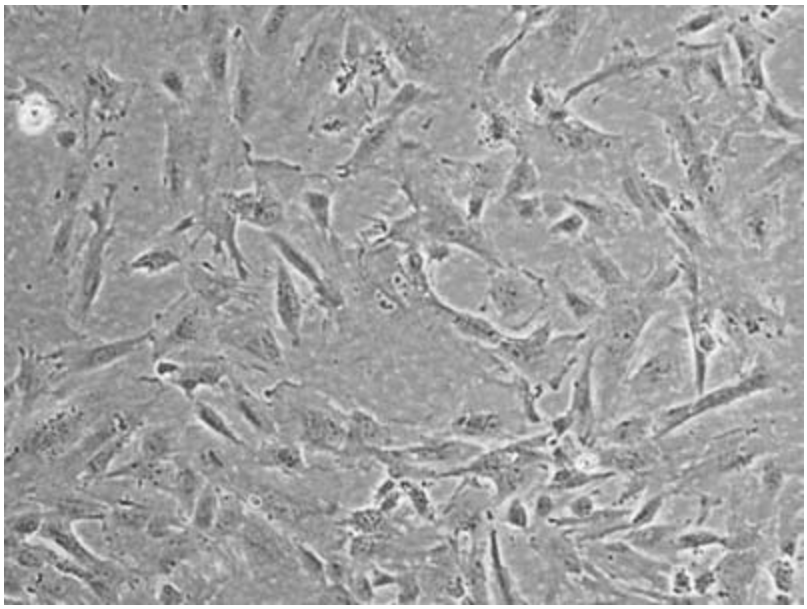

FIG. 13.7 Morphology of cultured pulpal cells. Many of the cells will have mesenchymal stem cell characteristics.

Electron micrograph of cultured pulpal cells shows the network of undifferentiated fibroblast-like cells that have stellate and spindle shapes.

Many experimental models have been developed to study wound healing in the pulp, particularly dentinal bridge formation after pulp exposure or pulpotomy. One study[100] demonstrated that mitotic activity preceding the differentiation of replacement odontoblasts appears to occur primarily among perivascular fibroblasts.

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Pulpal fibroblasts seem to take active part in signaling pathways in the dental pulp. For example, fibroblast growth and synthesis are stimulated by neuropeptides; in turn, fibroblasts produce nerve growth factor (NGF) and proinflammatory cytokines during inflammation.[32,415,420] NGF plays an important role not only in tooth development but also in regulating neuronal and possibly odontoblast responses to injury via activation of similar neurotrophin receptors expressed on both cell types (see also Plasticity of Intradental Nerve Fibers in this chapter).[415]

Macrophage

Macrophages are monocytes that have left the bloodstream, entered the tissues, and differentiated into various subpopulations. The different subpopulations can be studied by their antigenic properties in immunohistochemical studies. Many are found in close proximity to blood vessels. A major subpopulation of macrophages is active in endocytosis and phagocytosis. Because of their mobility and phagocytic activity, they are able to act as scavengers, removing extravasated red blood cells, dead cells, and foreign bodies from the tissue. Ingested material is destroyed by the action of lysosomal enzymes. Another subset of macrophages participates in immune reactions by processing antigen and presenting it to memory T cells.[287] The processed antigen is bound to class II major histocompatibility complex (MHC) molecules on the macrophage, where it can interact with specific receptors present on naive or memory T cells (Fig. 13.8).[135] Such interactions are essential for T-cell–dependent immunity. Similar to fibroblasts, macrophages take an active part in the signaling pathways in the pulp. When activated by the appropriate inflammatory stimuli, macrophages are capable of producing a large variety of soluble factors, including interleukin (IL) 1, tumor necrosis factor (TNF), growth factors, and other cytokines. One study showed that a subset of macrophages express lymphatic markers, indicating a link between macrophages and lymphatic function and development.[19]

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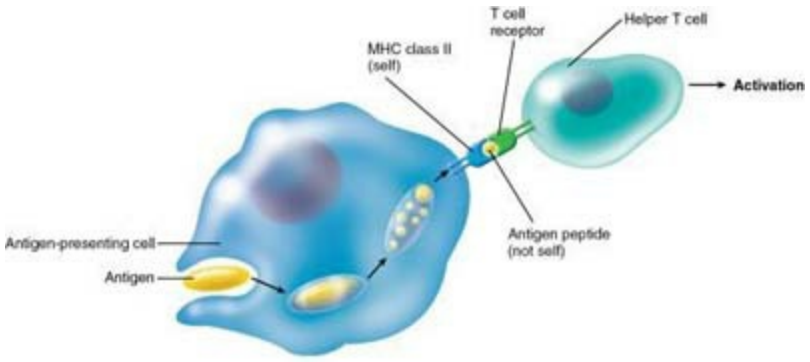

FIG. 13.8 Function of major histocompatibility complex *(MHC)* class II molecule-expressing cells. They act as antigen-presenting cells that are essential for the induction of helper T-cell-dependent immune responses.

Diagram of major histocompatibility complex with labels as follows: Antigen, antigen-presenting cell, MHC class II (self), antigen peptide (not self), T cell receptor, helper T cell, and activation.

Dendritic cell

Dendritic cells are accessory cells of the immune system. Similar cells are found in the epidermis and mucous membranes, where they are called *Langerhans cells*.[175,286] Dendritic cells are primarily found in lymphoid tissues, but they are also widely distributed in connective tissues, including the pulp (Fig. 13.9).[325] These cells are termed *antigen-presenting cells* and are characterized by dendritic cytoplasmic processes and the presence of class II MHC complexes on their cell surface. In the normal pulp they are mostly located in the periphery of the coronal pulp close to the predentin, but they migrate centrally in the pulp after antigenic challenge.[428] They are known to play a central role in the induction of T-cell–dependent immunity. Like antigen-presenting macrophages, dendritic cells engulf protein antigens and then present an assembly of peptide fragments of the antigens and MHC class II molecules. It is this assembly that T cells can recognize. Then the assembly binds to a T-cell receptor and T-cell activation occurs (see Fig. 13.8). Fig. 13.10 shows a cell-to-cell contact between a dendritic-like cell and a lymphocyte.

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FIG. 13.9 Class II antigen-expressing dendritic cells in the pulp and dentin border zone in normal human pulp, as demonstrated by immunocytochemistry. *D,* Dentin; *OB,* odontoblastic layer.

Electron micrograph of pulp shows a thick curved layer of columnar odontoblast cells below dentin layer.

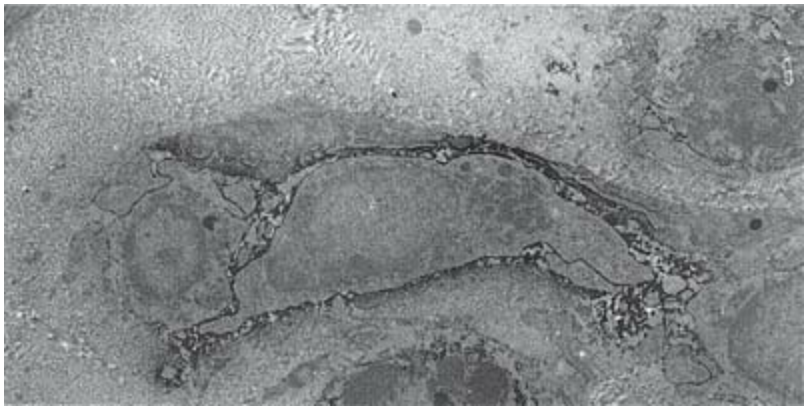

FIG. 13.10 Immunoelectron micrograph of a cell resembling a dendritic cell and a lymphocyte. They show cell-to-cell contact.

Electron micrograph shows a large irregular outline with narrow edges on both sides with a spherical cell on the left that has a small nucleus and condensed chromatin.

Lymphocyte

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Hahn and colleagues[135] reported finding T lymphocytes in normal pulps from human teeth. T8 (suppressor) lymphocytes were the predominant T-lymphocyte subset present in these samples. Lymphocytes have also been observed in the pulps of impacted teeth.[205] The presence of macrophages, dendritic cells, and T lymphocytes indicates that the pulp is well equipped with cells required for the initiation of immune responses.[175,325] B lymphocytes are scarcely found in the normal uninflamed pulp.

Mast cell

Mast cells are widely distributed in connective tissues, where they occur in small groups in relation to blood vessels. Mast cells are seldom found in the normal pulp tissue, although they are routinely found in chronically inflamed pulps.[325] The mast cell has been the subject of considerable attention because of its dramatic role in inflammatory reactions. The granules of mast cells contain heparin, an anticoagulant, and histamine, an important inflammatory mediator, as well as many other chemical factors.

Metabolism

The metabolic activity of the pulp has been studied by measuring the rate of oxygen consumption and the production of carbon dioxide (CO_2) or lactic acid.[26,97,98,99,137,326] An investigation employed an oxygen-sensitive microelectrode inserted into a rat incisor pulp with a micromanipulator, and the authors reported that odontoblasts consumed O_2 at the rate of 3.2 ± 0.2 mL/min/100 g of pulp tissue.[425]

Because of the relatively sparse cellular composition of the pulp, the rate of oxygen consumption is low in comparison with that of most other tissues. During active dentinogenesis, metabolic activity is much higher than after the crown is completed. As would be anticipated, the greatest metabolic activity is found in the region of the odontoblast layer, and the lowest is found in the central pulp, where most of the nerves and blood vessels are located.[25]

In addition to the usual glycolytic pathway, the pulp has the ability to produce energy through a phosphogluconate (i.e., pentose phosphate) shunt type of carbohydrate metabolism,[99] a metabolic pathway that permits tissues

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to function under varying degrees of ischemia. This could explain how the pulp manages to withstand periods of low perfusion resulting from vasoconstriction induced by epinephrine-containing infiltration anesthesia.[193]

Several commonly used dental materials (e.g., eugenol, zinc oxide and eugenol, calcium hydroxide, silver amalgam) inhibit oxygen consumption by pulp tissue, indicating that these agents may be capable of depressing the metabolic activity of pulpal cells.[98,174] One study[137] found that application of orthodontic force to human premolars for 3 days resulted in a 27% reduction in respiratory activity in the pulp. This study utilized carbon-14-labeled succinic acid in the medium. As cells metabolize succinic acid, they produce $^{14}CO_2$ that can be trapped and quantitated by a liquid scintillation counter.[137] This technique requires only a few milligrams of tissue.

The pulpal interstitium and ground substance

The interstitium consists of the interstitial fluid and the interstitial (extracellular) matrix and occupies the extracellular and extravascular space. It is amorphous and generally regarded as a gel rather than a solid. Its constituents are similar in all tissues, but their relative amount varies. The major structural component of the interstitium is collagen (Fig. 13.11). The network of collagen fibers also supports the other components of the interstitium, the proteoglycans, hyaluronan, and elastic fibers. The two former components represent the GAGs of the interstitial matrix.

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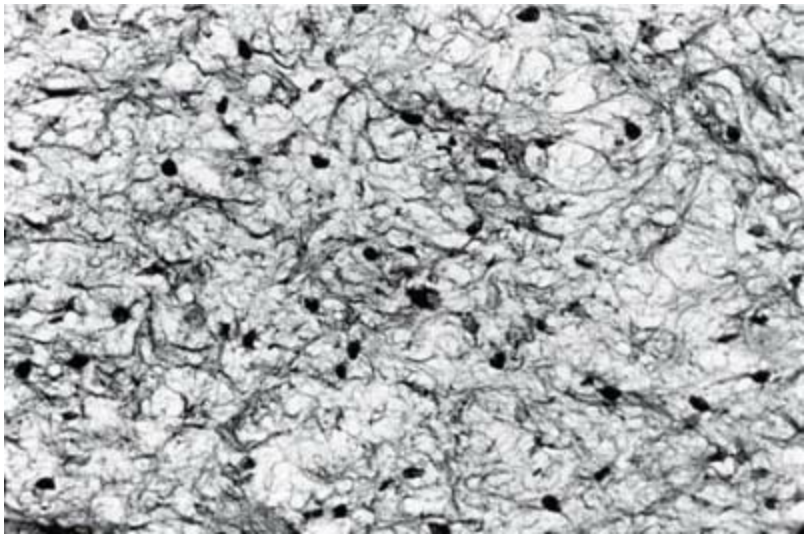

FIG. 13.11 Delicate network of pulpal collagen fibers as demonstrated by the Pearson silver impregnation method.

Electron micrograph of pulpal collagen fibers hold connective tissues together with scattered circular and irregular cells.

Because of its content of polyanionic polysaccharides, the interstitium is responsible for the water-holding properties of connective tissues and acts as a molecular sieve in regulating the diffusion of substances through this space.[411]

Connective tissue consists of cells and fibers, both embedded in ground substance or ECM. Cells that produce connective tissue fibers also synthesize the major constituents of the ECM. Whereas the fibers and cells have recognizable shapes, the ECM is described as being amorphous. It is generally regarded as a gel rather than a solid. Nearly all proteins of the ECM are glycoproteins.[242] Proteoglycans are an important subclass of glycoproteins.[125] These molecules support cells, provide tissue turgor, and mediate a variety of cell interactions. They have in common the presence of GAG chains and a protein core to which the chains are linked. Except for heparan sulfate and heparin, the chains are composed of disaccharides. The primary function of GAG chains is to act as adhesive molecules that can bond to cell surfaces and other matrix molecules.

Fibronectin is a major surface glycoprotein that, together with collagen, forms an integrated fibrillary network that influences adhesion, motility, growth, and differentiation of cells. Laminin, an important component of

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basement membranes, binds to type IV collagen and cell surface receptors.[120] Tenascin is another substrate adhesion glycoprotein.

In the pulp, the principal proteoglycans include hyaluronic acid dermatan sulfate, heparan sulfate, and chondroitin sulfate.[132] The proteoglycan content of pulp tissue decreases approximately 50% with tooth eruption.[224] During active dentinogenesis, chondroitin sulfate is the principal proteoglycan, particularly in the odontoblast and predentin layer, where it is somehow involved with mineralization; with tooth eruption, hyaluronic acid and dermatan sulfate increase, and chondroitin sulfate decreases greatly.

The consistency of a connective tissue (e.g., the pulp) is largely determined by the proteoglycan components of the ground substance. The long GAG chains of the proteoglycan molecules form relatively rigid coils constituting a network that holds water, thus forming a characteristic gel. Hyaluronic acid in particular has a strong affinity for water and is a major component of ground substance in tissues with a large fluid content, such as Wharton jelly of the umbilical cord. The water content of the young pulp is very high (approximately 90%), so the ground substance forms a cushion capable of protecting cells and vascular components of the tooth.

Ground substance also acts as a molecular sieve in that it excludes large proteins. Cell metabolites, nutrients, and wastes pass through the ground substance between cells and blood vessels. In some ways, ground substance can be likened to an ion exchange resin, because the polyanionic chains of the GAGs bind cations. In addition, osmotic pressures can be altered by excluding osmotically active molecules. Thus proteoglycans can regulate the dispersion of interstitial matrix solutes, colloids, and water, and (in large measure) they determine the physical characteristics of a tissue, such as the pulp.

Degradation of ground substance can occur in certain inflammatory lesions that have a high concentration of macrophage lysosomal enzymes. Proteolytic enzymes, hyaluronidases, and chondroitin sulfatases of lysosomal and bacterial origin are examples of the hydrolytic enzymes that can attack components of the ground substance. The pathways of inflammation and infection are strongly influenced by the state of polymerization of the ground substance components.

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Hyaluronan

Another major structural component of the interstitial matrix is hyaluronan. It is an unbranched, random-coil molecule from repeating nonsulfated disaccharide units and is found in the interstitium as free molecules or bound to cells, possibly via the connection to fibronectin.[209] Its large molecular weight together with its protein structure accounts for its unique properties. It has a high viscosity even at low concentration, exhibits exclusion properties, and has a strong affinity for water.

Hyaluronan is one of several types of GAGs in the pulp.[221,242] The hyaluronan receptor-1 is expressed on lymphatic vessels and also on immune cells in the dental pulp.[19] Hyaluronan is removed from the tissue by the lymphatics and metabolized in the lymph nodes[103] and by endothelial cells in the liver.[136,304]

Elastic fibers

Elastic fibers constitute an elastin core and a surrounding microfibrillar network and provide elasticity to the tissue.[295] The amount of elastin in the interstitial matrix in most tissue is small. There is no evidence for elastic fibers in the matrix in the pulp.[136,304]

The inflamed interstitium

Hyaluronidases, chondroitin sulfatases, and matrix metalloproteinases (MMPs) are examples of the enzymes that can attack components of the interstitium.

During infection and inflammation, the physical properties of the pulp tissue may then be altered due to production of such degrading enzymes.[147,324] In addition to their own damaging effect, they may also pave the way for the deleterious effects of bacterial toxins, increasing the magnitude of the damage.[179]

The pathways of inflammation and infection are strongly influenced by the particular composition of the interstitium in every tissue and its degradation by either host or microbial enzymes.

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Connective tissue fibers of the pulp

Two types of structural proteins are found in the pulp: collagen and elastin. Elastin fibers are confined to the walls of arterioles and, unlike collagen, are not a part of the ECM.

A single collagen molecule, referred to as *tropocollagen*, consists of three polypeptide chains, designated as either α-1 or α-2 depending on their amino acid composition and sequence. In the human pulp, the amount of collagen is reported to be 26% to 32% of dry weight in premolars and molars.[383] Type I and type III collagen represent the major subtypes of collagen in the pulp, and type I is found in thick striated fibrils throughout the pulp tissue.[210,342] The different combinations and linkages of chains making up the tropocollagen molecule have allowed collagen fibers and fibrils to be classified into a number of types:

- Type I collagen is found in skin, tendon, bone, dentin, and pulp.
- Type II collagen is found in cartilage.
- Type III collagen is found in most unmineralized connective tissues. It is a fetal form found in the dental papilla and the mature pulp. In bovine pulp, it constitutes 45% of the total pulp collagen during all stages of development.[383]
- Types IV and VII collagen are components of basement membranes.
- Type V collagen is a constituent of interstitial tissues.
- Type VI collagen is a heterotrimer of three distinct chains, α 2 (VI) and α 3 (VI), and is widely distributed in low concentrations in soft tissues at interfibrillar filaments.

Type I collagen is synthesized by odontoblasts and osteoblasts; fibroblasts synthesize types I, III, V, and VII collagen.

In collagen synthesis, the protein portion of the molecule is formed by the polyribosomes of the RER of connective tissue cells. The proline and lysine residues of the polypeptide chains are hydroxylated in the cisternae of the RER, and the chains are assembled into a triple-helix configuration in the smooth endoplasmic reticulum. The product of this assembly is termed *procollagen,* and it has a terminal unit of amino acids known as the *telopeptide of the procollagen molecule*. When these molecules reach the

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Golgi complex, they are glycosylated and packaged in secretory vesicles. The vesicles are transported to the plasma membrane and secreted by way of exocytosis into the extracellular milieu, thus releasing the procollagen. Here the terminal telopeptide is cleaved by a hydrolytic enzyme, and the tropocollagen molecules begin aggregating to form collagen fibrils. It is believed that the GAGs somehow mediate aggregation of tropocollagen. The conversion of soluble collagen into insoluble fibers occurs as a result of cross-linking of tropocollagen molecules. The transcription factor SOX9 is strongly expressed in normal dental pulp tissue and reduced in inflamed pulp.[232] The factor seems to promote collagen synthesis and seems to be important for ECM balance.

The presence of collagen fibers passing from the dentin matrix between odontoblasts into the dental pulp has been reported in fully erupted teeth.[29] Larger collagen fiber bundles are much more numerous in the radicular pulp than in the coronal pulp. The highest concentration of these larger fiber bundles is usually found near the apex (Fig. 13.12).[371]

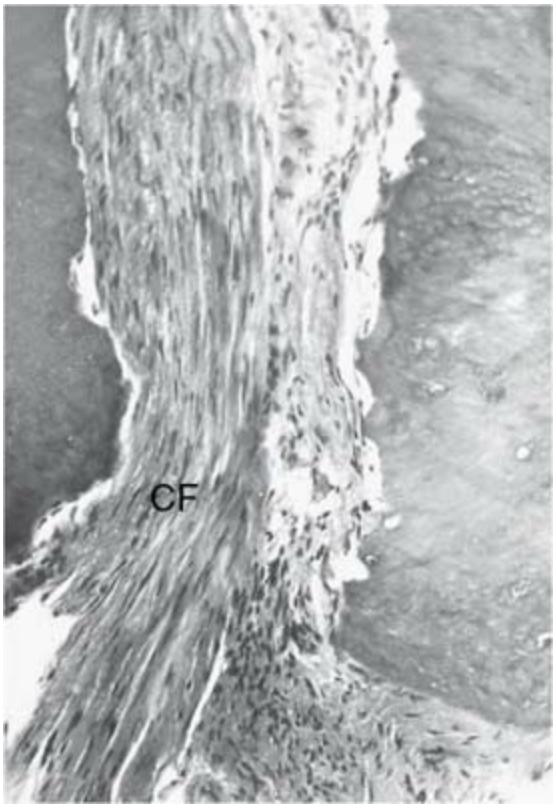

FIG. 13.12 Dense bundles of collagen fibers *(CF)* in the apical pulp.

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Electron micrograph of pulp shows a vertical and parallel bundle of collagen fibers with scattered cells throughout.

The trigeminal system

Innervation

Pain is a subjective phenomenon involving not only sensory physiologic responses, but also emotional, conceptual, and motivational aspects of behavior. The existence of peripheral “nociceptive” (pain-detecting) sensory neurons forms the basis for pain, and pain sensations of varying qualities and intensities are evoked by activation of the intradental nerves innervating teeth. Noxious stimuli in teeth are transmitted in primary afferent neurons located in the trigeminal ganglion via second-order neurons in the brain stem to the brain (Fig. 13.13) (see also Chapter 4 and later in this chapter). Transmission of sensory information consists of a cascade of events involving input, processing, and sensing,[335] so the control of dental pain should be based on an understanding of the origin of pain signals and the complex modulation that may take place locally and at higher levels. The sensory system of the pulp appears to be well suited for signaling potential damage to the tooth. The tooth is innervated by a large number of myelinated and unmyelinated axons. The number of axons entering a human premolar may reach 2000 or more, and each axon can arborize to form multiple points of innervation.[92,171,172]

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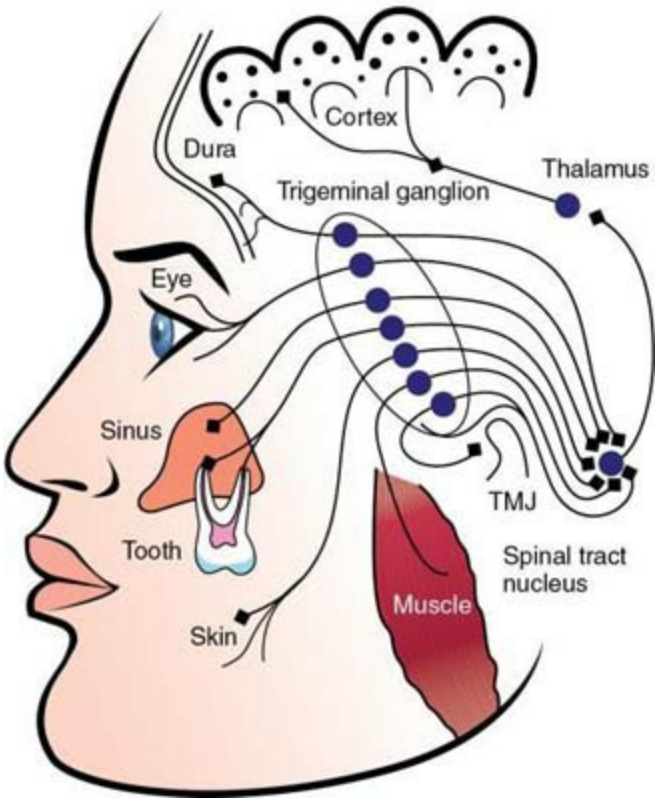

FIG. 13.13 Schematic drawing illustrating the convergence of sensory information from teeth to higher brain centers. *TMJ,* Temporomandibular joint.

Lateral view of face shows branches from eye, sinus, tooth, skin, muscle near spinal tract nucleus and, TMJ converged and leading to thalamus, trigeminal ganglion, dura, and cortex.

Regardless of the nature of the sensory stimulus (i.e., thermal, mechanical, chemical, electric [e.g., pulp tester]), almost all afferent impulses generated from pulp tissue result in the sensation of pain. However, when the pulp is weakly stimulated by an electric pulp tester under carefully controlled experimental conditions, a nonpainful sensation (i.e., prepain) has been reported.[252] Thus not all afferent neurons that innervate the pulp are nociceptors. The innervation of the pulp includes both *afferent neurons,* which conduct sensory impulses, and *autonomic or efferent neurons,*[165] which provide neurogenic modulation of the microcirculation, inflammatory reactions,[150] and perhaps regulate dentinogenesis.[47]

The sympathetic innervation of teeth derives from the superior cervical ganglion (SCG).[8,307] Postganglionic sympathetic nerves travel with the

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internal carotid nerve, join the trigeminal nerve at the ganglion, and supply teeth and supporting structures via the maxillary and mandibular division of the trigeminal nerve.[244] Sympathetic fibers appear with blood vessels at the time the vascular system is established in the dental papilla.[106] In the adult tooth pulp, sympathetic fibers form plexuses, usually around pulpal arterioles (Fig. 13.14). Stimulation of these fibers results in constriction of the arterioles and a decrease in blood flow.[1,82] The sympathetic neuron terminals contain the classic neurotransmitter, norepinephrine (NE), and neuropeptide Y (NPY) (see Neuropeptides later in this chapter). NPY is synthesized in sympathetic neurons and supplied to terminals by axonal transport. By contrast, NE is mainly produced locally in the terminals. Compared with the sensory nerves, these fibers are most often located in deeper parts of the pulp proper, but fibers have also been found in close relation to odontoblasts.[1,166]

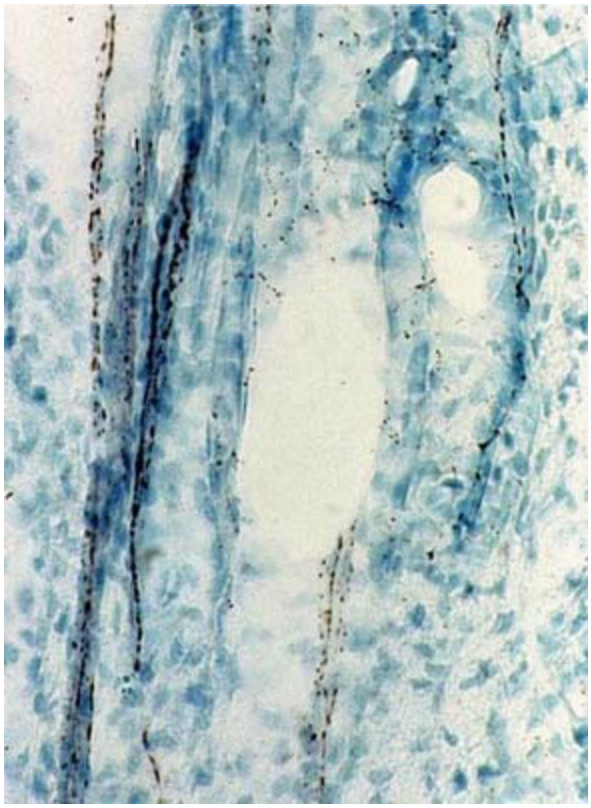

FIG. 13.14 Histologic section, immunohistologically stained for neuropeptide Y (NPY), shows the distribution of sympathetic nerves in the root pulp of a rat molar. NPY fibers are seen associated with blood vessels.

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Histologic section shows an oval light patch at the center surrounded by NPY fibers accompanying blood vessels in a tooth.

Source: (Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)

The presence of parasympathetic cholinergic nerves in dental tissues has been and is still controversial, although it has been concluded that there is absence of parasympathetic vasodilation in the cat dental pulp.[292,329] It has been reported that the neuropeptide vasoactive intestinal polypeptide (VIP) is localized in the parasympathetic neurons.[229,230] The origin of VIP-containing fibers in the pulp is uncertain insofar as no form of surgical denervation has resulted in complete loss of these fibers from the dental pulp.[397]

Sensory nerve fibers are usually classified according to their diameter, conduction velocity, and function (Table 13.1). The pulp contains two types of sensory nerve fibers: myelinated (A fibers) and unmyelinated (C fibers). It has been shown that there is some functional overlap between pulpal A and C fibers, as both fiber types can be nociceptors.[165,171,172,251,272] The A fibers include both A-β and A-δ fibers. The A-β fibers may be slightly more sensitive to stimulation than the A-δ fibers, but functionally these fibers are grouped together in the dental pulp, because both innervate the dentinal tubules, and both are stimulated by dentinal fluid movement (Fig. 13.15). Approximately 90% of the A fibers in dental pulp are A-δ fibers.[251] Table 13.2 summarizes the principal characteristics of the main sensory fibers.

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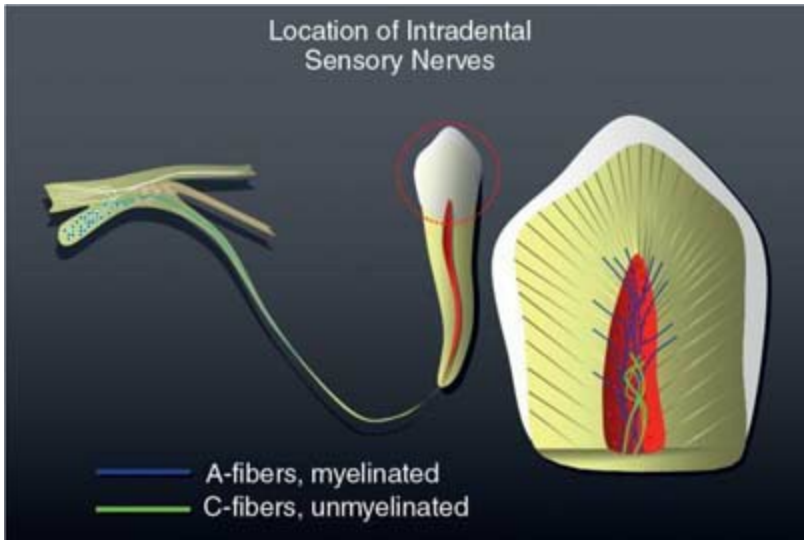

FIG. 13.15 Schematic drawing illustrating the location of A and C fibers in the dental pulp. Myelinated A fibers are located in the periphery of the pulp, penetrating the inner part of dentin. Unmyelinated C fibers are located in the deeper part of the pulp proper.

Location of intradental sensory nerves in crown of tooth is shown. To the right, pulp in the crown area has a mesh of myelinated A-fibers and unmyelinated C-fibers.

Table 13.1

Classification of Nerve Fibers

Type of Fiber	Function	Diameter (μm)	Conduction Velocity (m/s)
A-α	Motor, proprioception	12–20	70–120
A-β	Pressure, touch	5–12	30–70
A-γ	Motor, to muscle spindles	3–6	15–30
A-δ	Pain, temperature, touch	1–5	6–30
B	Preganglionic autonomic	<3	3–15
C dorsal root	Pain	0.4–1	0.5–2
Sympathetic	Postganglionic sympathetic	0.3–1.3	0.7–2.3

Table 13.2

Characteristics of Sensory Fibers

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Fiber	Myelination	Location of Terminals	Pain Characteristics	Stimulation Threshold
A-δ	Yes	Principally in region of pulp-dentin junction	Sharp, pricking	Relatively low
C	No	Probably distributed throughout pulp	Burning, aching, less bearable than A-δ fiber sensations	Relatively high, usually associated with tissue injury

During the bell stage of tooth development, "pioneer" nerve fibers enter the dental papilla following the path of blood vessels.[106] Although only unmyelinated fibers are observed in the dental papilla, a proportion of these fibers are probably A fibers that have lost or not developed their myelin sheath. Myelinated fibers are the last major structures to appear in the developing human dental pulp.[11] The number of nerve fibers gradually increases, and branching occurs as the fibers approach dentin. During the bell stage, very few fibers enter the predentin.[106]

The sensory nerves of the pulp arise from the trigeminal nerve and pass into the radicular pulp in bundles by way of the foramen in close association with arterioles and venules (Fig. 13.16). Each of the nerves entering the pulp is invested within Schwann cells, and the A fibers acquire their myelin sheath from these cells. With the completion of root development, the myelinated fibers appear grouped in bundles in the central region of the pulp (Fig. 13.17). Most of the unmyelinated C fibers entering the pulp are located within these fiber bundles; the remainder is situated toward the periphery of the pulp (see Fig. 13.15).[313] It should be noted that single neurons branch and innervate the pulps of multiple teeth in animal studies.[157] Assuming a similar innervation pattern in humans, such organization may contribute to the poor localization of dental pain and may also allow neurogenic vasodilation and inflammatory reactions to occur in an area of tissue wider than that affected by the original insult (Fig. 13.18). This phenomenon, also known as the axon reflex,[328] was first demonstrated in rat teeth and later confirmed in cat teeth where stimulation of a second premolar caused neurogenic inflammation in the canine tooth.[157,199] An alternative explanation to this clinical observation is that the pulp has a relatively low density of proprioceptors, and thus patients have difficulty identifying the inflamed tooth until the inflammation reaches the periradicular tissue, which is highly innervated with proprioceptors. This is discussed in greater detail in Chapter 4.

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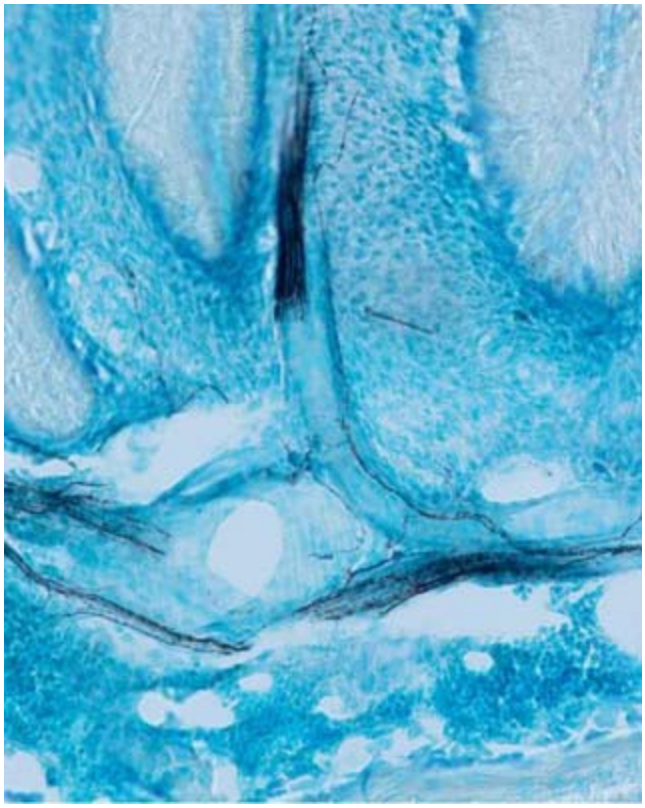

FIG. 13.16 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows distribution of sensory nerves in the apical area of a rat molar. Nerve fibers are seen associated with blood vessels and enter the dental pulp in nerve bundles.

Stained micrograph shows the dense bundle of sensory nerves passing along the inner wall of canal in root apex of a rat tooth.

Source: (Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)

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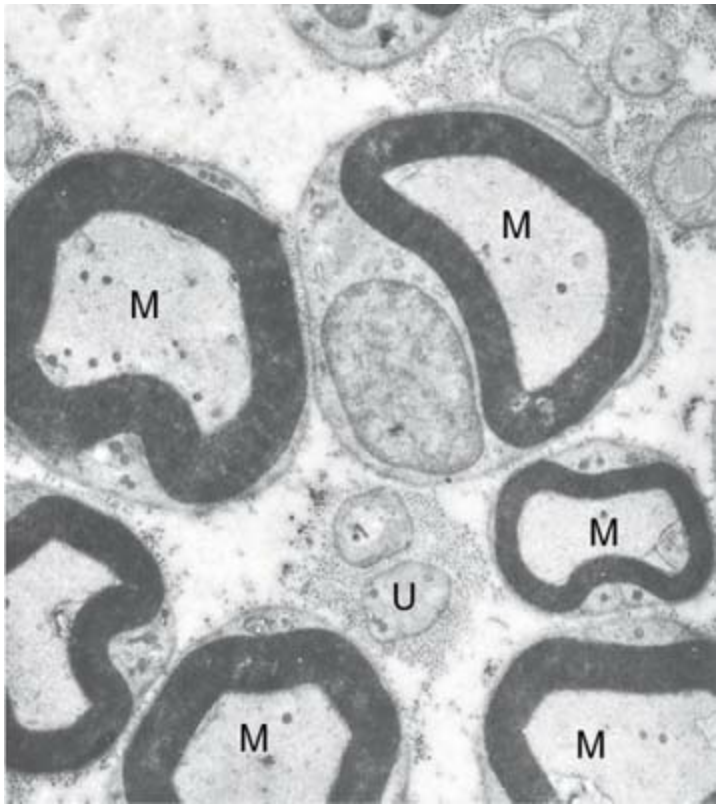

FIG. 13.17 Electron micrograph of the apical pulp of a young canine tooth, showing in cross section myelinated nerve axons *(M)* within Schwann cells. Smaller, unmyelinated axons *(U)* are enclosed singly and in groups by Schwann cells.

Electron micrograph shows light cells with dense material, marked unmyelinated axons, surrounded by large and irregular cells with dark peripheries, marked myelinated nerve axons.

Source: (Courtesy Dr. David C. Johnsen, School of Dentistry, Case Western Reserve University.)

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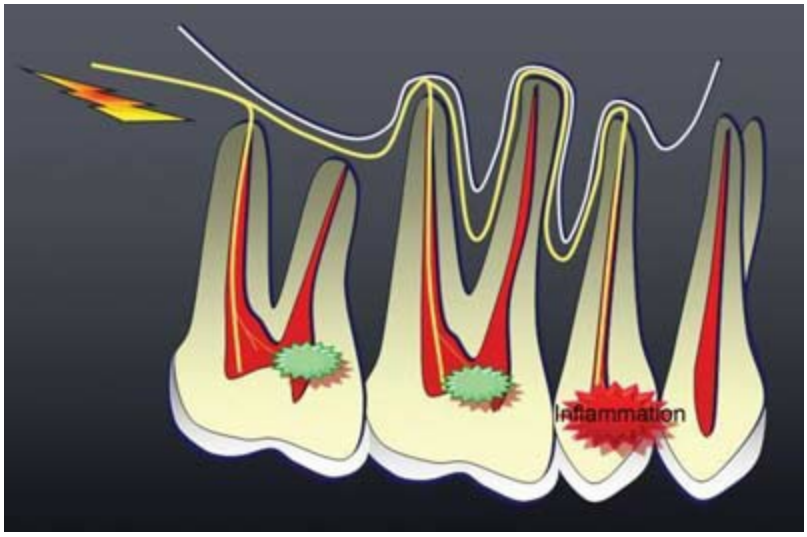

FIG. 13.18 Branches of a single nerve fiber may innervate more than a single tooth. Nerve fiber activation in one tooth may thus result in neurogenic inflammation in adjacent teeth innervated by the same axon. The mechanism is known as the axonal reflex. This phenomenon may also contribute to the poor localization of pain reported by patients.

A branch of nerve fiber enters three teeth through root canals, resulting in the inflammation in the crown of the third tooth.

In the human premolar, the number of unmyelinated axons entering the tooth at the apex reached a maximal number shortly after tooth eruption.[172] At this stage, an average of 1800 unmyelinated axons and more than 400 myelinated axons were found, although in some teeth fewer than 100 myelinated axons were present. Five years after eruption, the number of A fibers gradually increased to more than 700. The relatively late appearance of A fibers in the pulp may help to explain why the electric pulp test tends to be unreliable in young teeth, as A fibers are more easily electrically stimulated than C fibers.[110]

A quantitative study of axons 1 to 2 mm coronal to the root apex of fully developed human canine and incisor teeth[172] reported a mean of about 360 myelinated axons in canines and incisors, whereas there were 1600 to 2200 unmyelinated axons. However, this does not reflect the actual number of neurons supporting a single tooth, because multiple branching of the axons may occur in the peripheral tissues. Overall, approximately 80% of the axons were unmyelinated fibers.[171,172]

The nerve bundles pass upward through the radicular pulp together with

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blood vessels. Once they reach the coronal pulp, they fan out beneath the cell-rich zone, branch into smaller bundles, and finally ramify into a plexus of single-nerve axons known as the *plexus of Raschkow* (Fig. 13.19). Full development of this plexus does not occur until the final stages of root formation.[92] It has been estimated that each axon entering the pulp sends at least eight branches to the plexus of Raschkow. There is prolific branching of the fibers in the plexus, producing a tremendous overlap of receptor fields.[145,272,273,274,278] It is in the plexus that the A fibers emerge from their encircling Schwann cells and branch repeatedly to form the subodontoblastic plexus. Finally, terminal axons pass between the odontoblasts as free nerve endings (see Figs. 13.19–13.21). The extent to which dentin is innervated has been the subject of numerous investigations.[43,46,47,50,92,219] With the exception of the innervation of dentinal tubules, discussed later in this chapter, the bulk of dentin is devoid of sensory nerve fibers. This offers an explanation as to why pain-producing agents (e.g., potassium chloride) do not always elicit pain when applied to exposed dentin. Similarly, application of topical anesthetic solutions to dentin does not decrease its sensitivity. Hence a high concentration of lidocaine solution is needed to block the response of intradental nerves to mechanical stimulation of the dentin.[4]

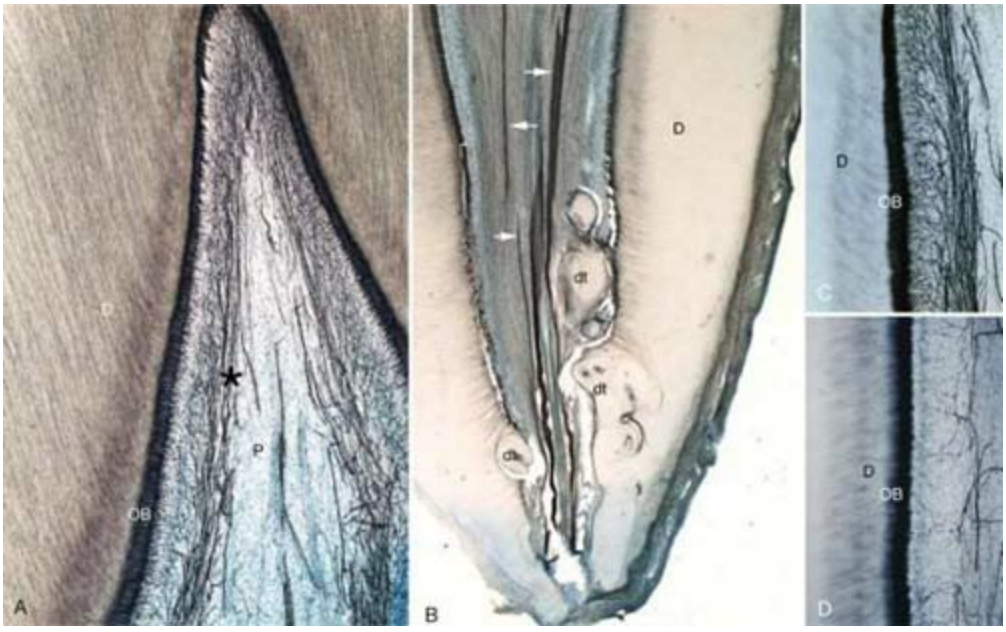

FIG. 13.19 Histological sections from a human premolar labeled with the neurochemical marker protein gene product (PGP) 9.5, showing the

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dental pulp and surrounding dentin. **A,** Nerve fibers entering the coronal pulp *(P)* form a dense network beneath the odontoblasts *(OB)*, known as the plexus of Raschkow *(*)*. **B,** Nerves are entering the apical area and pass through the radicular pulp as nerve bundles *(arrows)*. Attached and free denticles *(dt)* are seen close to dentin in the apical area. **C** and **D,** Compared to dense peripheral branching of nerves in the coronal pulp **(C)**, the branching of nerves in the radicular pulp is sparse **(D)**.

Four micrographs marked A through D show the nerve fibers close to the inner walls of a root canal, passing of nerve fibers through apical foramen, and comparison of nerves density in the last two micrographs.

Source: (Courtesy Dr. Inger Hals Kvinnsland.)

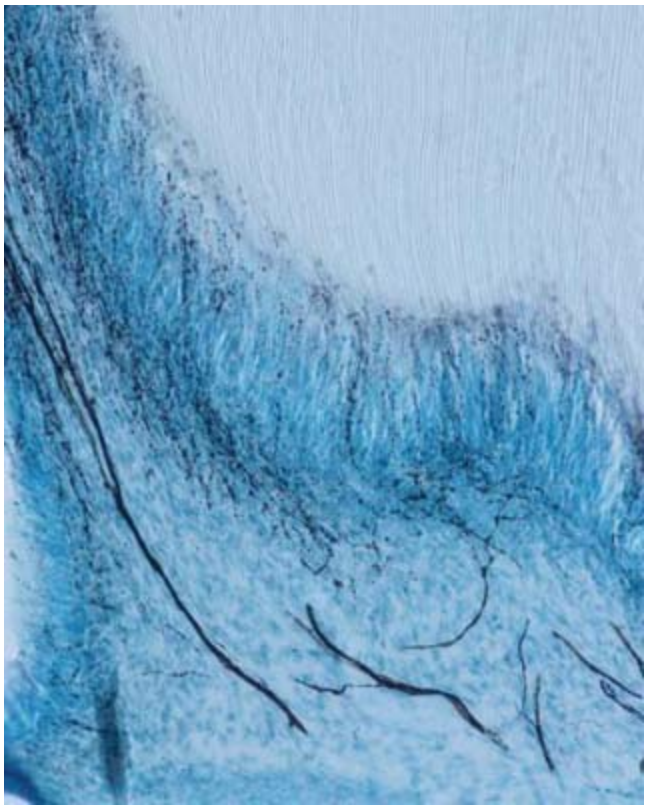

FIG. 13.20 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows the distribution of sensory nerves in a rat molar. Nerves enter the coronal pulp in bundles and ramify in a network beneath the odontoblasts (i.e., plexus of Raschkow), before entering between the odontoblasts and the inner part of dentin.

Histological section shows black thread-like structures beneath the columnar

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cells.

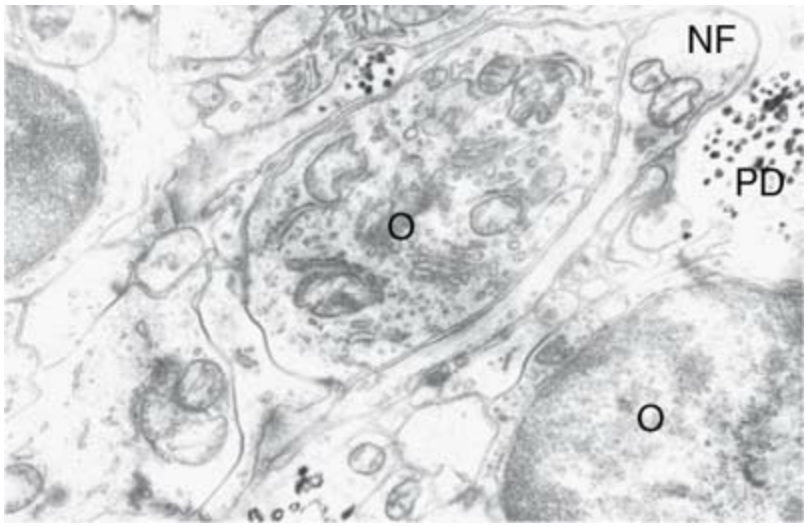

FIG. 13.21 Unmyelinated nerve fiber *(NF)* without a Schwann cell covering located between adjacent odontoblasts *(O)* overlying pulp horn of a mouse molar tooth. Predentin *(PD)* can be seen at upper right. Within the nerve, there are longitudinally oriented fine neurofilaments, microvesicles, and mitochondria.

Cross-section view shows the narrow canal of unmyelinated nerve fiber at the center surrounded by odontoblast cells with predentin cells present at the top right.

Source: (From Corpron RE, Avery JK: The ultrastructure of intradental nerves in developing mouse molars, *Anat Rec* 175:585, 1973.)

One investigator[133] studied the distribution and organization of nerve fibers in the dentin-pulp border zone of human teeth. On the basis of their location and pattern of branching, several types of nerve endings were described (Fig. 13.22). Some fibers were found running from the subodontoblastic nerve plexus toward the odontoblast layer. However, these fibers do not reach the predentin; they terminate in extracellular spaces in the cell-rich zone, the cell-poor zone, or the odontoblast layer. Other fibers extend into the predentin and run through a dentinal tubule in close association with an odontoblast process. Most of these intratubular fibers extend into the dentinal tubules for only a few micrometers, but a few may penetrate as far as 100 μm (see Figs. 13.19 and 13.20). The area covered by a

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single such terminal complex often reaches thousands of square micrometers.[133,273]

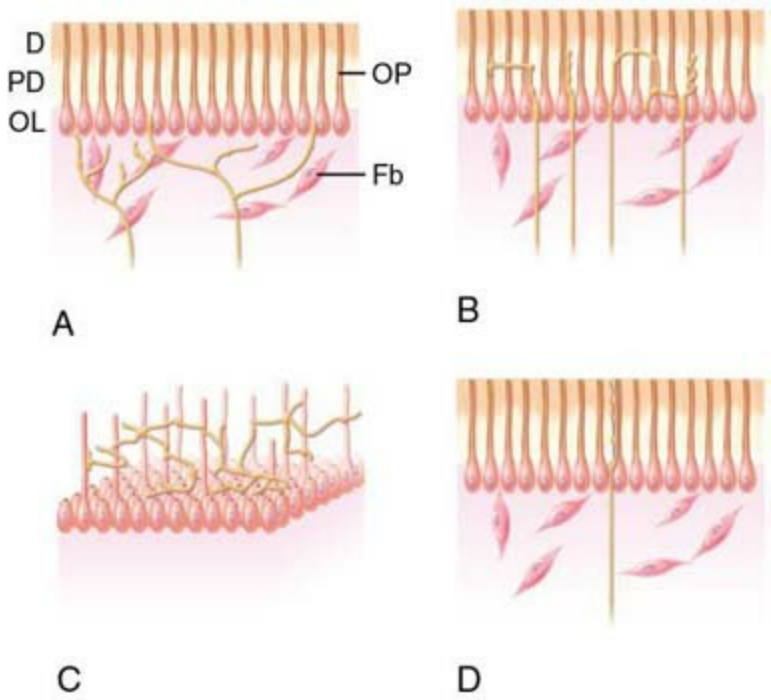

FIG. 13.22 Schematic drawing showing distribution of nerve fibers in the dentin-pulp border zone. **A,** Fibers running from the subodontoblastic plexus to the odontoblast layer. **B,** Fibers extending into the dentinal tubules in the predentin. **C,** Complex fibers that branch extensively in the predentin. **D,** Intratubular fibers extending into the dentin. *D,* Dentin; *Fb,* fibroblast; *OL,* odontoblast layer; *OP,* odontoblast process; *PD,* predentin.

Four drawings marked A through D explain the organization and branching pattern of nerve fibers in tooth.

Intratubular nerve endings are most numerous in the area of the pulp horns, where as many as 40% of the tubules may contain fibers.[47,219] The number of intratubular fibers decreases in other parts of the dentin, and in root dentin only about 1% of dentinal tubules contain fibers. This notion has been challenged in a study that stained pulps for protein gene-product 9.5, a specific marker for nerves.[235] In that study, root dentin appeared to be as well innervated as coronal dentin. The anatomic relationship between the odontoblast processes and sensory nerve endings has led to much speculation

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as to the functional relationships between these structures, if any.[46] When present, nerve fibers lie in a groove or gutter along the surface of the odontoblast process, and toward their terminal ends they twist around the process like a corkscrew. The cell membranes of the odontoblast process and the nerve fiber are closely approximated and run closely parallel for the length of their proximity, but they are not synaptically linked.[162]

Although it may be tempting to speculate that the odontoblasts and their associated nerve axons are functionally interrelated and that together they play a role in dentin sensitivity, there is a paucity of evidence supporting this hypothesis. If the odontoblast were acting as a classic receptor cell,[†] it would have chemical, electric, or mechanical communication with the adjacent nerve fiber. However, researchers have been unable to find classic anatomic structures (e.g., synaptic junctions) that could functionally couple odontoblasts and nerve fibers together. With regard to the membrane properties of odontoblasts, it has been reported that the membrane potential of the odontoblast is low (−24 to −30 mV),[202,231] and that the cell does not respond to electric stimulation.[292,413] It would appear that the odontoblast does not possess the properties of an excitable cell. Further, the sensitivity of dentin is not diminished after disruption of the odontoblast layer.[38,220] It is still possible that odontoblasts could modulate neuronal function via alteration in sodium channel activity or the release of paracrine factors that diffuse to the closely approximated nerve terminal.

Another study showed that a reduction in pulpal blood flow (PBF), induced by stimulation of sympathetic fibers leading to the pulp, results in depressed excitability of pulpal A fibers.[83] The excitability of C fibers is less affected than that of A fibers by a reduction in blood flow.[370]

Of clinical interest is the evidence that nerve fibers of the pulp may be resistant to necrosis[83,260] because their cell bodies are found in ganglia outside the pulp. Because nerve bundles in general are more resistant to autolysis than other tissue elements, even in degenerating pulps, C fibers might still be able to respond to noxious stimulation. It may be that C fibers remain excitable even after blood flow has been compromised in the diseased pulp, as C fibers are often able to function in the presence of hypoxia.[370] This may explain why instrumentation of the root canals of apparently nonvital teeth sometimes elicits pain. On the other hand, histologic studies on nonvital

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teeth failed to demonstrate high levels of innervation, leading to the suggestion that pain may be due to the transfer of noxious chemicals to terminals located in periapical tissues.[260]

Steps and mechanisms in pain perception

When activated by a stimulus sufficient to cause tissue damage or release of inflammatory mediators, nerve endings in the pulp and periradicular tissues begin to send bursts of messages to the central nervous system (CNS) that may eventually be perceived as pain. The anatomic pathway for this transmission of information has been fairly well established, and it is tempting to view the perception of pain of orofacial origin as a simple graded response to the intensity of the stimulus. However, researchers have come to realize that the pain system is a complex, multilevel system that begins with the *detection* of tissue-damaging stimuli in the periphery, the *processing* of that input at the level of the medullary spinal cord, and the *perception* of what is felt as pain in higher brain regions such as the cerebral cortex (Fig. 13.23). After a noxious stimulus is detected in the periphery, there is ample opportunity for a great deal of endogenous and possibly exogenous modification of the message prior to its ultimate perception. The clinician deals with all three levels of the pain system in diagnosing and treating odontalgia, and a practitioner with a basic understanding of each level will be able to recognize therapeutic opportunities and apply effective pain control methods.

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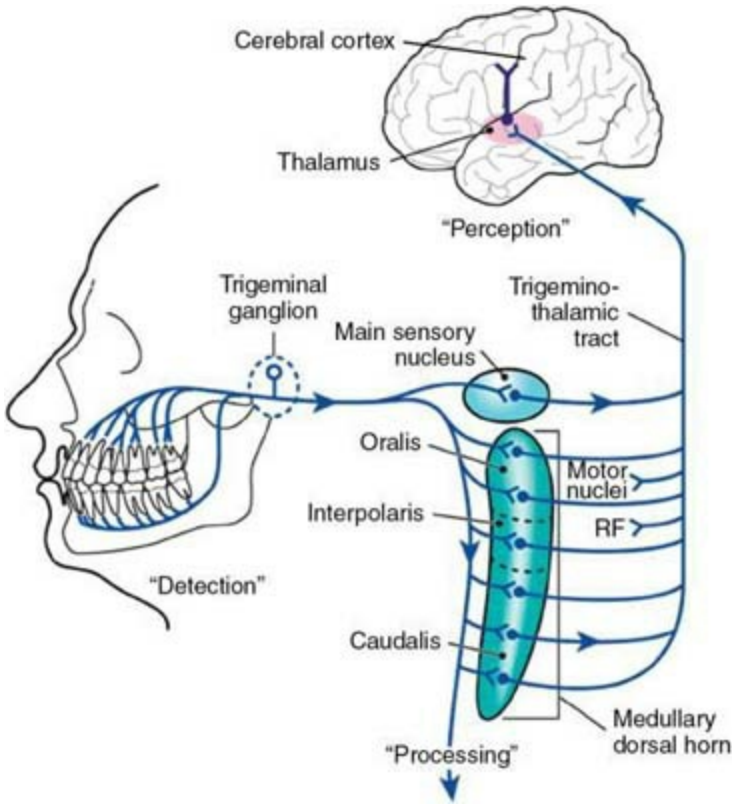

FIG. 13.23 Schematic diagram of the pathway of transmission of nociceptive information from the orofacial region. The trigeminal pain system is a complex multilevel system that begins with the *detection* of tissue damaging stimuli in the periphery, the *processing* of that input at the level of the medullary spinal cord, and the final *perception* of what is felt as pain in the cerebral cortex. There is growing appreciation for the concept that once a noxious stimulus is detected in the periphery, there is the opportunity for a great deal of modification of the message prior to ultimate perception. *RF,* Reticular formation.

Damaged tissue is detected in the lower face, after which trigeminal ganglion leads to processing in medullary dorsal horn and main sensory nucleus. Labels in medullary dorsal horn are as follows: oralis interpolaris, and caudalis. Motor nuclei and RF along with main sensory nucleus are collectively leading to trigeminothalamic tract followed by perception in brain. Two labels in brain are cerebral cortex and thalamus.

Detection: The first step in pain perception

Various types of peripheral neurons are found in the trigeminal system, including large-diameter, heavily myelinated Aα, Aβ, and Aγ fibers associated with motor, proprioception, touch, pressure, and muscle spindle

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stretch functions. But it is the smaller, less myelinated Aδ and yet smaller and unmyelinated C fibers that conduct information likely to be perceived as pain. These two classes of pain-sensing nerve fibers, or *nociceptors*, are both found in the tooth pulp, but there are three to eight times more unmyelinated C fibers than A-δ fibers.[42,46,172,376] It should be noted that this classification system is based purely on the size and myelination of the neurons and does not necessarily indicate function. For example, another class of pulpal C fibers are the postganglionic sympathetic efferents found in association with blood vessels, where they regulate PBF[2,190,305,306] and may also influence the activity of peripheral nociceptors (for reviews, see Perl[302] or Hargreaves[139]). Because most pulpal sensory fibers are nociceptive, their terminal branches are free nerve endings, and physiologic stimulation by any modality (temperature, hyperosmotic fluids) results in the perception of pure pain, which can be difficult for patients to localize. Under experimental conditions, electrical stimulation can result in a *prepain* sensation that is also difficult to localize. Once inflammation has extended to the periodontal ligament, which is well endowed with Aβ discriminative touch receptors, the localization of pain is more predictable with light mechanical stimuli such as the percussion test.

In the normal uninflamed pulp and periradicular tissues, a noxious stimulus causes depolarization of nociceptors sufficient to generate action potentials by means of the opening of voltage-gated sodium channels (Na_v). After generation of an action potential, not only is information sent to the CNS, but also in an *antidromic* fashion (i.e., in the reverse direction of the impulse, also termed as an axon reflex). In this way, proinflammatory neuropeptides such as substance P (SP), calcitonin gene-related peptide (CGRP), neurokinins, and the classic neurotransmitter, glutamate, are released from afferent terminals in the pulp and periradicular tissues, even including neighboring teeth (see Fig. 13.18).

Neuropeptides

Of immense importance in pulp biology is the presence of neuropeptides in pulpal nerves.[43,47,50,356] Pulpal nerve fibers contain neuropeptides such as CGRP (see Figs. 13.16 and 13.20),[42] SP,[273,291] NPY (see Fig. 13.14),

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neurokinin A (NKA),[12] and VIP.[233,396] In rat molars, the largest group of intradental sensory fibers contains CGRP. Some of these fibers also contain other peptides, such as SP and NKA.[43,46] Release of these peptides can be triggered by numerous stimuli, including tissue injury,[12] complement activation, antigen-antibody reactions,[286] or antidromic stimulation of the inferior alveolar nerve.[288,291] Once released, vasoactive peptides produce vascular changes that are similar to those evoked by histamine and bradykinin (BK) (i.e., vasodilation).[288] In addition to their neurovascular properties, SP and CGRP contribute to inflammation and promote wound healing.[32,374] The release of CGRP can be modified by sympathetic agonists and antagonists,[117,140] offering the promise of using such agonists to treat dental pain. This latter point is important because clinicians use sympathetic agonists every day—the vasoconstrictors present in local anesthetic solutions may have direct effects on inhibiting dental nerve activity. Local anesthetic reduction in pain may be due to the actions of both the local anesthetic and the vasoconstrictor.[139,140] In cats, capsaicin acutely activates and chronically blocks the *transient receptor potential subtype V1* (TRPV1)-expressing classes of C and A-δ nociceptors in the pulp.[165] In addition, the chronic application of capsaicin ointment to skin has been shown to relieve pain in patients, suggesting that clinical trials evaluating chronic application of capsaicin for treating pulpal or periradicular pain may be of value.

It has been reported[251,291] that mechanical stimulation of dentin produces vasodilation within the pulp, presumably by causing the release of neuropeptides from intradental sensory fibers (neurogenic inflammation).[291] Electric stimulation of the tooth has a similar effect.[154] The pulpal concentrations of CGRP, SP, and NKA are elevated in painful human teeth over healthy control teeth extracted for orthodontic reasons.[12] These peptides are also elevated in pulps beneath advancing carious lesions.[316,317]

Pulp testing

The electric pulp tester delivers a current sufficient to overcome the resistance of enamel and dentin and stimulate the sensory A fibers at the dentin-pulp border zone. Smaller C fibers of the pulp do not respond to the conventional pulp tester because significantly more current is needed to

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stimulate them.[272] Bender and associates[17] found that in anterior teeth, the optimal placement site of the electrode is the incisal edge, as the response threshold is lowest at that location and increases as the electrode is moved toward the cervical region of the tooth.

Cold tests using CO_2 snow or liquid refrigerants and heat tests employing heated gutta-percha or hot water activate hydrodynamic forces within the dentinal tubules, which in turn excite the intradental A fibers. C fibers are generally not activated by these tests unless they produce injury to the pulp. It has been shown that cold tests do not injure the pulp.[110] Heat tests have a greater potential to produce injury, but if the tests are used properly, injury is not likely.

Sensitivity of dentin

The mechanisms underlying dentin sensitivity have been a subject of interest for many years. How are stimuli relayed from the peripheral dentin to the sensory terminals located in the region of the dentin-pulp border zone? Converging evidence indicates that movement of fluid in the dentinal tubules is the basic event in the arousal of dentinal pain.[36,38,268,392,394,395] It now appears that pain-producing stimuli, such as heat, cold, air blasts, and probing with the tip of an explorer, have in common the ability to displace fluid in the tubules.[36,251] This is referred to as the *hydrodynamic mechanism of dentin sensitivity*. The hydrodynamic theory suggests that dentinal pain associated with stimulation of a sensitive tooth ultimately involves mechanotransduction. Classical mechanotransducers have been recognized on pulpal afferents, providing a mechanistic support to this theory.[151] Thus fluid movement in the dentinal tubules is translated into electric signals by receptors located in the axon terminals innervating dentinal tubules. Using single-fiber recording techniques, a positive correlation was found between the degree of pressure change and the number of nerve impulses leaving the pulp (Figs. 13.24 and 13.25).[251,271,391] Thus the outward fluid movements (negative pressure) produce a much stronger nerve response than inward movements.[251,394]

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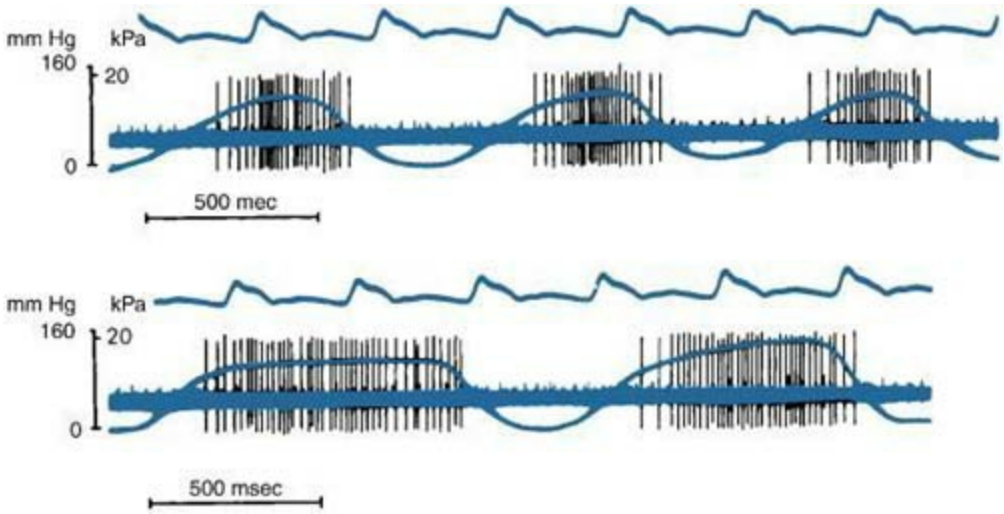

FIG. 13.24 Response of a single dog pulp nerve fiber to repeated hydrostatic pressure stimulation pulses. Lower solid wavy line of each recording indicates the stimulation pressure applied to the pulp. Upper line *(kPa)* is the femoral artery blood pressure curve recorded to indicate the relative changes in the pulse pressure and the heart cycle.

First recording shows vertical axis marked 0 to 160 on the left and 20 near the top right in mm Hg. It has three crests and two troughs. The upper oscillating line measures kPa. The scale reads, 500 msec. Second recording shows two wide crests and one trough only.

Source: (Modified from Närhi M: Activation of dental pulp nerves of the cat and the dog with hydrostatic pressure, *Proc Finn Dent Soc* 74[suppl 5]:1, 1978.)

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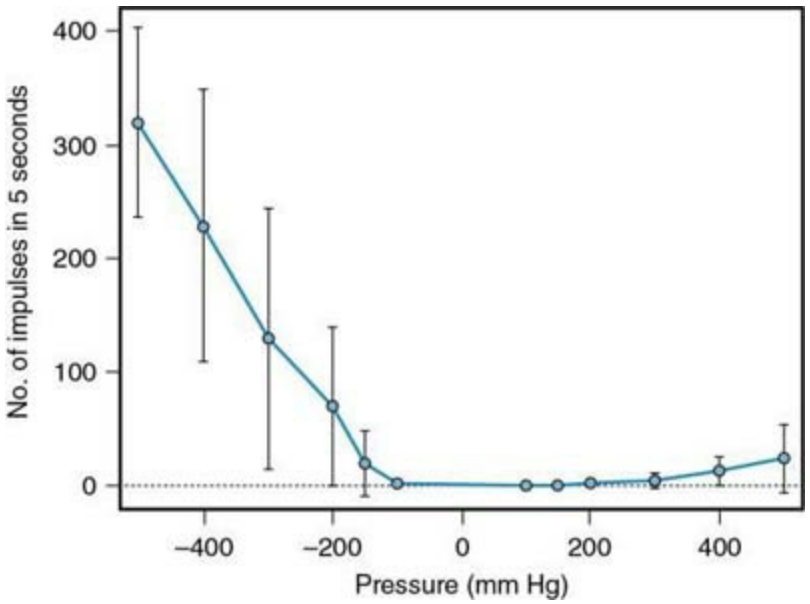

FIG. 13.25 The average number of impulses recorded from dentine after application of pressure stimuli to the dentine. More impulses are recorded after application of negative pressure (outward fluid flow) than after positive pressure (inward fluid flow).

Graph plots number of impulses in 5 seconds against pressure in mm Hg. The vertical axis ranges from 0 near the base to 400 at the top. The horizontal axis ranges from negative 400 (near the base) to positive 400. The data points are as follows: (negative 480, 320), (negative 400, 230), (negative 300, 120), (negative 200, 70), (negative 150, 20), (negative 110, 0), (positive 110, 0), (positive 150, 0), (positive 200, 0), (positive 250, 5), (positive 400, 10), and (positive 500, 20). All data are approximate.

Source: (From Vongsavan N, Matthews B: The relationship between the discharge of intradental nerves and the rate of fluid flow through dentine in the cat, *Arch Oral Biol* 52:643, 2007.)

In experiments on humans, brief application of heat or cold to the outer surface of premolar teeth evoked a painful response before the heat or cold could have produced temperature changes capable of activating sensory receptors in the underlying pulp.[271,377] The evoked pain was of short duration: 1 or 2 seconds. The thermal diffusivity of dentin is relatively low, yet the response of the tooth to thermal stimulation is rapid, often less than 1 second. Evidence suggests that thermal stimulation of the tooth results in a rapid movement of fluid into the dentinal tubules. This results in activation of the sensory nerve terminal in the underlying pulp. Presumably, heat expands the fluid within the tubules faster than it expands dentin, causing the fluid to

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flow toward the pulp, whereas cold causes the fluid to contract more rapidly than dentin, producing an outward flow. It is speculated that the rapid movement of fluid across the cell membrane of the axon terminal activates a mechanosensitive receptor, similar to how fluid movement activates hair cells in the cochlea of the ear. All axon terminals have membrane channels through which charged ions pass, and this initial receptor current, if sufficient, can trigger voltage-gated sodium channels to depolarize the cell, leading to a barrage of impulses to the brain. Some ion channels are activated by voltage, some by chemicals, and some by mechanical pressure.[251,268,273] In the case of pulpal nerve fibers that are activated by hydrodynamic forces, pressure would be transduced, gating mechanosensitive ion channels.

The dentinal tubule is a capillary tube with an exceedingly small diameter.[‡,113] The physical properties of capillarity are significant because fluid force increases as the diameter is reduced. If fluid is removed from the outer end of exposed dentinal tubules by dehydrating the dentinal surface with an air blast or absorbent paper, capillary forces produce a rapid outward movement of fluid in the tubule (Fig. 13.26). According to Brännström,[36] desiccation of dentin can theoretically cause dentinal fluid to flow outward at a rate of 2 to 3 mm/s. In addition to air blasts, dehydrating solutions containing hyperosmotic concentrations of sucrose or calcium chloride can produce pain if applied to exposed dentin.

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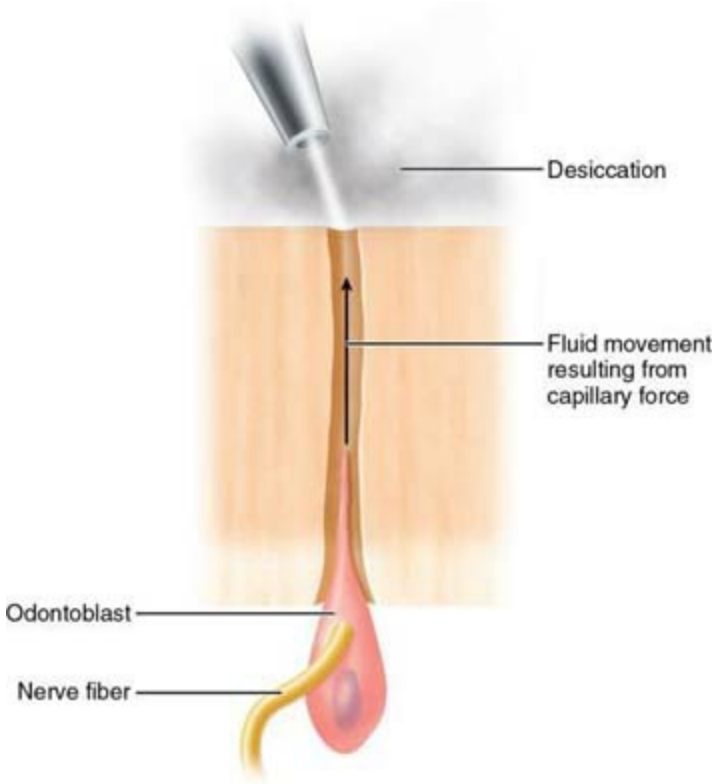

FIG. 13.26 Diagram illustrating movement of fluid in the dentinal tubules resulting from the dehydrating effect of a blast of air from an air syringe.

Pulp of tooth undergoing desiccation with labels as follows: Nerve fiber, odontoblast, and fluid movement resulting from capillary force.

Investigators have shown that it is the A fibers rather than the C fibers that are activated by hydrodynamic stimuli (e.g., heat, cold, air blasts) applied to exposed dentin.[270,278] However, if heat is applied long enough to increase the temperature of the dentin-pulp border by several degrees Celsius, then C fibers may respond, particularly if the heat produces injury. It seems that the A fibers are mainly activated by a rapid displacement of the tubular contents.[268] Slow heating of the tooth produced no response until the temperature reached 111°F (43.8°C), at which time C fibers were activated, presumably because of heat-induced injury to the pulp. These C fibers are called *polymodal nociceptors* because they contain numerous receptors that confer the ability to detect and respond to many different types of stimuli.[273,274] Capsaicin, the pungent active ingredient in hot peppers, is known to simulate C fibers and a subset of A-δ fibers.[165] Capsaicin activates

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a receptor termed the “TRP, subtype vanilloid 1” or TRPV1.[59] The TRPV1 receptor is expressed primarily on a major subclass of nociceptors and responds to heat (>110°F [>43°C]), certain inflammatory mediators, and acid (pH < 6). Thus TRPV1 has been considered a molecular integrator of polymodal noxious stimuli.[283] The ability of the TRPV1 antagonist, capsazepine, to inhibit acid-, heat-, and capsaicin-activated trigeminal neurons[225] has led to the development of new drugs (i.e., TRPV1 antagonists) for the treatment of pulpal pain. Eugenol is known to activate and ultimately desensitize TRPV1, and this may explain the anodyne action of zinc oxide eugenol temporary restorations.[422]

It has also been shown that pain-producing stimuli are more readily transmitted from the dentin surface when the exposed tubule apertures are open[159,173] and the fluid within the tubules is free to flow outward.[173,251,394] For example, acid treatment of exposed dentin to remove the smear layer opens the tubule orifices and makes the dentin much more responsive to stimuli such as air blasts and probing.[159,274]

Perhaps the most difficult phenomenon to explain is dentinal pain associated with light probing of dentin. Even light pressure of an explorer tip can produce strong forces.[§] These forces have been shown to mechanically compress dentin and close open tubule orifices with a smear layer that causes sufficient displacement of fluid to excite the sensory receptors in the underlying pulp (Fig. 13.27).[55,56] Considering the density of the tubules in which hydrodynamic forces would be generated by probing, multiple nerve endings would be simultaneously stimulated when a dental explorer is scratched across dentin. Another newly suggested explanation is that the nerves innervating teeth are special in nature and that low-threshold mechanoreceptors are signaling nociceptive input in teeth.[105] Similar low-threshold mechanoreceptors convey tactile sensation in skin. The authors used the term low-threshold “algoneurons” for these nerves. This theory is not in conflict with the hydrodynamic theory but may help to explain the sensation of pain felt after weak mechanical stimuli like air puffs and water spray.

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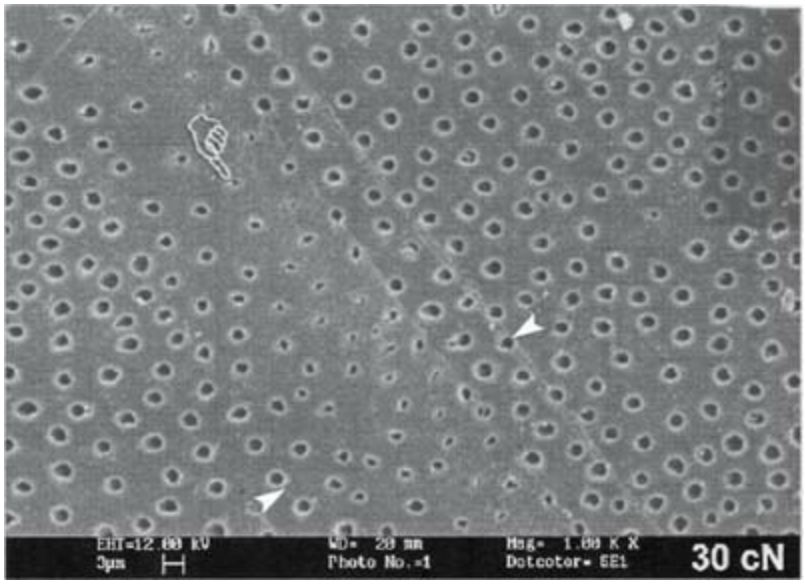

FIG. 13.27 Scanning electron micrograph of a shallow groove *(between white arrowheads)* created in polished dentin by a dental explorer tine under a force of 30 g (30 cN). Note the partial occlusion of the tubules by smeared matrix.

SEM shows the matrix of rounded cells faded at the center.

Source: (From Camps J, Salomon JP, Meerbeek BV, et al: Dentin deformation after scratching with clinically relevant forces, *Arch Oral Biol* 48:527, 2003.)

Another example of the effect of strong hydraulic forces that are created within the dentinal tubules is the phenomenon of odontoblast displacement. In this reaction, the nuclei and cell bodies of odontoblasts are displaced upward in the dentinal tubules, presumably by a rapid movement of fluid in the tubules produced when exposed dentin is desiccated, as with the use of an air syringe or cavity-drying agents (Fig. 13.28).[206] Such cellular displacement results in the destruction of odontoblasts, because cells thus affected soon undergo autolysis and disappear from the tubules. Displaced odontoblasts may eventually be replaced by stem cells that migrate from the cell-rich zone of the pulp, as discussed later in the chapter.

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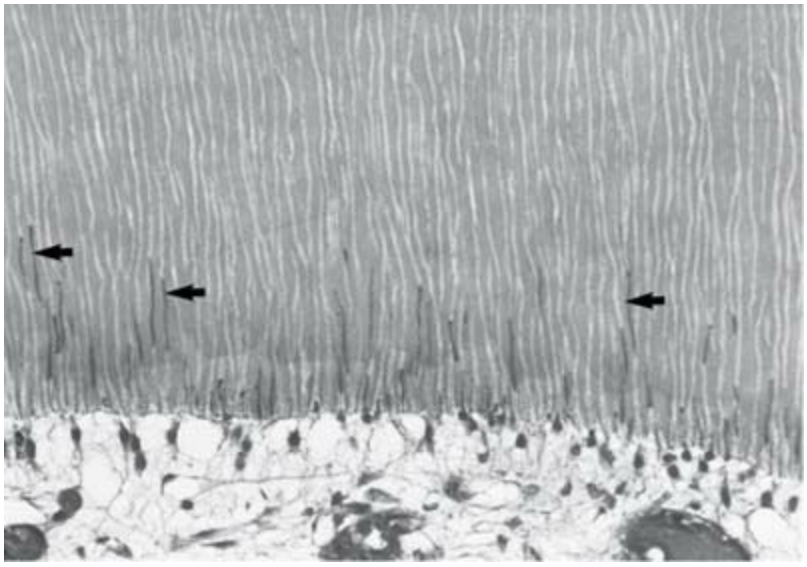

FIG. 13.28 Odontoblasts *(arrows)* displaced upward into the dentinal tubules.

Micrograph shows dark thread-like structures in dentin layer of tooth.

The hydrodynamic theory can also be applied to an understanding of the mechanism responsible for hypersensitive dentin.[37,38] There is controversy regarding whether exposed dentin is simply sensitive or becomes truly hypersensitive.[273,274,278] Growing evidence indicates that new sodium channels responsible for activating nerves are expressed in nerve tissue exposed to inflammation.[121,139,314] An increase in the density of sodium channels or their sensitivity may contribute to dentinal hypersensitivity. Hypersensitive dentin is also associated with the exposure of dentin normally covered by cementum or enamel. The thin layer of cementum is frequently lost as gingival recession exposes cementum to the oral environment. Cementum is subsequently worn away by brushing, flossing, or using toothpicks. Once exposed, the dentin may respond to the same stimuli to which any exposed dentin surface responds (e.g., mechanical pressure, dehydrating agents). Although the dentin may at first be very sensitive, within a few weeks the sensitivity usually subsides. This desensitization is thought to occur as a result of gradual occlusion of the tubules by mineral deposits, thus reducing hydrodynamic forces. In addition, deposition of reparative dentin over the pulpal ends of the exposed tubules probably also reduces sensitivity, because reparative dentin is less innervated by sensory

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nerve fibers.[45] However, some hypersensitive dentin does not spontaneously desensitize, so hypersensitivity may be caused by either inflammatory changes in the pulp or mechanical changes in the patency of dentinal tubules.

Currently, the treatment of hypersensitive teeth is directed toward reducing the functional diameter of the dentinal tubules to limit fluid movement. Four possible treatment modalities[293,378] can accomplish this objective:

1. Formation of a smear layer on the sensitive dentin by burnishing the exposed root surface[159,273,274]
2. Application of agents, such as oxalate compounds, that form insoluble precipitates within the tubules[159,298]
3. Application of agents such as hydroxyethyl methacrylate (HEMA) with or without glutaraldehyde that are thought to occlude tubules with precipitated plasma proteins in dentinal fluid[331]
4. Application of dentin bonding agents to seal off the tubules[309]

Dentin sensitivity can be modified by laser irradiation, but clinicians must be concerned about its effect on the pulp.[353,363]

Peripheral sensitization

Following repeated noxious stimuli, both A and polymodal C fiber nociceptors undergo a process of sensitization manifested by three obvious changes in response patterns. First, firing thresholds may decrease, so that previously non-noxious stimuli may trigger discharges, contributing to the sensation of pain *(allodynia).* Second, after-discharges may occur, so that noxious stimuli may produce an even greater increase in the perceived intensity of pain *(hyperalgesia).* And third, firing may occur spontaneously, contributing to the development of spontaneous pain. These changes are often seen in endodontic pain patients and may be explained in part by the effects of chemical mediators released into inflamed pulp and periradicular tissues. Such mediators include substances produced from damaged tissues, agents of vascular origin, and peptides released from the nerve fibers themselves (Table 13.3). Other mechanisms of peripheral sensitization are listed in Box 13.1.

Table 13.3

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Effect of Inflammatory Mediators on Nociceptive Afferent Fibers

Mediator	Effect on Nociceptors	Effect on Human Volunteers
Potassium[176]	Activate	++
Protons[222,350]	Activate	++
Serotonin[16,176]	Activate	++
Bradykinin[16,176,215]	Activate	+++
Histamine[176]	Activate	+
Tumor necrosis factor-α	Activate	?
Prostaglandins[27]	Sensitize	±
Leukotrienes[27,234]	Sensitize	±
Nerve growth factor[217,303]	Sensitize	++
Substance P[134]	Sensitize	±
Interleukin 1[101]	Sensitize (?)	?

+, Positive; ++, very positive; +++, extremely positive; ±, equivalent; ?, unknown.

Modified from Fields H: *Pain,* New York, 1987, McGraw-Hill.

Box 13.1

Peripheral Mechanisms Contributing to Hyperalgesia and Allodynia

- Composition and concentration of inflammatory mediators[144]
- Changes in afferent fiber: activation and sensitization[203,248,330]
- Changes in afferent fiber: sprouting[52]
- Changes in afferent fiber: proteins[52,121,416]
- Tissue pressure[269,270]
- Tissue temperature[255]
- Sympathetic primary afferent fiber interactions[168,214,301]
- Aβ fiber plasticity [277]

Modified from Hargreaves KM, Swift JQ, Roszkowski MT, et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release, *Oral Surg Oral Med Oral Pathol* 78:503, 1994.

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Hyperalgesia and allodynia

Three characteristics of hyperalgesia are (1) spontaneous pain, (2) a decreased pain threshold, and (3) an increased response to a painful stimulus.[139] The peripheral mechanisms for these symptoms include a decrease in firing threshold, an increase in responsiveness to noxious stimuli, and development of spontaneous discharges of nociceptors. All three of these characteristics can be seen in patients experiencing inflammatory pain of pulpal origin (Table 13.4). It is recognized that hyperalgesia can be produced by sustained inflammation, as in the case of sunburned skin. Clinical observation has shown that the sensitivity of dentin often increases when the underlying pulp becomes acutely inflamed, and the tooth may be more difficult to anesthetize. This is due in part to the upregulation of tetrodotoxin-resistant (TTX-resistant) sodium channels in inflamed neural tissue.[121,139] NGF also seems to play an important role in hyperalgesia.[303] NGF regulates chronic inflammatory hyperalgesia by controlling gene expression in sensory neurons,[279] including genes involved in inflammatory hyperalgesia in the dental pulp.[75] Although a precise explanation for hyperalgesia is lacking, apparently localized elevations in tissue pressure and inflammatory mediators that accompany acute inflammation play an important role.[153,156,351,367,384] Clinically, we know that when the pulp chamber of a painful tooth with an abscessed pulp is opened, drainage of exudate soon produces a reduction in the level of pain. This suggests that mechanical stimuli may contribute substantially to pain during inflammatory hyperalgesia.

Table 13.4

Signs of Hyperalgesia and Allodynia and Endodontic Diagnostic Tests

Signs of Hyperalgesia	Related Diagnostic Tests or Symptoms
Spontaneous pain	Spontaneous pain
Reduced pain threshold	Percussion test, palpation test, throbbing pain
Increased response to painful stimuli	Increased response to pulp test (electric or thermal test)

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From Hargreaves KM, Swift JQ, Roszkowski MT, et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release, *Oral Surg Oral Med Oral Pathol* 78:503, 1994.

From a clinical point of view, *thermal allodynia* is the term that best describes a patient whose chief complaint is, “I have pain when I drink cold beverages.” *Mechanical allodynia* is involved when the chief complaint is “It now hurts when I bite on this tooth.” These previously nonnoxious stimuli now cause the perception of pain. Hyperalgesia is manifested in endodontic pain patients when noxious stimuli (e.g., refrigerant sprays or CO_2 snow used in the cold test) produce much more pain than they would in teeth with normal pulp tissues. Spontaneous pain involves episodes of pain that seem to be unprovoked. All these changes can be partly explained by sensitization of peripheral nerve endings in the pulp and periradicular tissues.

Many silent nerve fibers are present in the normal pulp[273,274] and are termed *silent* because they are not excited by ordinary external stimuli. Once they are sensitized through pulpal inflammation, they begin to respond to hydrodynamic stimuli.[47,273,274,278] This phenomenon may provide an additional mechanism for dentin hypersensitivity. The molecular mechanisms of this activation are not known in detail but involve the upregulation of numerous genes and their products.[12,46,121]

Inflammatory mediators

Among the best characterized of the inflammatory mediators are the prostaglandins (PGs), which are derived from arachidonic acid via the action of the cyclooxygenase (COX) enzyme systems. The human COX enzyme is known to exist in at least two forms, COX-1 and COX-2. COX-1 is constitutively expressed and produces PGs that are involved in basic housekeeping functions such as cytoprotection in the stomach, regulation of blood flow in the kidneys, and the formation of thromboxane A_2. The formation of thromboxane A_2 can ultimately lead to platelet aggregation; therefore inhibition of thromboxane A_2 should decrease platelet aggregation. COX-2 is inducible, synthesized in inflamed tissues (including dental pulp),[267] and is important in the production of the proinflammatory PGs as well as the vasodilating prostacyclin (PGI_2). Although they do not produce

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pain if applied alone, PGs are known to sensitize peripheral nociceptors, which increases the algogenic (pain-producing) properties of serotonin and BK.[77,126] The exact mechanism by which PGs increase neuronal excitability is not clear, but there is a growing body of evidence to suggest that they activate the PG E receptor subtypes EP2 and EP3 in the trigeminal system[299] and exert their effects by regulating the activity of certain ion channels,[401] including voltage-gated sodium channels (for a review, see England[88]). For example, application of prostaglandin E_2 (PGE_2) to isolated dorsal root ganglion neuronal somata more than doubles the responsiveness of certain sodium channels found predominantly on nociceptors—channels thought to be relatively resistant to lidocaine.[9,122] When administered to rats before an inflammatory insult, ibuprofen, a nonselective COX inhibitor, has been shown to block the increased expression of $Na_v1.7$ and $Na_v1.8$.[129,130] Therefore if the concentrations of PGs in inflamed pulp and periradicular tissues can be decreased with nonsteroidal antiinflammatory drugs (NSAIDs) or corticosteroids, postoperative pain may be relieved; in addition, more profound local anesthesia may be achieved in patients with hyperalgesia of pulpal origin.[142,163,259] It is interesting to note that sensory neurons themselves are a source of PGs; during inflammation, the levels of PGE_2 appear to increase in dorsal root ganglia and spinal cord, suggesting that NSAIDs also have a central site of action (discussed in Chapter 6).[240,412]

BK is a proinflammatory mediator derived from circulating plasma proteins and also causes direct activation of nociceptive neurons, resulting in pain. Increased levels of BK have been demonstrated in the inflamed dental pulp,[211] and the presence of growth factors associated with inflammation (e.g., NGF) have been reported to cause an increase in the expression of mRNA encoding B1 and B2 receptors in primary cultures of rat trigeminal ganglia,[368] as well as other receptors such as TRPV1 and TRPA1.[75,170] The TRPV1 is the “capsaicin receptor”; it plays a key role in mediating inflammatory pain. TRPA1 is expressed on capsaicin-sensitive neurons[75] and interacts with TRPV1.[320] BK likely increases the excitability of nociceptive neurons through its action on TRPV1 and TRPA1 (for review, see Tominaga et al.[364]).

Cytokines are a diverse group of regulatory proteins synthesized and

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secreted by a variety of cell types, such as leukocytes, neurons, and glia. In particular, TNF-α and the interleukins IL-1β, IL-6, and IL-8 are thought to play a role in the neuroplastic changes that occur in nociceptors innervating inflamed tissues, leading to hyperalgesia.[201] Application of TNF-α rapidly sensitizes TRPV1,[185] contributing to activation of the capsaicin-sensitive class of nociceptors. All of these are thought to exist in the inflamed pulp (for review, see Fouad[102]) and are thought to act at least in part by causing increased release of prostanoids.[354]

Painful pulpitis

From the foregoing, it is apparent that pain associated with the stimulation of the A fibers does not necessarily signify that the pulp is inflamed or that tissue injury has occurred. Clinically, pain produced by A fibers in response to the hydrodynamic mechanism has a sharp or bright quality as contrasted with the dull, boring, or throbbing pain associated with C fibers. A fibers have a relatively low threshold of excitability to external stimuli,[251,272] and painful pulpitis is more likely to be associated with nociceptive C fiber activity indicative of pulpal tissue injury.[272–274,278] The clinician should carefully examine symptomatic teeth to rule out the possibility of hypersensitive dentin, cracked or leaky fillings, or fracture lines—each of which may initiate hydrodynamic forces—before establishing a diagnosis of reversible or irreversible pulpitis (see also Chapters 1 and 22).

Pain associated with an inflamed or degenerating pulp may be either provoked or spontaneous. The hyperalgesic pulp may demonstrate a lowered threshold of pain by responding to stimuli that usually do not evoke pain (allodynia), or the pain may be exaggerated and persist longer than normal (hyperalgesia).[6] On the other hand, the tooth may commence to ache spontaneously in the absence of any external stimulus.[139] Spontaneous, unprovoked pain generally indicates a pulp that is seriously damaged and generally will not respond to noninvasive therapy.

Plasticity of intradental nerve fibers

It has become apparent that the innervation of the tooth is a dynamic process in which the number, size, and cytochemistry of nerve fibers can change

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because of aging,[104,227,356] tooth injury,[43,46,47,50] and dental caries.[316] For example, in rats, nerve fibers sprout into inflamed tissues surrounding sites of pulpal injury, and the content of CGRP and SP increases in these sprouting fibers.[43,46,47,50,54] When inflammation subsides, the number of sprouts decreases. Fig. 13.29 compares the normal distribution of CGRP-immunoreactive sensory fibers in an adult rat molar with those beneath a shallow cavity preparation. The innervational pattern in normal and inflamed teeth is governed by neuronal growth factors. Neurotrophic and target-derived factors regulate neuronal structure, survival, and function, and are important for the maintenance of neuronal phenotype characteristics. During development, all dental fibers appear to require NGF and express its receptor, tropomyosin receptor kinase A (TrkA),, at some stages,[249] whereas in adult teeth the large trigeminal neurons are potentially dependent only on dental pulp–derived, glial cell line–derived neurotrophic factor (GDNF); the smaller trigeminal neurons remain dependent on NGF.[204,311] This suggests that GDNF may function as a neurotrophic factor for the subset of larger neurons supporting the tooth, which apparently mediate mechanosensitive stimuli, whereas NGF is suggested to support neurons responsible for nociception.[264] NGF is the most extensively investigated among the trophic factors.[213] Binding of target-derived NGF is dependent on specific TrkA receptors located on the axonal surface, with subsequent internalization and transport to the cell body, where the effects are mediated.

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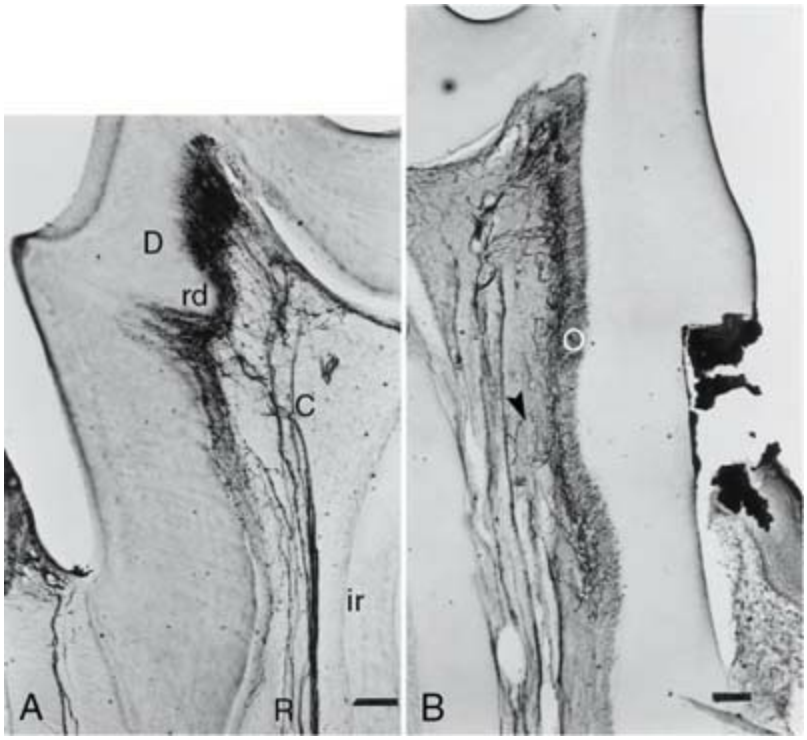

FIG. 13.29 **A,** Normal distribution of calcitonin gene–related peptide (CGRP)-immunoreactive sensory fibers in adult rat molar. Nerve fibers typically are unbranched in the root *(R)*, avoid interradicular dentin *(ir)*, and form many branches in coronal pulp *(C)* and dentin *(D)*. Nerve distribution is often asymmetric, with endings concentrated near the most columnar odontoblasts (in this case on the left side of the crown). When reparative dentin *(rd)* forms, it alters conditions so that dentinal innervation is reduced. **B,** Shallow class I cavity preparation on the cervical root of a rat molar was made 4 days earlier. Primary odontoblast *(O)* layer survived, and many new CGRP-immunoreactive terminal branches spread beneath and into the injured pulp and dentin. Terminal arbor can be seen branching *(arrowhead)* from a larger axon and growing into the injury site. Scale bar: 0.1 mm.

Two electron micrographs marked A and B compare the location and branching pattern of CGRP in the molar tooth of adult rat.

Source: (From Taylor PE, Byers MR and Redd PE: Sprouting of CGRP nerve fibers in response to dentin injury in rat molars, *Brain Res* 461:371–376, 1988.)

Regulation of neural changes during inflammation seems to be a function of NGF expression.[48,54] NGF receptors are found on intradental sensory fibers and Schwann cells.[46] Evidence indicates that NGF is synthesized by fibroblasts in the coronal subodontoblastic zone (i.e., cell-rich zone),

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particularly in the tip of the pulp horn.[46] Maximal sprouting of CGRP- and SP-containing nerve fibers corresponds to areas of the pulp with increased production of NGF.[54] Fig. 13.30 shows the expression of NGF-mRNA in a pulp horn subjacent to cavity preparation.

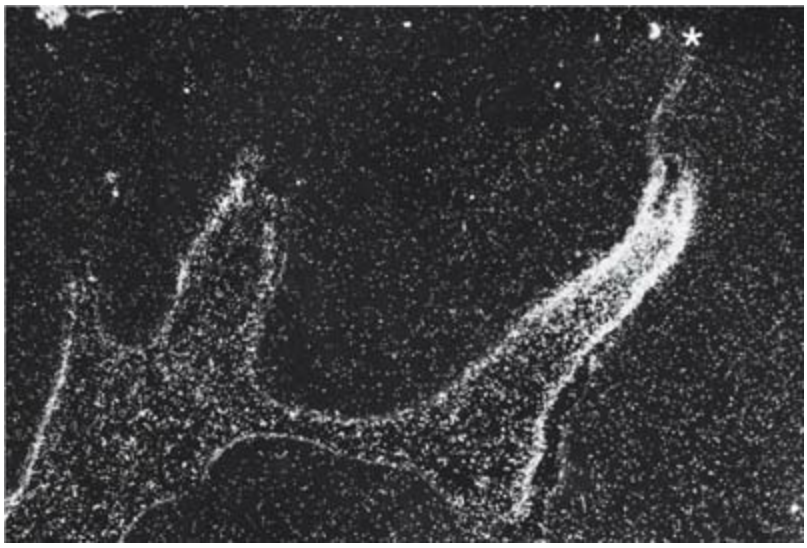

FIG. 13.30 NGF-mRNA is upregulated in the mesial pulp horn 6 hours after cavity preparation.

Electron micrograph shows two pulp horns in tooth where one pulp horn is large and extended toward the crown with a U-shaped mark at the top.

Source: (From Byers MR, Wheeler EF, and Bothwell M: Altered expression of NGF and p75 NGF-Receptor mRNA by fibroblasts of injured teeth precedes sensory nerve sprouting, *Growth Factors* 6:41–45, 1992.)

It has been suggested that neuroimmune interactions take place in the dental pulp, because a coordinated increase of pulpal nerves and immune cells has been demonstrated.[175,325] In addition, recruitment of immunocompetent cells has been demonstrated in the dental pulp after electrical tooth stimulation.[67,108] Similar responses have been seen in periapical bone in rats with radicular pulpitis.[198] Neurogenic inflammation is generally thought to enhance healing, because denervated teeth show poorer healing following pulp exposures than innervated teeth.[43,51]

Another consideration in the neural response to inflammation is the possibility of a change in the distribution and activity of voltage-gated

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sodium channels. In particular, mice lacking the gene for $Na_v1.7$ show reduced painlike behaviors when treated with a variety of proinflammatory agents.[275] Also implicated in the altered firing characteristics of nociceptors innervating inflamed tissues are the sodium channels that are resistant to TTX, the biotoxin found in the tetraodon pufferfish. The two main TTX-R sodium channels are $Na_v1.8$ and $Na_v1.9$, and both have been shown to be increased twofold to fourfold in inflamed dental pulp collected from patients with a diagnosis of irreversible pulpitis.[403,409] When exposed to PGE_2, neurons isolated from dorsal root ganglia cells have been shown to increase TTX-resistant sodium channel currents within minutes,[122] indicating an increased activation of existing channels, rather than de novo protein synthesis. These sodium channels are relatively resistant to lidocaine,[319] and this may explain the difficulty in achieving profound anesthesia in inflamed tissues (see also Chapter 6).[142]

Tissue injury and deafferentation

When a peripheral nerve is cut or crushed, an interruption of the afferent input to the CNS occurs, which is called *deafferentation.* It would be logical to assume that the result of deafferentation would be anesthesia of the formerly innervated area, but occasionally other symptoms may occur, which may surprisingly include pain. Following nerve injury, a dramatic shift in transcription of neuropeptides, receptors, and sodium channels has been documented. The bidirectional contact between the nerve cell and the peripheral target tissue is lost, and the neurons change into a state of either regeneration or neuronal cell death. The impact on neurons in the trigeminal ganglion is dependent on the injury site. A peripheral injury has less effect than a more centrally located one. However, even a small pulp exposure induces neuronal changes, both in the trigeminal ganglion and at the second-order neuronal level in the brain stem.[44,398] Because each single-rooted tooth contains about 2000 nerve fibers,[171,172] extirpation of the pulp is shown to cause both neurochemical and degenerative changes of their cell bodies in the trigeminal ganglion.[131,188,340] The central projection of these nerves to the spinal nucleus of the trigeminal nerve is also affected,[372] and there is evidence for transsynaptic changes[336,340] that are reflected in the sensory

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cortex. Even larger responses would be expected from tooth extraction, where both periodontal ligament and pulpal innervation are destroyed.

When an axon is severed peripherally, a complete degeneration of the cell bodies may not always occur.[399] Attempted regeneration by axonal sprouting may result in altered expression of various receptors, resulting in sensitivity to NE (via increased adrenergic receptor activity)[284] or acetylcholine (ACh) (via increased cholinergic receptor activity),[73] sensitizing sensory neurons to autonomic activity. In addition, dorsal horn neurons, deprived of their normal sensory input, may begin to respond to other nearby afferents. Thus normal inhibitory influences are reduced, and a widening of the sensory receptive field is produced, which can produce central sensitization (see Central Sensitization later in this chapter). *Phantom tooth pain* is another term often used synonymously with pain following deafferentation. Different reports suggest an incidence of persistent pain following pulpectomy to be in the range of 3% to 6%.[243,389]

Following tissue injury or tissue inflammation, extensive changes occur in the gene expression of sensory ganglion neurons and, by way of transsynaptic mechanisms, in their central projections (Fig. 13.31).[46,107,188,340] One example is the upregulation of inducible gene transcription factors such as c-*fos*[44] and different subsets of sodium channels.[119] This is thought to result in alterations in threshold properties and the size of receptor fields. C-*fos* is not normally expressed in neurons in the brain stem, but chronic pulpitis causes prolonged increases in c-*fos* expression is some brain stem neurons (see Fig. 13.31).[44]

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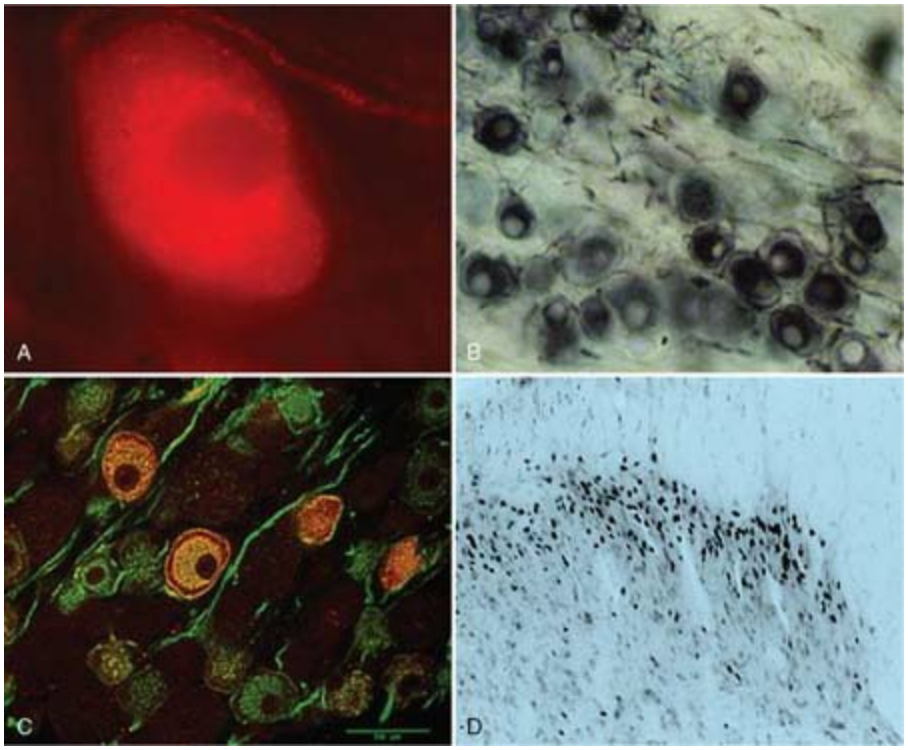

FIG. 13.31 Plastic changes in the trigeminal system. After injury, trigeminal neurons upregulate production of autonomic neuropeptides like neuropeptide Y (NPY) (**A**) and growth-associated protein 43 (GAP 43) connected to neurite outgrowth and regeneration (**B**). **C,** Confocal image showing that NPY is upregulated in injured neurons expressing GAP 43. **D,** The interstitial cells of the trigeminal tract of medullary dorsal horn express c-*fos* during chronic tooth pulp inflammation in rat.

A) Magnified view of a cell with a light circular spot inside on a dark background.

B) Tooth pulp shows circular and oval dark cells. Some of them have white round cells inside.

C) The inflamed neurons with NPY.

D) Microscopic view shows neuronal changes in pulp horn after exposure.

Source: (**B** and **C,** Adapted from Khullar SM, Fristad I, Brodin P, et al: Upregulation of growth associated protein 43 expression and neuronal co-expression with neuropeptide Y following inferior alveolar nerve axotomy in the rat, *J Peripher Nerv Sys* 3:79, 1998. **D,** Adapted from Byers MR, Chudler EH, Ladarola MJ: Chronic tooth pulp inflammation causes transient and persistent expression of Fos in dynorphin-rich regions of rat brainstem, *Brain Res* 861:191, 2000.)

If such changes also occur in humans, they may help to explain why

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certain patients may complain of vague, poorly described pain for months following endodontic treatment. If their pulpitis caused sprouting of periapical nerves,[50,51,54,198] then these nerves may have taken part in transport of peripheral signaling molecules to the cell body by way of retrograde axoplasmic flow.[43,54] This could induce changes in the expression of many genes, resulting in central sensitization[6,42] that may require many months to correct.[75] Reports of nerve sprouting in human inflamed pulps have been confirmed in different studies.[316,318,325] Such reactions might contribute to increases in dentin sensitivity as well as expansion of receptive fields.[273,274,278] Sprouting of sympathetic fibers has also been reported,[149] but the timing appears to be different. The functional implications and how these relate to pain mechanisms are unknown, but it has been suggested that these reactions are involved in healing and nociception following pulpal inflammation.[117,149,150]

Processing: The second step in pain perception

The medullary dorsal horn

After activation of peripheral nociceptors, nerve impulses in the form of action potentials convey information about the intensity (encoded by firing frequency), quality (encoded by type of neuron activated), and temporal features (encoded by onset, duration, and offset of depolarization) of the peripheral stimuli to the CNS. In the trigeminal pain system, these action potentials arrive at the trigeminal spinal tract nuclear complex located in the medulla.[143,218,329] Three distinct subnuclei can be found in this complex. Named for their anatomic position, they are the *subnuclei oralis*, *interpolaris*, and *caudalis* (see Fig. 13.23). Although the more rostral subnuclei (oralis and interpolaris) receive some nociceptive input from oral tissues,[71] most such input is received at the level of the subnucleus caudalis.[238,335] Because of its organizational similarity to the dorsal horn of the spinal cord (which receives nociceptive input from the somatosensory system), the subnucleus caudalis has been termed the *medullary dorsal horn*.

Components of the medullary dorsal horn

The medullary dorsal horn relays information to higher centers in the brain

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and serves as the site of much potential processing of the signals from primary afferent sensory nerve fibers. Output from this region can be increased (hyperalgesia), decreased (analgesia), or misinterpreted (referred pain). Understanding the functional components involved in such processing not only helps explain some of these clinical phenomena but also allows evaluation of potential therapeutic modalities currently under investigation. Functional components include the central terminals of primary nociceptors (Aδ and C fiber afferents), the second-order projecting neurons, interneurons, the terminals of descending neurons, and glial[246] cells (for review, see Hargreaves[139]).

Primary afferent fibers (whose cell bodies are located in the trigeminal ganglion) transmit signals to projection neurons via the release of transmitters such as the excitatory amino acid, glutamate, and the neuropeptide, SP. Receptors for these neurotransmitters are found on postsynaptic membranes and include the N-methyl-d-aspartate receptor (NMDA) and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPA) classes of glutamate receptors and the neurokinin 1 (NK1) class of SP receptors. Antagonists to these receptors have been shown to reduce hyperalgesia in animal studies.[60] In a human clinical trial using an oral surgery model, the AMPA/kainate antagonist, LY293558, was shown to be antihyperalgesic.[119] NK1 antagonists have shown promising results in animal studies, but in general, they have displayed limited analgesic efficacy in humans.[158]

The cell bodies of the second-order (projection) neurons in the trigeminal pain system are found in the medullary dorsal horn; their processes cross the midline and project rostrally to the thalamus via the trigeminothalamic tract (Fig. 13.32). From the thalamus, third-order neurons relay information to the cerebral cortex via a thalamocortical tract. Once signals have reached the cortex, the input may be perceived as pain. Evidence exists that referred pain is caused by convergence of afferent input from different areas onto the same projection neurons.

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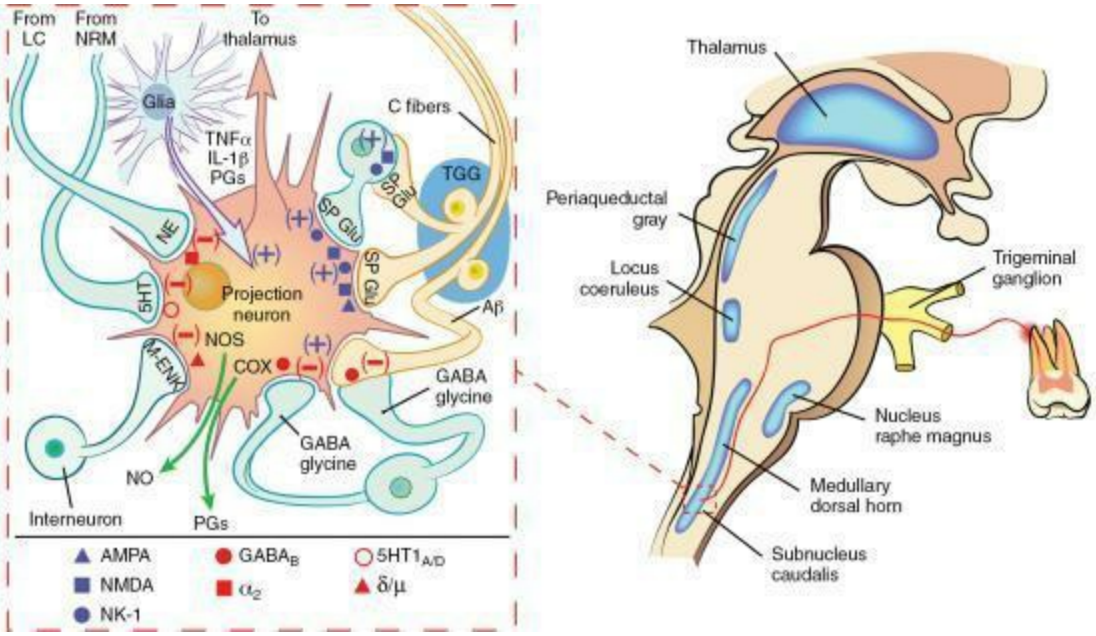

FIG. 13.32 Schematic diagram of the perception and modulation of orofacial pain. Activation of primary afferent fibers (in this example from an inflamed maxillary molar) leads to the entry of a nociceptive signal that is conveyed across a synapse in the subnucleus caudalis of the trigeminal spinal nucleus. The second-order neuron projects to the thalamus; the information is then relayed to the cortex. A great deal of processing of nociceptive input can occur at the level of the medullary dorsal horn (MDH). The *inset* depicts a typical wide dynamic range (WDR) projection neuron and its relationship with other components of the MDH. Primary afferent fibers release the excitatory amino acid, glutamate—which binds and activates either AMPA or NMDA receptors —and substance P—which activates NK-1 receptors on the WDR neuron or excitatory interneurons. Descending fibers from the locus coeruleus *(LC)* and nucleus raphe magnus *(NRM)* secrete serotonin *(5HT)* and norepinephrine *(NE)*, respectively, which inhibit transmission. Release of γ amino butyric acid *(GABA)*, the amino acid, glycine, and endogenous opioid peptides such as met-enkephalin *(M-ENK)* also inhibit transmission of nociceptive information. Projection neurons may have autocrine or paracrine effects by the synthesis and release of prostaglandins *(PGs)* and nitric oxide *(NO)* via the action of cyclooxygenase *(COX)* and nitric oxide synthase *(NOS)*, respectively. Glial cells can modulate nociceptive processing by the release of cytokines such as tumor necrosis factor α *(TNF-α)* and interleukin 1 β *(IL-1β)*. The + sign indicates an excitatory action, whereas the—sign denotes an inhibitory action. *AMPA,* α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; *NK,* neurokinin; *NMDA, N*-methyl-d-aspartate; *SP,* substance P; *TGG,* trigeminal ganglion.

Regions associated with perception and modulation of orofacial pain are as follows: Trigeminal ganglion, nerve from inflamed tooth leads to medullary

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dorsal horn, subnucleus caudalis, locus coeruleus, periaqueductal gray, and thalamus. An inset from subnucleus caudalis shows projection of neuron and associated components.

Approximately 50% of subnucleus caudalis neurons are estimated to receive convergence of sensory input from cutaneous and deep structures.[335] In one study of a cat, a single nucleus caudalis neuron received input from sensory neurons innervating the cornea, the skin overlying the maxilla, a maxillary premolar tooth, and a mandibular canine and premolar tooth on one side.[339] Subnuclei oralis and interpolaris also receive converging input from orofacial and muscle afferents.[338] This would explain the clinical observation of patients who perceive pain in a particular tooth that actually originates from either a different tooth or structure (see also Chapter 4). In such cases, anesthetizing the tooth suspected by the patient would afford no relief. However, if an anesthetic is delivered selectively to the suspected primary source of pain, the patient's discomfort should greatly diminish.[285] Likewise, if the source of a perceived toothache were located in a muscle of mastication, palpation of that muscle should aggravate the pain.[417]

In the medullary dorsal horn, local circuit interneurons have the potential to affect the transmission of nociceptive input from primary afferents to projection neurons. Depending on the transmitter released, these neurons have the ability to enhance or diminish the signal. Typically, excitatory interneurons release glutamate or SP, whereas inhibitory interneurons release the amino acid, glycine, or γ amino butyric acid (GABA).[218,337]

The terminals of neurons that descend from brain structures such as the locus coeruleus and nucleus raphe magnus tend to inhibit nociceptive transmission at the level of the medullary dorsal horn.[14] These terminals release a variety of neuroeffective agents, including the endogenous opioid peptides (EOPs). The EOPs, similar in three-dimensional structure to many of the exogenous opiates from which their name derives, are released in response to nociceptive input and act to suppress the pain system. The EOPs likely are partly responsible for the placebo effect seen in pain control studies, because this effect can be reversed by administration of the opioid antagonist, naloxone.[141,216]

The final component of the medullary dorsal horn complex to be considered is the glial cell population. Historically considered to be solely

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supportive in function, they are now recognized to play an important role in the pain processing system.[308,404] Following nociceptive input from primary afferents, glia release cytokines such as TNF-α and IL-1, as well as certain PGs that may facilitate the activity of projection neurons. Glial modulating agents have been shown to be effective in experimental models of neuropathic pain,[355] and NSAIDs potentially could exert part of their analgesic mechanism by acting at this level.

Central sensitization

Central sensitization can be defined as an increased responsiveness of central nociceptive neurons to peripheral stimulation that occurs in addition to *peripheral sensitization* of the primary afferent nociceptors. Central sensitization is thought to be a major cause of hyperalgesia and allodynia.[208] Clinical trials implicate central sensitization in patients reporting pain due to irreversible pulpitis. In one survey of nearly 1000 patients, 57% of patients with irreversible pulpitis reported mechanical allodynia (pain due to percussion).[294] This appears to be due at least in part to central sensitization, as both the ipsilateral (pulpitis) tooth and a contralateral (normal) tooth demonstrated mechanical allodynia to a force transducer.[187] Thus central sensitization contributes to a spread of endodontic pain, and the clinical application of bite force transducers may provide a novel method for diagnosing pain mechanisms.[186,187]

Different studies shed light on the molecular mechanisms involved in central sensitization (for review, see Cousins and Power[64] and Hargreaves[139]), but the process is generally initiated by a barrage of nociceptive impulses from peripheral C fibers. The level and duration of pain prior to endodontic intervention have been cited in several studies as predictors of postoperative endodontic pain,[369,400] and this may be due to such a prolonged and intense input from C nociceptors. Any reduction of such a barrage should limit the occurrence of central sensitization and the development of pain of longer duration after tissue injury (including surgical and nonsurgical endodontic procedures). The use of long-acting local anesthetics following tonsillectomies and third molar extractions has been shown to provide pain relief far beyond the duration of the peripheral tissue anesthesia.[127,169]

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A reduction in the chemical mediators of inflammation at the level of the medullary dorsal horn also should reduce sensitization of second-order neurons. Decreasing the synthesis of proinflammatory PGs, cytokines, nitric oxide (NO), or the use of drugs that block the receptors of such agents probably will become accepted pharmacotherapy in the future. For example, application of an inflammatory agent to the tooth pulp of rat maxillary molars results in an increased receptive field of Aβ touch receptors on the face. This can be blocked by pretreatment with a glutamate NMDA receptor antagonist, indicating that such centrally acting drugs may offer highly efficacious means of treating odontogenic pain.[60] A similar investigation implicated NO synthesis at the level of the subnucleus caudalis in the development of tactile hypersensitivity following dental injury.[423] Reduction in NO synthase levels may also provide protection from central sensitization.[239,253]

Perception: Thalamus to cortex

The final anatomic step in the trigeminal pain pathway relies on neurons that leave the thalamus and extend to the cerebral cortex (see Fig. 13.23). The patient actually perceives a stimulus as painful at the cortical level. It is interesting to note (but likely of no surprise to the experienced clinician) that a disproportionately large portion of the sensory cortex in humans is devoted to input from orofacial regions.[300]

It is becoming increasingly obvious that higher-order (i.e., cortical) perceptual processes have a profound effect on the ultimate state of pain the patient experiences (for a review, see Yaksh[418]). Memories of previous pain experiences provide a framework by which similar new experiences are judged and serve to shape the patient's response to a given stimulus. In the field of dentistry, the anxiety level of the patient at the time of treatment has been shown to affect not only the patient's response to pain experienced during treatment,[79,414] but also the tendency of the patient to recall the experience as painful or unpleasant even 18 months after treatment.[114] The clinician should do everything possible to control a patient's anxiety level prior to endodontic treatment. One simple pretreatment method is to provide patients with positive written information regarding the control of pain during their endodontic treatment. In a placebo-controlled clinical trial, 437 endodontic patients were given one of five informative paragraphs to read

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prior to treatment. One of the paragraphs contained positive information about pain during treatment. Patients completed questionnaires following treatment that evaluated their dental anxiety and dental fear. Subjects given positive information were shown to be less fearful of pain during endodontic therapy.[386] Along with a positive and caring attitude, pharmacologic intervention may help reduce anxiety. Nitrous oxide has been shown to be effective in a dental setting,[76] but it may interfere with radiography procedures during endodontic therapy. In a placebo-controlled clinical trial in patients undergoing the extraction of impacted third molars, 0.25 mg of oral triazolam (a benzodiazepine) provided comparable anxiolysis to intravenous diazepam titrated to a typical clinical endpoint.[178] Of course, the patient so medicated must be provided transportation to and from the dental office, and the potential drug-drug interactions with other centrally acting agents such as opioids, barbiturates, and alcohol must be considered. One interaction that should be considered is the capacity of grapefruit juice to prolong the half-life of triazolam.[221] It has been shown that furanocoumarins in grapefruit juice inhibit cytochrome P450 3A4,[296] which is the enzyme responsible for the metabolism of triazolam in the liver. Patients should be told not to take oral triazolam with grapefruit juice.

Vascular supply

Blood from the dental artery enters the tooth by way of arterioles having diameters of 100 μm or less. These vessels pass through the apical foramen or foramina with nerve bundles. Smaller vessels may enter the pulp by way of lateral or accessory canals. They are richly innervated by autonomic and sensory nerves, and the regulation of blood flow seems to be dominated by neuronal control (Fig. 13.33).[2,20,191,289,365]

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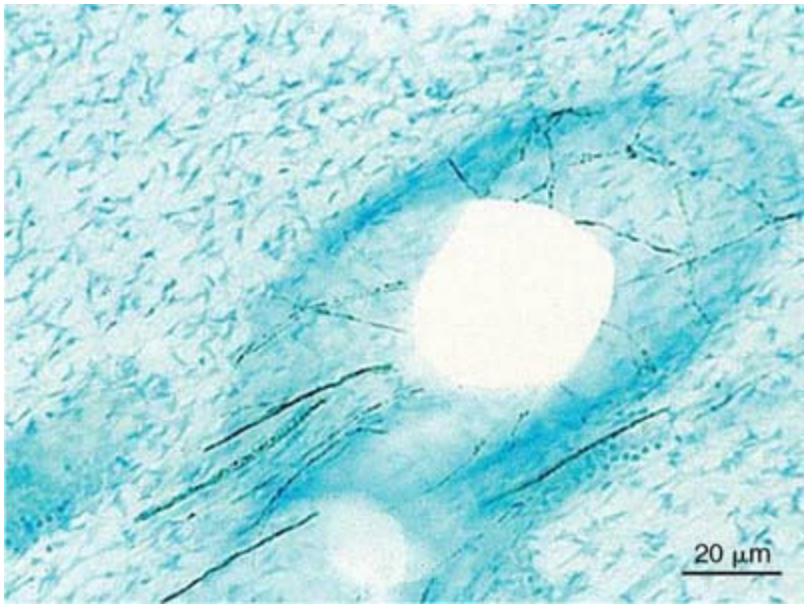

FIG. 13.33 Substance P–positive nerve fibers in the wall of pulpal blood vessels.

Stained micrograph depicted on the scale of 20 micrometers shows an oval cell with a round light spot at the center surrounded by dark thread-like structures.

Source: (Courtesy Dr. K.J. Heyeraas.)

The arterioles course up through the central portion of the radicular pulp and give off branches that spread laterally toward the odontoblast layer, beneath which they ramify to form a capillary plexus (Fig. 13.34).[200] As the arterioles pass into the coronal pulp, they fan out toward the dentin, diminish in size, and give rise to a capillary network in the subodontoblastic region (Fig. 13.35).[358] This network provides the odontoblasts with a rich source of metabolites.

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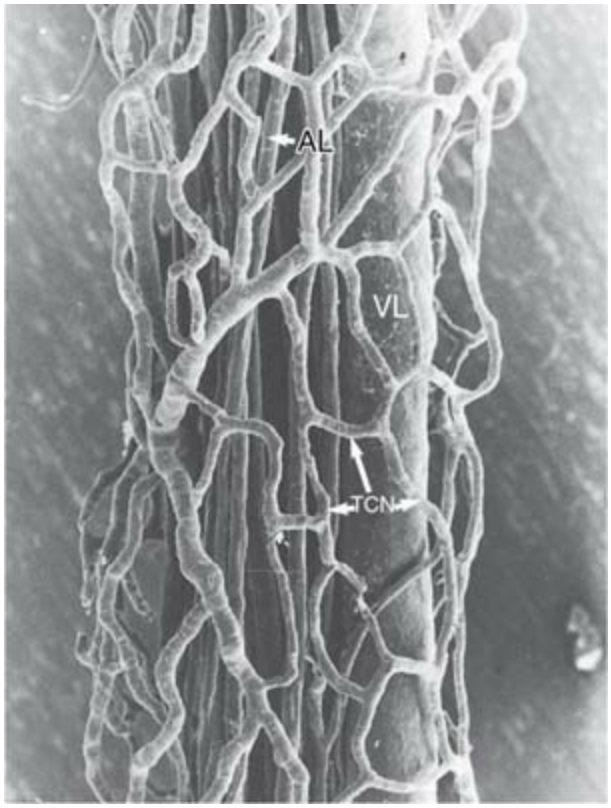

FIG. 13.34 High-power scanning electron micrograph of vascular network in the radicular pulp of a dog molar showing the configuration of the subodontoblastic terminal capillary network *(TCN)*. Venules *(VL)* and arterioles *(AL)* are indicated.

SEM shows vertical column of branched arterioles accompanied by large venules in-between forming a network of capillaries in subodontoblastic layer of tooth.

Source: (Courtesy Dr. Y. Kishi, Kanagawa Dental College, Kanagawa, Japan.)

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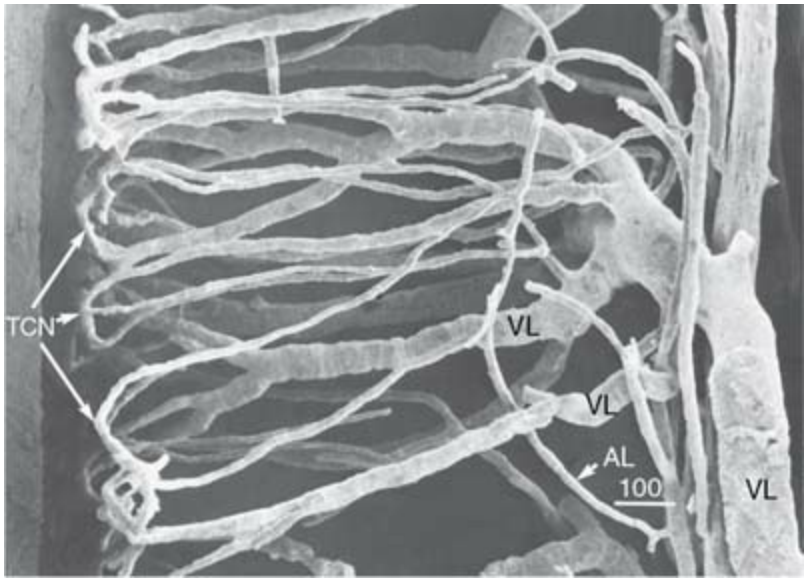

FIG. 13.35 Subodontoblastic terminal capillary network *(TCN)*, arterioles *(AL)*, and venules *(VL)* of young canine pulp. Dentin would be to the far left and the central pulp to the right. Scale bar: 100 µm.

Scanning electron micrograph depicted on the scale of 100 micrometers shows thin arterioles accompanied by wide venules and terminal capillary network.

Source: (From Takahashi K, Kishi Y, Kim S: A scanning electron microscopic study of the blood vessels of dog pulp using corrosion resin casts, *J Endod* 8:131, 1982.)

Capillary blood flow in the coronal portion of the pulp is nearly twice that in the root portion.[195] Moreover, blood flow in the region of the pulp horns is greater than in all other areas of the pulp.[254] In young teeth, capillaries commonly extend into the odontoblast layer, thus ensuring an adequate supply of nutrients for the metabolically active odontoblasts (Fig. 13.36). In the subodontoblastic capillaries, fenestrations are observed in the vessel wall.[312] These fenestrations are thought to promote rapid transport of fluid and metabolites from the capillaries to the adjacent odontoblasts. The average capillary density is about 1400/mm^2, which is greater than in most other tissues of the body.[393]

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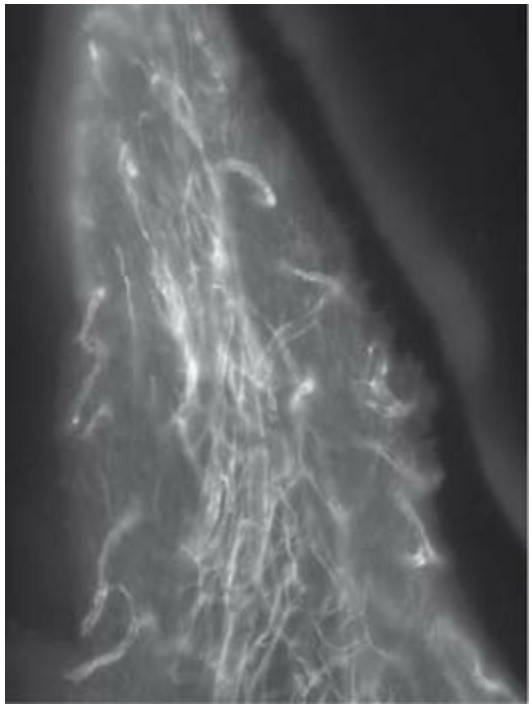

FIG. 13.36 Blood vessels in the pulp horn fan out into the odontoblast layer.

Electron micrograph shows greater density of blood vessels in the pulp comparatively to the odontoblastic layer.

Source: (Courtesy Dr. S.R. Haug.)

Blood passes from the capillary plexus, first into postcapillary venules (see Figs. 13.35 and 13.37) and then into larger venules.[200] Venules in the pulp have unusually thin walls, and the muscular layer is discontinuous,[70] which may facilitate the movement of fluid in or out of the vessel. The collecting venules become progressively larger as they course to the central region of the pulp. The largest venules have a diameter that may reach a maximum of 200 μm, which is considerably larger than the arterioles of the pulp.

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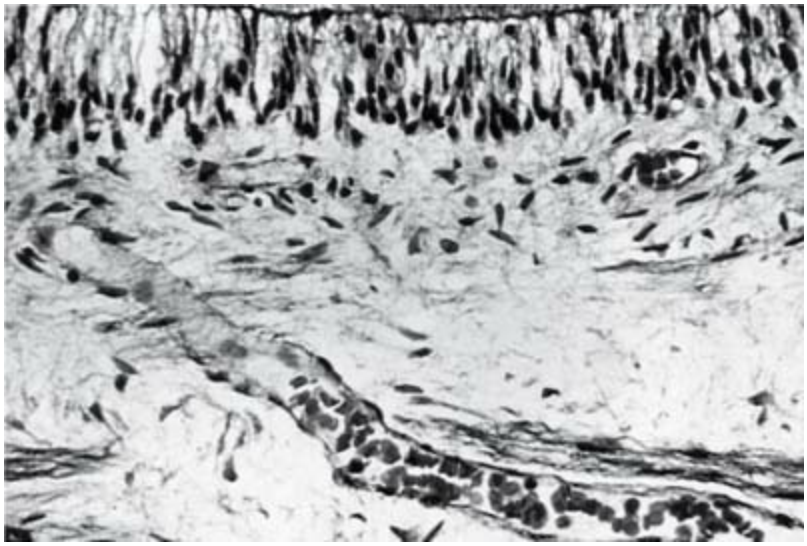

FIG. 13.37 Postcapillary venule draining blood from subodontoblastic capillary plexus.

Electron micrograph shows narrow channel of postcapillary venule with blood cells in pulp below odontoblast layer.

The resting PBF is relatively high averaging 0.15 to 0.60 mL/min/g tissue.[250,367] Blood volume represents about 3% of pulpal wet weight,[31] approximately the same as in mammary tumor tissue.[410] As would be anticipated, PBF is greater in the peripheral layer of the pulp (i.e., the subodontoblastic capillary plexus)[200] where the oxygen consumption has been shown to be higher than in the central pulp.[26]

Changes in PBF can be measured through dentin using laser Doppler flowmeters. Sensitivity to movement requires that they are stabilized in an occlusal stent or a modified rubber dam clamp.[95,327] Because up to 80% of the Doppler signal originates from periodontal tissue, it is helpful to cover periodontal tissues with a black rubber dam.[146] Laser Doppler flowmetry can be used to detect revascularization of traumatized teeth.[80,90] Although measurement of PBF would be an ideal tool for determining pulp vitality, the use of laser Doppler and other techniques is limited due to sensitivity, specificity, reproducibility, and costs.

Regulation of pulpal blood flow

Under normal physiologic conditions, pulpal vascular tone is controlled by neuronal, paracrine, and endocrine mechanisms that keep the blood vessels in

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a state of partial constriction. The PBF is also influenced by vascular tone in neighboring tissues. Vasodilatation in these tissues has been shown to cause a drop in PBF due to reduction in local arterial pressure of the teeth and thereby reduced pulpal perfusion pressure.[366] The "stealing" of dental perfusion pressure makes the dental pulp vulnerable in clinical situations with inflammatory processes in the adjacent tissues, as in gingivitis and periodontitis. There is no autoregulation of PBF as it follows passively changes in systemic blood pressure.[189]

Neuronal regulation of blood flow is extensive in the pulp. There is little or no vasoconstrictor tone of sympathetic origin in the dental pulp during resting conditions,[167,365] but a neuronal vasodilator tone caused by release of sensory neuropeptides has been demonstrated (Fig. 13.38).[21,20]

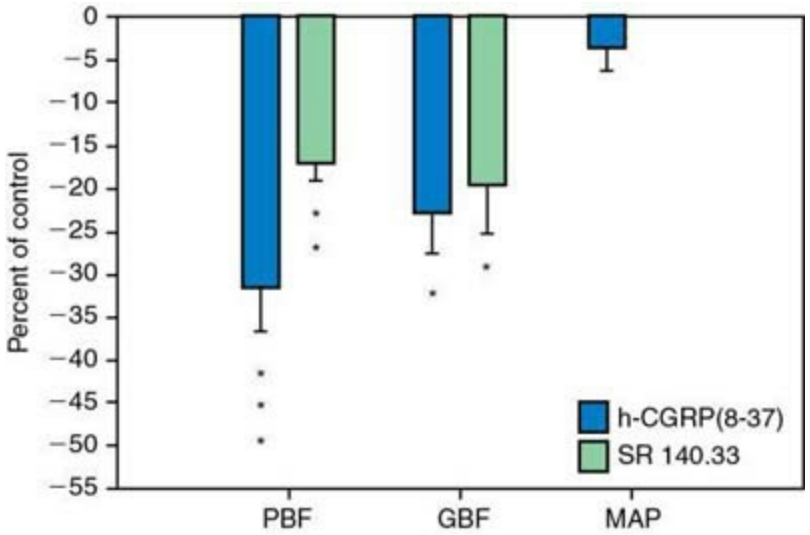

FIG. 13.38 Effect of antagonist infusion of h-*CGRP(8-37)* (calcitonin gene–related peptide inhibitor) and *SR* 140.33 (substance P inhibitor) on basal pulpal blood flow *(PBF)* and gingival blood flow *(GBF)*. *MAP,* Mean arterial pressure. ***, p <0.001, **, p <0.005, *, p <0.05, as compared with control recordings before antagonist infusion.

Bar graph plots percent of control against PBF, GBF, and MAP on the horizontal axis. The vertical axis ranges from 0 to negative 55 from bottom to top. The data are as follows. PBF: h-CGRP(8-37): negative 32, SR 140.33: negative 18, GBF: h-CGRP(8-37): negative 24, SR 140.33: negative 20, and h-CGRP(8-37): negative 4.

Source: (From Berggreen E, Heyeraas KJ: Effect of the sensory neuropeptide antagonists h-CGRP[8-37] and SR 140.33 on pulpal and

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gingival blood flow in ferrets, *Arch Oral Biol* 45:537, 2000.)

There are α-adrenergic receptors in the pulp,[164] and stimulation of the cervical sympathetic trunk causes vasoconstriction and fall in PBF that can be partially reversed by α-receptor blockade.[191,365] NPY, colocalized with NE in pulpal sympathetic nerve fibers, also contributes to vasoconstriction in the pulp.[82,197]

Increase in PBF is observed after electrical tooth stimulation and is caused by the release of sensory neuropeptides followed by vasodilatation.[20,155,183] CGRP released from sensory nerve fibers is mainly responsible for the observed vasodilatation.[20,21]

Glutamate, present in CGRP negative sensory afferent nerve fibers in the pulp, also has a vasodilatory effect when applied in the pulp during experimental conditions.[426]

There is evidence for sympathetic modulation of sensory neuropeptide release in the dental pulp[140]; presynaptic adrenoceptors are found on the sensory nerve terminals and attenuate the release of vasodilators from the sensory nerves.[35,183]

Muscarinic receptors have been identified in the pulp,[33] and the parasympathetic neurotransmitter ACh causes vasodilatation and increases blood flow in the tissue.[424] The vasodilation evoked by ACh has been demonstrated to be partly dependent on NO production.

VIP, which coexists with ACh in postganglionic neurons, is found in the dental pulp[382,397] and has been demonstrated to cause vasodilatation and increase in PBF in cats.[289]

On the other hand, Sasano and coworkers[329] failed to demonstrate parasympathetic nerve-evoked vasodilatation in the cat dental pulp, leaving pulpal vascular responses to parasympathetic neurotransmitters with some uncertainty.

Local control of blood flow

The microvascular bed in the dental pulp has the ability to regulate hemodynamics in response to local tissue demands. Endothelin-1 is located in the endothelium of pulpal vasculature,[58] and close intraarterial infusions of endothelin-1 reduce PBF.[22,118,424] However, endothelin-1 does not seem to

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influence blood vessel vascular tone under basal, resting conditions.[22]

The endothelium in pulpal blood vessels modulates vascular tone by release of vasodilators such as prostacyclin and NO. A basal synthesis of NO provides a vasodilator tone on pulpal vessels.[20,226] The shear forces that blood flow exerts on endothelial cells seem to regulate the release of NO.[77]

Adenosine is released from ischemic and hypoxic tissue and is probably important in the metabolic regulation of blood flow in periods of low pulpal oxygen tension. When applied from the extraluminal side of the vessel wall, adenosine mediates vasodilatation in pulpal vessels.[424]

Humoral control of blood flow

Evidence for humoral control of PBF exists and takes place when vasoactive substances transported by the bloodstream reach the receptors in the pulp tissue. Angiotensin II is produced by activation of the renin/angiotensin system and exerts a vasoconstrictive basal tone on pulpal blood vessels.[22] The angiotensin II receptors, AT1 and AT2, have been identified in rat pulp.[348]

Similarly to the effect of NE released from sympathetic nerve fibers in the pulp, epinephrine released from the adrenal medulla will cause vasoconstriction due to activation of α-adrenergic receptors in the pulp. Another catecholamine, dihydroxyphenylalanine (DOPA), also induces vasoconstriction in pulpal arterioles when applied intraarterially.[424]

Fluid drainage

Interstitial fluid, which accumulates in the tissue during normal conditions through net filtration out of the blood vessels or during inflammation where the net filtration is increased, must be removed in order to maintain normal fluid balance. In most tissues in the body, the lymphatic vessels drain excess fluid from the peripheral tissue and return it into the blood vessel system. In addition, the lymphatic system is important because it transports captured antigen and presents it in the lymph nodes. The existence of lymphatics in the pulp has been a matter of debate because it is difficult to distinguish between blood and lymphatic vessels by ordinary microscopic techniques without specific lymphatic markers.

Several specific lymphatic markers have now been applied, and

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contradictory conclusions have been drawn. One of the markers tested is vascular endothelial growth factor receptor (VEGFR-3), known to be expressed by lymphatic endothelial cells in adult tissue.[114,242] The receptor expression was reported in human and mouse pulp tissue, but to identify lymphatic vessels use of more than one lymphatic marker is recommended. VEGFR-3 has recently been demonstrated in blood vessels and on various immune cells and is not specific to lymphatic vessels.[30,390] However, other studies have failed to demonstrate lymphatic markers in the pulp [116,247] and one study showed lymphatic vessel endothelium receptor-1 (LYVE-1) staining in immune cells in human pulp, but not in vascular structures (Fig. 13.39). The same observation was done in a characterization of the lymphatic system of the mouse head.[228] The authors showed that lymphatic vessels were found in the mandibular canal. We have demonstrated lymphatic vessels in the mandibular canal in rats starting below the molars (Fig. 13.40). Taken together, we can conclude that the pulp is not supplied with draining lymphatic vessels.

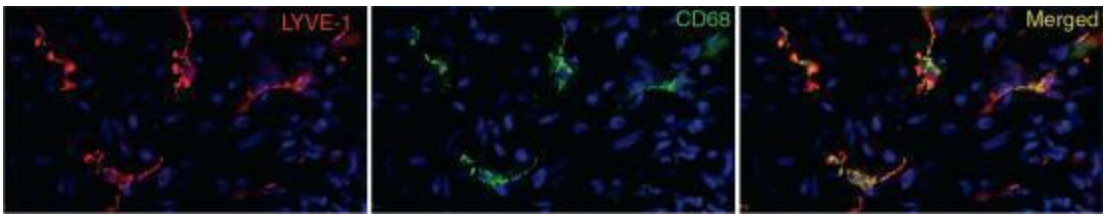

FIG. 13.39 Immune cells in normal human pulp are immunopositive to *LYVE-1*, known as a lymphatic vessel marker. The *CD68*$^{+}$*/LYVE-1*$^{+}$ cells derive from the monocytic lineage of cells. Immunostaining demonstrated the lack of *LYVE-1*$^{+}$ lymphatic vessels in the pulp.

Set of three confocal images show the presence of LYVE-1, CD68, and merged cells, respectively, in the tooth pulp.

Source: (Courtesy Dr. A Virtej.)

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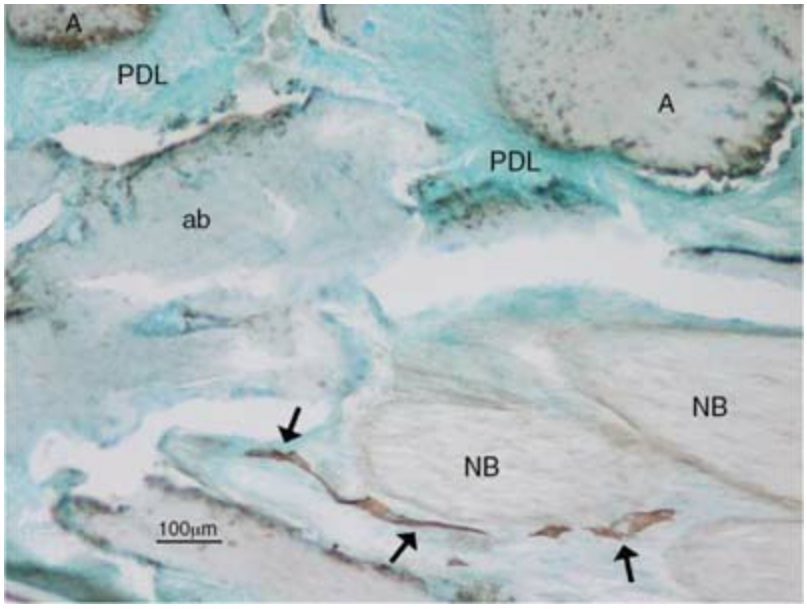

FIG. 13.40 A lymphatic vessel *(arrows)* in the mandibular canal in a rat jaw below the apex *(A)* of the molars. *ab,* Alveolar bone; *NB,* nerve bundle; *PDL,* periodontal ligament.

Micrograph shows a fragmented lymphatic vessel below the root tip of molar with labels as follows: Nerve bundle, alveolar bone, periodontal ligament, and apex. The scale bar reads, 100 micrometers.

Source: (Courtesy Dr. Anca Virtej.)

Transcapillary fluid exchange

In all tissues in the body, the fluid transport between the blood vessels and the interstitial space is regulated by differences in colloid osmotic and hydrostatic pressures in the plasma and the interstitium, and by properties in the capillary membrane (Fig. 13.41). From the interstitium excess fluid is transported back to the blood circulation through the lymphatic system. The pulp seems to be an exception along with, for example, the brain and bone marrow, as lymphatic vessels are not detected in the tissue (see the previous section). During normal conditions, a steady state is achieved as the fluid filtered into the interstitial space equals the amount of fluid transported out of the same compartment. Using radioisotopes, the interstitial fluid volume in the pulp was measured and averaged 0.6 ± 0.03 mL/g wet weight,[31] demonstrating that as much as 60% of the extracellular fluid in dental pulp is located outside the vascular system. Measurements of interstitial fluid

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pressure in the pulp with the micropuncture method have given values that range from 6 to 10 mm Hg,[20,152] but higher values measured with different methods have also been reported.[40,385,392]

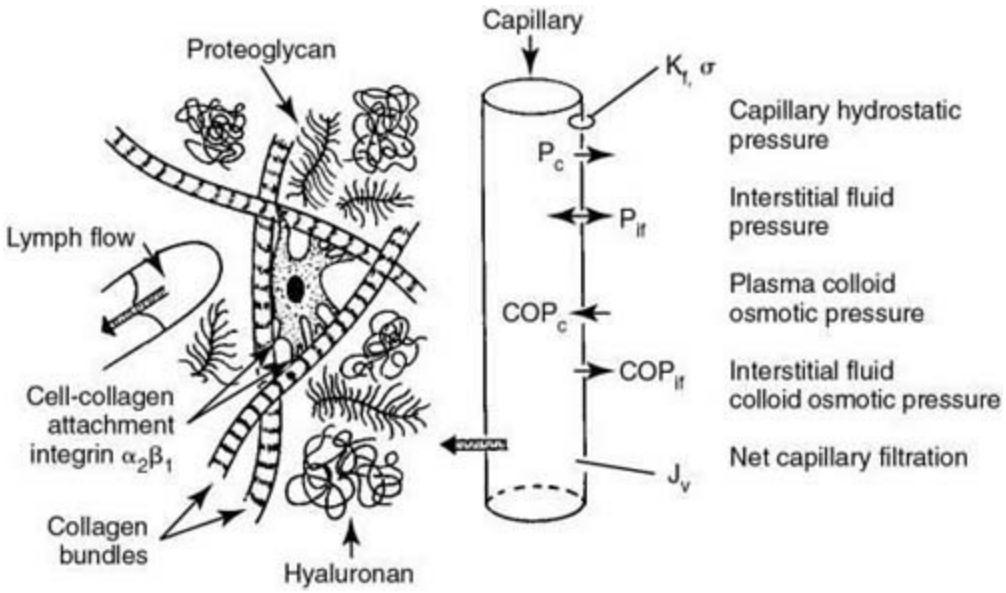

FIG. 13.41 Interstitial structure and pressures that govern transcapillary fluid transport. K_f, Capillary filtration coefficient; σ, capillary reflection coefficient for plasma proteins.

Capillary with labels as follows: K sub f, alpha, capillary hydrostatic pressure (P sub c), interstitial fluid pressure (P sub i f), plasma colloid osmotic pressure (COP sub c), interstitial fluid colloid osmotic pressure (COP sub i f), and net capillary filtration (J sub v). Labels associated with neuron are as follows: Hyaluronan, collagen bundles, cell-collagen attachment integrin alpha sub 2 beta sub 1, lymph flow, and proteoglycan.

Source: (From Wiig H, Rubin K, Reed RK: New and active role of the interstitium in control of interstitial fluid pressure: potential therapeutic consequences, *Acta Anaesthesiol Scand* 47:111, 2003.)

Colloid osmotic pressure (COP) measurements in interstitial fluid isolated from rat incisors have shown a relatively high pulpal COP, reaching 83% of plasma COP.[31] The high value may imply that the normal permeability of pulpal vessels to plasma proteins is relatively high or the drainage of plasma proteins is ineffective.

Because lymphatic vessels are lacking inside the pulp, excess interstitial fluid and proteins must be transported out of the pulp by other transport

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routes in order to achieve a steady-state situation. Two possibilities exists: (1) transport of fluid in the interstitial compartment toward the apical part of the pulp and furthermore out of the apex, and (2) a combination of fluid reabsorption into pulpal blood vessels in addition to transport of protein-rich fluid toward the apex.

Circulation in the inflamed pulp

Inflammation in the pulp takes place in a low-compliance environment composed of rigid dentinal walls. *Compliance* is defined as the relationship between volume (V) and interstitial pressure (P) changes: $C = \Delta V/\Delta P$. Consequently, in the low-compliant pulp, an increase in blood or interstitial volume will lead to a relatively large increase in the hydrostatic pressure in the pulp. The acute vascular reactions to an inflammatory stimulus are vasodilatation and increased vascular permeability, both of which will increase pulp interstitial fluid pressure[153,156,365,384] and may tend to compress blood vessels and counteract a beneficial blood flow increase (Fig. 13.42).

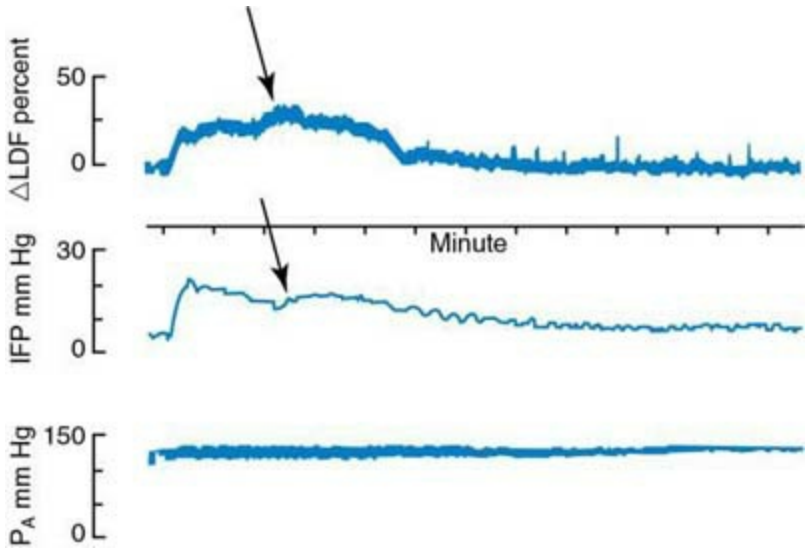

FIG. 13.42 Original simultaneous recordings of percent change in pulpal blood flow *(*ΔLDF%), interstitial fluid pressure *(IFP)*, and systemic blood pressure *(P_A* mm Hg) in a cat during electrical tooth stimulation. Note that when IFP is first decreasing after an initial rise, the pulpal blood flow reaches its maximal level *(arrows),* demonstrating compression of vessels in the first phase.

Three graphs compare the recordings of uppercase delta LDF percent, IFP,

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and P sub A in mm Hg against minutes. In LDF, the LDF reading originates from 0, reaches a height at 30, slops down to 0 and oscillates throughout. IFP reading originates from 0, reaches a height at 27, slopes down to 24 (when LDF increases), oscillates, and terminates at 8. P sub A originates from 110 and remains almost constant throughout. All data are approximate.

Source: (Courtesy Dr. K.J. Heyeraas.)

Classical studies have demonstrated that an increase in intrapulpal tissue pressure promoted absorption of tissue fluid back into the circulation, thereby reducing the pressure.[153,156] This observation can explain why pulpal tissue pressure in inflamed pulps may persist in local regions for long observation periods,[367] contradicting the old concept of a wide, generalized collapse of pulpal venules and cessation of blood flow (pulpal strangulation theory).

The delivery of dental restorative procedures may lead to substantial increases or decreases in PBF, depending on the precise procedure and time point sampled.[191] Vasoactive mediators are locally released upon an inflammatory insult, and in the pulp, PGE_2, BK, SP, and histamine have all been demonstrated to increase PBF after application.[194,290] In contrast, serotonin (5-HT) is released primarily from the platelets, and given intraarterially, it has been shown to reduce PBF.[196,424]

Acute inflammation in the dental pulp induces an immediate rise in blood flow and can reach a magnitude of up to nearly 200% of control flow followed by increased vascular permeability.[154,156]

A common outcome of pulpal inflammation is development of tissue necrosis. One study found circulatory dysfunction developed in the pulp after exposure to lipopolysaccharide (LPS) from gram-negative bacteria.[31]

In addition, the inflammatory cytokines IL-1 and TNF-α are elevated in the inflamed pulp. When the endothelium is exposed to endotoxin, it expresses cytokines, chemokines, and thromboxane A2. The latter has been demonstrated to be produced in the pulp exposed to LPS[287] and induces vasoconstriction. The set of changes in endothelial function have been called *endothelial perturbation* and were first described in endothelial cells exposed to endotoxin or to cytokines such as IL-1, TNF-α, and IL-6.[24,276] The activated endothelium also participates in procoagulant reactions that promote fibrin clot formation.[352] A reduced pulpal perfusion due to

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endothelial perturbation might be the consequence of bacterial infection impairing pulpal defense mechanisms and promoting necrosis. Downregulation of vascular endothelial growth factor (VEGF) expression in stromal cells and reduced microvessel density have been observed in human dental pulps with irreversible pulpitis.[10] VEGF is an essential proangiogenic factor, and the reduced microvessel density might also lead to reduced pulpal perfusion and contribute to development of pulpal necrosis.

Vascular permeability

Increased vascular permeability takes place as a result of acute inflammation, and vascular leakage has been demonstrated in the pulp after release of inflammatory mediators such as PG, histamine, BK, and the sensory neuropeptide, SP.[184,194,241]

LPS and lipoteichoic acid from gram-negative and gram-positive bacteria, respectively, cause upregulation of VEGF in activated pulpal cells.[34,360] VEGF increases vascular permeability,[93,334] and it is likely that it also causes leakage in pulpal vessels. It is a potent agent, because its ability to enhance microvascular permeability is estimated to be 50,000 times higher than that of histamine.[341] Cytokines such as IL-1 and TNF-α are released into the pulp interstitial fluid during inflammation[31] and upregulate VEGF mRNA gene expression in pulpal fibroblasts.[61]

The resulting increased vascular permeability allows increased transport of proteins through the capillary vessel wall and results in increased COP in the tissue. In acute pulpitis induced by LPS, it has been shown that COP in the pulp can reach the level of plasma COP, meaning that a protein transport barrier between plasma and interstitium can be eliminated.[31]

The lack of lymphatic vessels in the pulp may in this situation disfavor the pulps ability to recover and reestablish a normal COP since the transport of proteins is dependent on a pressure difference for fluid transport out of the pulp. Lack of lymphatic vessel for transport of protein-rich fluid may make the pulp more vulnerable to development of necrosis after severe pulpitis.

Clinical aspects

The influence of posture on PBF has been observed in humans.[95] Significantly greater PBF was measured when subjects changed from an

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upright to a supine position. The supine position increases venous return from all tissues below the level of the heart, thereby increasing cardiac output and producing a transient increase in systemic blood pressure. The increase in blood pressure stimulates baroreceptors that reflexively decrease sympathetic vasoconstriction to all vascular beds, thereby increasing peripheral blood flow. Patients with pulpitis often report an inability to sleep at night because they are disturbed by throbbing tooth pain. In addition to the lack of distractions normally present during the day, the following mechanism may be operative in patients with inflamed pulps. When these patients lie down at the end of the day, their PBF probably increases due to the cardiovascular postural responses described earlier. This may increase their already elevated pulpal tissue pressure,[153,156,351,367,384] which is then sufficient to activate sensitized pulpal nociceptors and initiate spontaneous pulpal pain. Thus the "throbbing" sensation of toothache was previously considered as an effect of the pulsation in the pulp that follows heart contractions (systole), but a study investigating the rhythm of toothache and the patient pulse showed lack of synchrony between the two parameters.[256] The authors raised an alternative hypothesis: that the throbbing quality is not a primary sensation but rather an emergent property, or perception, whose "pacemaker" lies within the CNS.

Pulpal repair

The inherent healing potential of the dental pulp is well recognized. As in all other connective tissues, repair of tissue injury commences with débridement by macrophages, followed by proliferation of fibroblasts, capillary buds, and the formation of collagen. Local circulation is of critical importance in wound healing and repair. An adequate supply of blood is essential to transport immune cells into the area of pulpal injury and to dilute and remove deleterious agents from the area. It is also important to provide fibroblasts with nutrients from which to synthesize collagen. Unlike most tissues, the pulp has essentially no collateral circulation, making it theoretically more vulnerable than most other tissues. In the case of severe injury, healing would be impaired in teeth with a limited blood supply. It seems reasonable to assume that the highly cellular pulp of a young tooth, with a wide-open apical foramen and rich blood supply, has a much better healing potential than an

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older tooth with a narrow foramen and a restricted blood supply.

Dentin can be classified as primary, secondary, or tertiary, depending on when it was formed. *Primary dentin* is the regular tubular dentin formed before eruption, including mantle dentin. *Secondary dentin* is the regular circumferential dentin formed after tooth eruption, whose tubules remain continuous with that of primary dentin. *Tertiary dentin* is the irregular dentin that is formed in response to abnormal stimuli, such as excess tooth wear, cavity preparation, restorative materials, and caries.[65,66] In the past, tertiary dentin has been called *irregular dentin, irritation dentin, reparative dentin,* and *replacement dentin*. Much of the confusion was caused by a lack of understanding of how tertiary dentin is formed.

If the original odontoblasts that made *secondary dentin* are responsible for focal tertiary dentin formation, that particular type of tertiary dentin is termed *reactionary dentin*.[345] Generally, the rate of formation of dentin is increased, but the tubules remain continuous with the secondary dentin.[349] However, if the provoking stimulus caused the destruction of the original odontoblasts, the new, less tubular, more irregular dentin formed by newly differentiated odontoblast-like cells is called *reparative dentin*. In this dentin, the tubules are usually not continuous with those of secondary dentin. Initially, the newly formed cells tend to be cuboidal in shape, without the odontoblast process that is necessary to form dentinal tubules. They seem to form in response to the release of growth factors that were bound to collagen during the formation of secondary dentin.[94,315,345] The loss of the continuous layer of odontoblasts exposes unmineralized predentin that is thought to contain both soluble and insoluble forms of TGF-β, insulin-like growth factor (IGF)-1 and IGF-2, bone morphogenetic proteins (BMPs), VEGF, and other growth factors that attract and cause proliferation and differentiation of MSC to form reparative dentin and new blood vessels. During caries progression, bacterial acids may solubilize these growth factors from mineralized dentin, liberating them to diffuse to the pulp, where they could stimulate reactionary dentin formation. This is also thought to be the mechanism of action of calcium hydroxide during apexification treatment. Despite its high pH, calcium hydroxide has a slight demineralizing effect on dentin and has been shown to cause the release of TGF-β.[344] TGF-β and other growth factors stimulate and accelerate reparative dentinogenesis. Attempts have been made to apply

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growth factors to dentin to allow it to diffuse through the tubules to the pulp.[322,323,346] Although this has been successful, the remaining dentin thickness must be so thin that this approach may not be practical from a therapeutic perspective. Others have inserted deoxyribonucleic acid (DNA)-sequenced BMP-7 into retroviruses to transfect ferret pulpal fibroblasts to stimulate increased BMP-7 production. Although this was successful in normal pulps,[322] it was unsuccessful in inflamed pulps.[321] Specific amelogenin gene splice products, A + 4 and A − 4, adsorbed onto agarose beads and applied to pulp exposures, induced complete closure and mineralization of the root canal in rat molars.[124] The regulation of peritubular dentin formation is not fully understood. Some have claimed that this is a passive process resulting in occlusion of the tubules over time, but it has also been claimed that this is a mechanism under odontoblast control. If odontoblasts could be stimulated to form excessive peritubular dentin by the application of an appropriate biologic signaling molecule to the floor of cavity preparations, the tubules of the remaining dentin could be occluded, rendering this dentin impermeable and protecting the pulp from the inward diffusion of noxious substances that might leak around restorations.[297] These are examples of how molecular biology may be used in future restorative dentistry.

The term most commonly applied to irregularly formed dentin is *reparative dentin,* presumably because it frequently forms in response to injury and appears to be a component of the reparative process. It must be recognized, however, that this type of dentin has also been observed in the pulps of normal, unerupted teeth without any obvious injury.[281]

It should be recalled that secondary dentin is deposited circumpulpally at a slow rate throughout the life of the vital tooth.[349] In contrast, when a carious lesion has invaded dentin, the pulp usually responds by depositing a layer of tertiary dentin over the dentinal tubules of the primary or secondary dentin that communicate with the carious lesion (Fig. 13.43). Similarly, when occlusal wear removes the overlying enamel and exposes the dentin to the oral environment, tertiary dentin is deposited on the pulpal surface of the exposed dentin. Thus the formation of tertiary dentin allows the pulp to retreat behind a barrier of mineralized tissue.[375]

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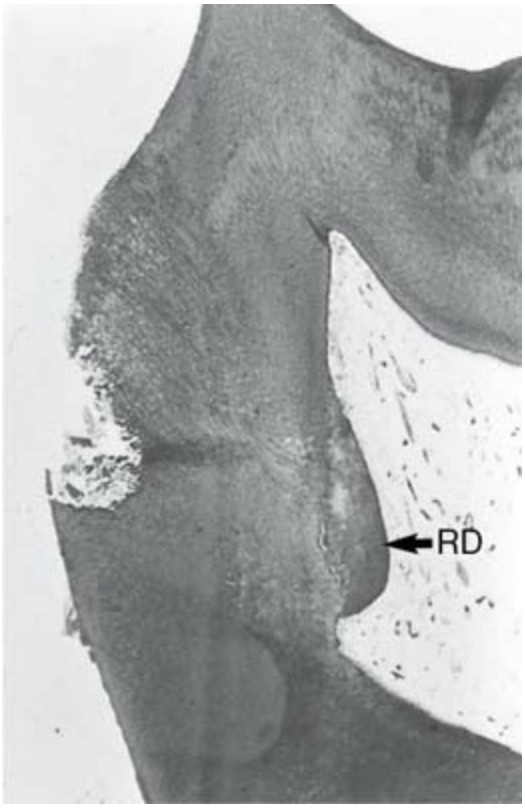

FIG. 13.43 Reparative dentin *(RD)* deposited in response to a carious lesion in the dentin.

Micrograph shows a U-shaped carious on the left and protuberance on the right.

Source: (From Trowbridge HO: Pathogenesis of pulpitis resulting from dental caries, *J Endod* 7:52, 1981.)

Compared with primary or secondary dentin, tertiary dentin tends to be less tubular, and the tubules tend to be more irregular with larger lumina. In some cases, particularly when the original odontoblasts are destroyed, no tubules are formed. The cells that form reparative dentin are often cuboidal and not as columnar as the primary odontoblasts of the coronal pulp (Fig. 13.44). The quality of tertiary dentin (i.e., the extent to which it resembles primary or secondary dentin) is quite variable. If irritation to the pulp is relatively mild, as in the case of a superficial carious lesion, then the tertiary dentin formed may resemble primary dentin in terms of tubularity and degree of mineralization. On the other hand, dentin deposited in response to a deep carious lesion may be relatively atubular and poorly mineralized, with many areas of interglobular dentin. The degree of irregularity of this dentin is

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probably determined by numerous factors, such as the amount of inflammation present, the extent of cellular injury, and the state of differentiation of the replacement odontoblasts.

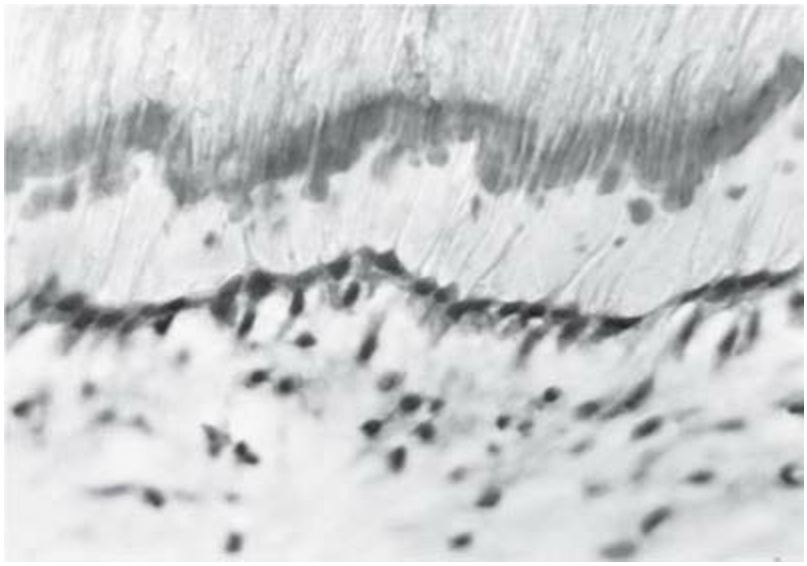

FIG. 13.44 Layer of cells forming reparative dentin. Note the decreased tubularity of reparative dentin compared with the developmental dentin above it.

Electron micrograph shows reparative dentin cells in odontoblast layer of tooth resulting in diminishing of columnar cells.

The poorest quality of reparative dentin is usually observed in association with marked pulpal inflammation.[65,375] In fact, the dentin may be so poorly organized that areas of soft tissue are entrapped within the dentinal matrix. In histologic sections, these areas of soft-tissue entrapment impart a Swiss-cheese appearance to the dentin (Fig. 13.45). As the entrapped soft tissue degenerates, products of tissue degeneration are released that further contribute to the inflammatory stimuli assailing the pulp.[375]

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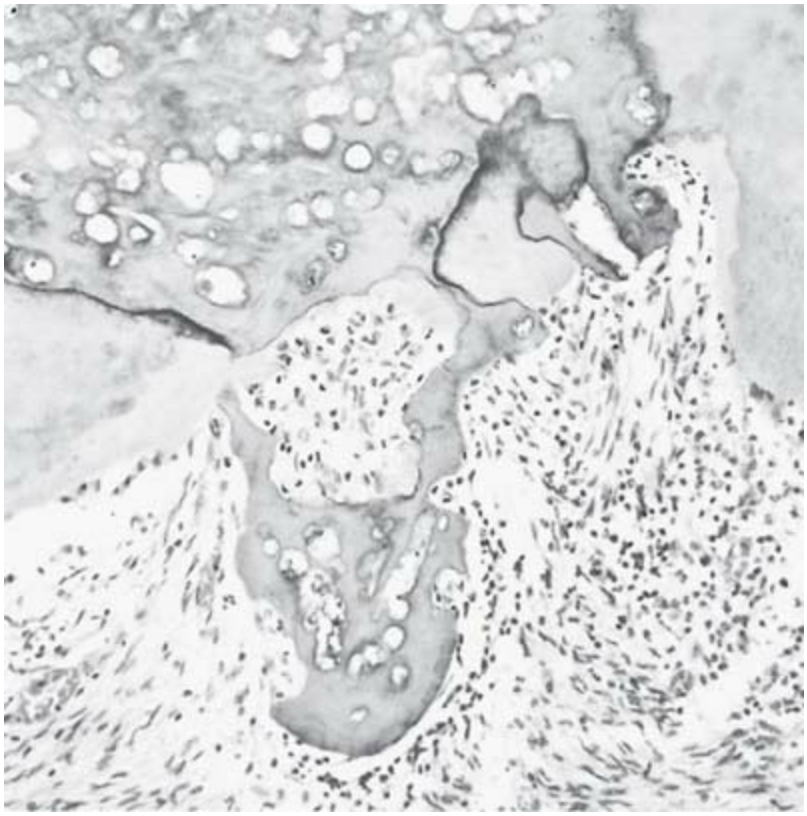

FIG. 13.45 Swiss-cheese type of reparative dentin. Note the numerous areas of soft-tissue inclusion and infiltration of inflammatory cells in the pulp.

Electron micrograph shows an irregular reparative dentin in pulp depicted as transparent irregular circles.

It has been reported that trauma caused by cavity preparation that is too mild to result in the loss of primary odontoblasts does not lead to reparative dentin formation, even if the cavity preparation is relatively deep.[74] This has been confirmed in both rat[52] and human teeth.[261] However, chronic pulpal inflammation associated with deep caries produces reparative dentin. This reparative dentin is formed by new odontoblast-like cells. For many years, it has been recognized that destruction of primary odontoblasts is soon followed by increased mitotic activity within fibroblasts of the subjacent cell-rich zone. It has been shown that the progeny of these dividing cells differentiate into functioning odontoblasts.[100] Investigators[421] have studied dentin bridge formation in healthy teeth of dogs and found that pulpal fibroblasts appeared to undergo dedifferentiation and revert to undifferentiated MSC (Fig. 13.46). The similarity of primary odontoblasts to

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replacement odontoblasts was established by D'Souza and colleagues.[78] They were able to show that cells forming reparative dentin synthesize type I (but not type III) collagen, and they are immunopositive for dentin sialoprotein.

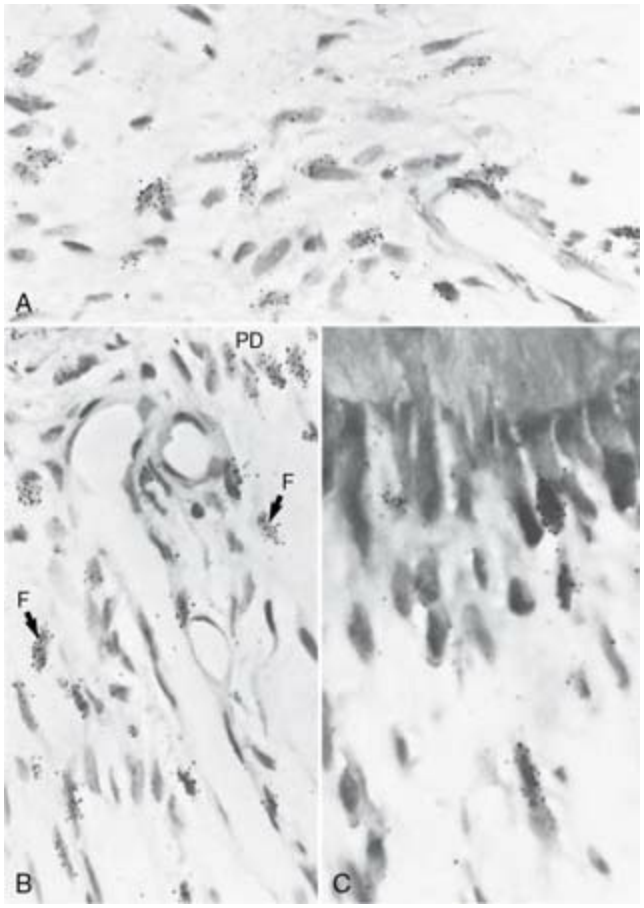

FIG. 13.46 Autoradiographs from dog molars illustrating uptake of ^{3}H-thymidine by pulp cells preparing to undergo cell division after pulpotomy and pulp capping with calcium hydroxide. **A,** Two days after pulp capping. Fibroblasts, endothelial cells, and pericytes beneath the exposure site are labeled. **B,** By the fourth day, fibroblasts *(F)* and preodontoblasts adjacent to the predentin *(PD)* are labeled, which suggests that differentiation of preodontoblasts occurred within 2 days. **C,** Six days after pulp capping, new odontoblasts are labeled, and tubular dentin is being formed. (Titrated thymidine was injected 2 days after the pulp capping procedures in [**B]** and [**C]**.)

Set of three radiographs marked A through C shows the development of columnar odontoblast cells below the dentin layer in molar tooth of dog.

Source: (From Yamamura T, Shimono M, Koike H, et al: Differentiation and induction of undifferentiated mesenchymal cells in tooth and

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periodontal tissue during wound healing and regeneration, *Bull Tokyo Dent Coll* 21:181, 1980.)

Destruction of primary odontoblasts can occur from cutting cavity preparations dry,[81,206] from bacterial products such as endotoxins shed from deep carious lesions,[18,402] or from mechanical exposure of pulps.[262] Such pulpal wounds do not heal if the tissue is irreversibly inflamed.[65] Local fibroblast-like cells divide, and the new cells then differentiate into new odontoblasts. Recalling the migratory potential of ectomesenchymal cells from which the pulpal fibroblasts are derived, it is not difficult to envision the differentiating odontoblasts moving from the subodontoblastic zone to the area of injury to constitute a new odontoblast layer. Activation of antigen-presenting dendritic cells by mild inflammatory processes may also promote osteoblast/odontoblast-like differentiation and expression of molecules implicated in mineralization. Recognition of bacteria by specific odontoblast and fibroblast membrane receptors triggers an inflammatory and immune response within the pulp tissue that would also modulate the repair process.[123]

Although many animal studies have shown dentin bridge formation in healthy pulps following pulp capping with adhesive resins,[65] such procedures fail in normal human teeth.[62]

When small mechanical pulp exposures are inadvertently made in healthy teeth, the recommendation has been to place a small, calcium hydroxide–containing dressing on the wound. After setting, the surrounding dentin can be bonded using a no-rinse, self-etching primer adhesive.[177] Calcium silicate cements like mineral trioxide aggregate (MTA) have also been recognized to promote hard-tissue formation, and available information indicates that the dentin bridge formed under these cements is more dense and has fewer defects compared with traditional calcium hydroxide–containing dressings.[3,5,266,373]

The formation of atubular “fibrodentin” is another potential product of newly differentiated odontoblasts, provided that a capillary plexus develops beneath the fibrodentin.[15] This is consistent with the observation that the newly formed dentin bridge is composed first of a thin layer of atubular dentin on which a relatively thick layer of tubular dentin is deposited.[65,100]

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The fibrodentin was lined by cells resembling mesenchymal cells, whereas the tubular dentin was associated with cells closely resembling odontoblasts.

Researchers have also studied reparative dentin formed in response to relatively traumatic experimental class V cavity preparations in human teeth.[349] They found that seldom was reparative dentin formed until about the 30th postoperative day. The rate of dentin formation was 3.5 μm/day for the first 3 weeks after the onset of dentinogenesis, after which it decreased markedly. By postoperative day 132, dentin formation had nearly ceased. Assuming that most of the odontoblasts were destroyed during traumatic cavity preparation, as was likely in this experiment, the 30-day delay between cavity preparation and the onset of reparative dentin formation is thought to reflect the time required for the proliferation, migration, and differentiation of new replacement odontoblasts.

Does reparative dentin protect the pulp, or is it simply a form of scar tissue? To serve a protective function, it would have to provide a relatively impermeable barrier that would exclude irritants from the pulp and compensate for the loss of developmental dentin. The junction between developmental and reparative dentin has been studied using a dye diffusion technique, which demonstrated the presence of an atubular zone situated between secondary dentin and reparative dentin (Fig. 13.47).[96] In addition to a dramatic reduction in the number of tubules, the walls of the tubules along the junction were often thickened and occluded with material similar to peritubular matrix.[332] Taken together, these observations would indicate that the junctional zone between developmental and reparative dentin is an atubular zone of low permeability. Moreover, the accumulation of pulpal dendritic cells was reduced after reparative dentin formation, which may indicate the reduction of incoming bacterial antigens.[325]

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FIG. 13.47 Diffusion of dye from the pulp into reparative dentin. Note atubular zone between reparative dentin (RD) and primary dentin on the left.

Autoradiograph shows the longitudinal view of highly dense dentin layer on the left, followed by a blank narrow zone, and wide zone of sparse tubules on the right.

Source: (From Fish EW: *Experimental investigation of the enamel, dentin, and dental pulp,* London, 1932, John Bale Sons & Danielson.)

Periodontally diseased teeth have smaller root canal diameters than teeth that are periodontally healthy.[207] The root canals of such teeth are narrowed by the deposition of large quantities of reactionary dentin along the dentinal walls.[333] The decrease in root canal diameter with increasing age, in the absence of periodontal disease, is more likely to be the result of secondary dentin formation.

One study showed that in a rat model, frequent scaling and root planing resulted in reparative dentin formation along the pulpal wall subjacent to the instrumented root surface.[148] However, given that normal rat root dentin is only 100 μm thick, these procedures are probably more traumatic to the pulp

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in the rat model than in humans, where normal root dentin is more than 2000 µm thick.

Not uncommonly, the cellular elements of the pulp are largely replaced by fibrous connective tissue over a span of five decades. It appears that in some cases, the pulp responds to noxious stimuli by accumulating large fiber bundles of collagen, rather than by elaborating reparative dentin (Fig. 13.48). However, fibrosis and reparative dentin formation often go hand in hand, indicating that both are expressions of a reparative potential. In periodontally diseased teeth, the pulp tissue is found to be the site of an enhanced process of collagenous fibrosis associated with an inflammatory infiltrate.[57]

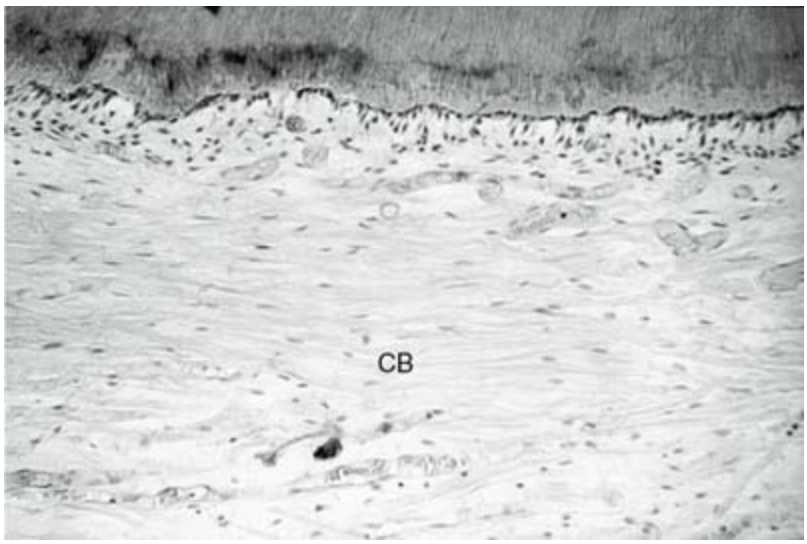

FIG. 13.48 Fibrosis of dental pulp showing replacement of pulp tissue by large collagen bundles *(CB)*.

Micrograph shows diffused collagen fibers replacing the cells in pulp below odontoblasts.

With the expanding knowledge of tooth regeneration and biologic mechanisms of functional dental tissue repair, current treatment strategies are beginning to give way to evolving fields such as tissue engineering and biomimetics. Pulpal stem cells in scaffolds have been shown to produce pulplike tissues with tubular-like dentin,[89] and in animal models, root perforations have been treated with scaffolds of collagen, pulpal stem cells, and dentin matrix protein 1, resulting in organized matrix similar to that of

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pulpal tissue.[310]

Studies investigating new possibilities for regeneration of the pulp/dentin complex are now frequently reported and are described in detail in Chapter 12. A first interesting case report, which has been followed up by others, has led to new strategies for treatment of necrotic immature roots (Fig. 13.49).[13] In the future, the field of pulpal repair will probably develop rapidly, and new treatment strategies will appear.

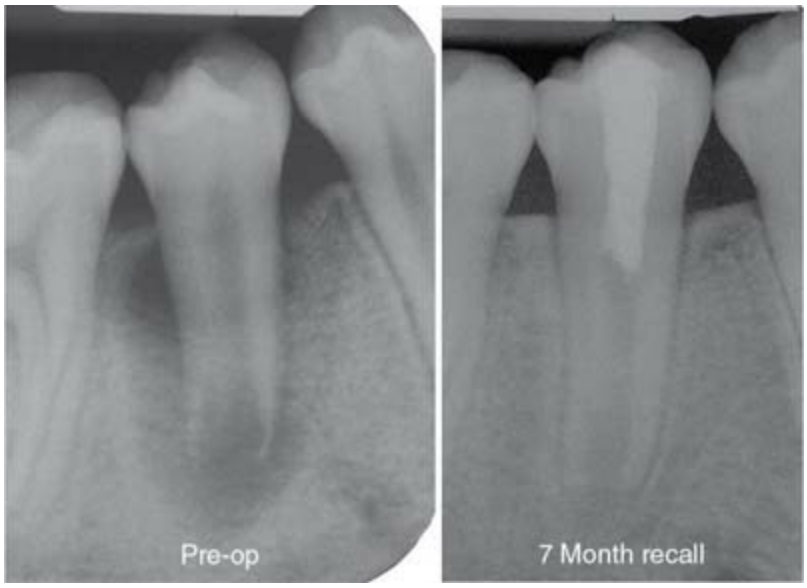

FIG. 13.49 Immature tooth with a necrotic infected canal with apical periodontitis. The canal is disinfected with copious irrigation with sodium hypochlorite and an antibiotic paste. Seven months after treatment, the patient is asymptomatic, and the apex shows healing of the apical periodontitis and some closure of the apex.

Pre-operative radiograph shows open root canal with a radiolucent patch at the root tip and porous tissue surrounding the root.

Radiograph after 7 month recall shows partially filled root canal.

Source: (From Banchs F, Trope M: Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? *J Endod* 30:196, 2004.)

Pulpal calcifications

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The calcification of pulp tissue is a common occurrence. Although estimates of the incidence of this phenomenon vary widely, it is safe to say that one or more pulp calcifications are present in at least 50% of all teeth. In the coronal pulp, calcification usually takes the form of discrete, concentric pulp stones (Fig. 13.50), whereas in the radicular pulp, calcification tends to be diffuse (Fig. 13.51).[379] There is no clear evidence as to whether pulp calcification is a pathologic process related to various forms of injury or a natural phenomenon. The clinical significance of pulp calcification is that it may hinder root canal treatment.

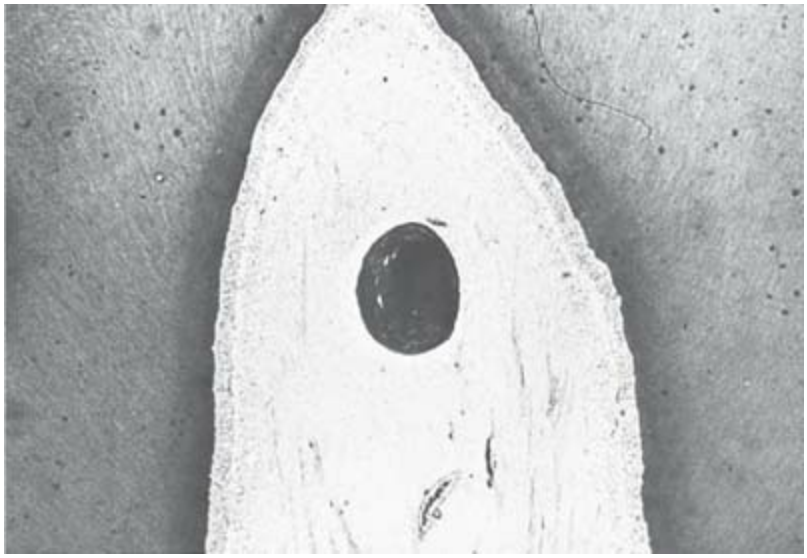

FIG. 13.50 Pulp stone with a smooth surface and concentric laminations in the pulp of a newly erupted premolar extracted in the course of orthodontic treatment.

Micrograph shows a round dark stone in the pulp.

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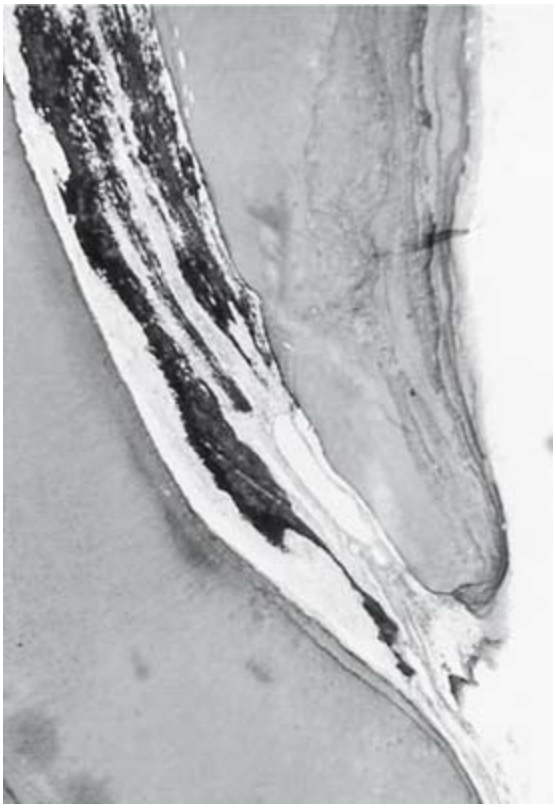

FIG. 13.51 Diffuse calcification near the apical foramen.

Micrograph shows the root apex of tooth with diffused calcification in the canal.

Pulp stones (denticles) range in size from small, microscopic particles often seen in association with the wall of arterioles to accretions that occupy almost the entire pulp chamber (Fig. 13.52). The mineral phase of pulp calcifications has been shown to consist of typical carbonated hydroxyapatite.[379] Histologically, two types of stones are recognized: (1) those that are round or ovoid, with smooth surfaces and concentric laminations (see Fig. 13.50), and (2) those that assume no particular shape, lack laminations, and have rough surfaces (Fig. 13.53). Laminated stones appear to grow by the addition of collagen fibrils to their surface, whereas unlaminated stones develop by way of the mineralization of preformed collagen fiber bundles. In the latter type, the mineralization front seems to extend out along the coarse fibers, making the surface of the stones appear fuzzy (Fig. 13.54). Often these coarse fiber bundles appear to have undergone hyalinization, thus resembling old scar tissue.

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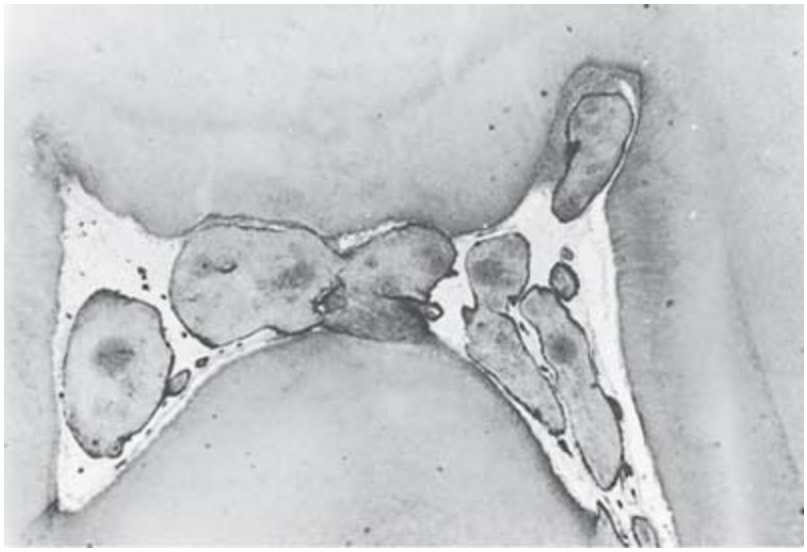

FIG. 13.52 Pulp stones occupying much of the pulp chamber.

Pulp chamber of tooth is filled with irregular and large stones. One stone is present in the pulp horn on the right.

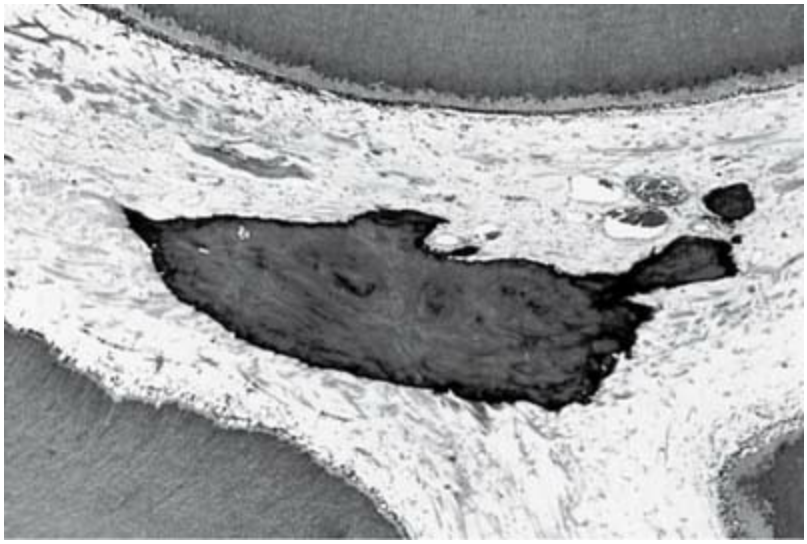

FIG. 13.53 Rough surface form of pulp stone. Note hyalinization of collagen fibers.

Magnified image shows roughly oval and irregular structure of pulp stone.

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FIG. 13.54 High-power view of a pulp stone from Fig. 13.53, showing the relationship of mineralization fronts to collagen fibers.

Highly magnified view shows half pulp stone surrounded by collagen fibers.

Pulp stones may also form around epithelial cells (i.e., remnants of Hertwig epithelial root sheath). Presumably the epithelial remnants induce adjacent MSC to differentiate into odontoblasts. Characteristically these pulp stones are found near the root apex and contain dentinal tubules (see Fig. 13.19).

The cause of pulpal calcification is largely unknown. Calcification may occur around a nidus of degenerating cells, blood thrombi, or collagen fibers. Many authors believe that this represents a form of dystrophic calcification. In this type of calcification, calcium is deposited in tissues that are degenerating. Calcium phosphate crystals may be deposited within the cells themselves. Initially this takes place within the mitochondria because of the increased membrane permeability to calcium resulting from a failure to maintain active transport systems within the cell membranes. Thus degenerating cells serving as a nidus may initiate calcification of a tissue. In the absence of obvious tissue degeneration, the cause of pulpal calcification is enigmatic. It is often difficult to assign the term *dystrophic calcification* to

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pulp stones because they so often occur in apparently healthy pulps, suggesting that functional stress need not be present for calcification to occur. Calcification in the mature pulp is often assumed to be related to the aging process, but in a study involving 52 impacted canines from patients between 11 and 76 years of age, there was a constant incidence of concentric denticles for all age groups, indicating no relation to aging.[281] Diffuse calcifications, on the other hand, increased in incidence to age 25 years; thereafter they remained constant in successive age groups.

At times, numerous concentric pulp stones with no apparent cause are seen in all the teeth of young individuals. In such cases, the appearance of pulp stones may be ascribed to individual biologic characteristics (e.g., tori, cutaneous nevi).[281]

Although soft-tissue collagen does not usually calcify, it is common to find calcification occurring in old hyalinized scar tissue in the skin. This may be due to the increase in the extent of cross-linking between collagen molecules (because increased cross-linkage is thought to enhance the tendency for collagen fibers to calcify). A relationship may exist between pathologic alterations in collagen molecules within the pulp and pulpal calcification.

Calcification replaces the cellular components of the pulp and may possibly hinder the blood supply, although concrete evidence for this strangulation theory is lacking. Idiopathic pulpal pain was classically attributed to the presence of pulp stones. Modern knowledge of mechanisms of nociceptor activation coupled with the observation that pulp stones are so frequently observed in teeth lacking a history of pain have largely discounted this hypothesis. Therefore from a clinical perspective, it would be unlikely that a patient's unexplained pain symptoms are due to pulpal calcifications, no matter how dramatic they may appear on a radiograph.

Luxation of teeth as a result of trauma may result in calcific metamorphosis, a condition that can, in a matter of months or years, lead to partial or complete radiographic obliteration of the pulp chamber. The cause of radiographic obliteration is excessive deposition of mineralized tissue resembling cementum or, occasionally, bone on the dentin walls, also referred to as *internal ankylosis* (Fig. 13.55). Histologic examination invariably reveals the presence of some soft tissue, and cells resembling cementoblasts can be observed lining the mineralized tissue. This calcific

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metamorphosis of the pulp has also been reported in replanted teeth of the rat.[280]

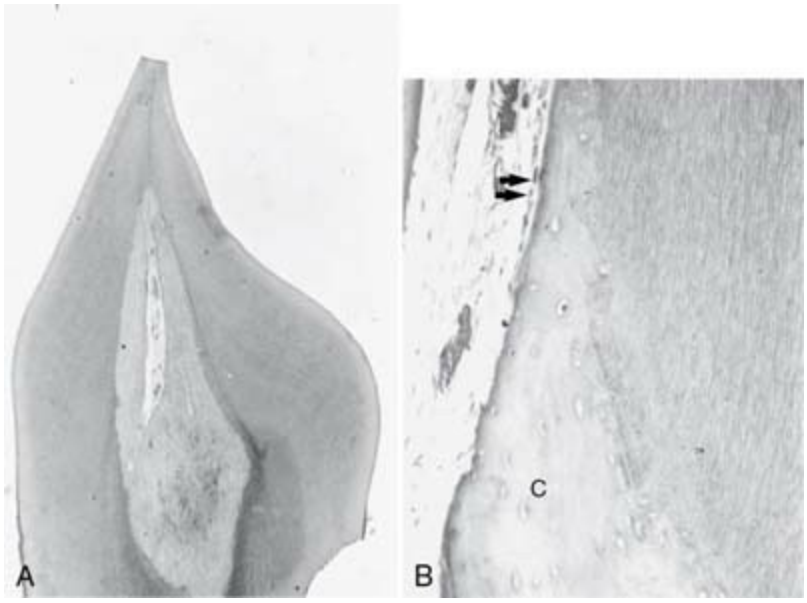

FIG. 13.55 **A,** Calcific metamorphosis of pulp tissue after luxation of tooth as a result of trauma. Note presence of soft-tissue inclusion. **B,** High-power view showing cementoblasts *(arrows)* lining cementum *(C)*, which has been deposited on the dentin walls.

A) Pulp shows deposition of calcium in root canal.

B) Arrows point toward the cementoblasts in pulp close to the dentin layer.

Clinically, the crowns of teeth affected by calcific metamorphosis may show a yellowish hue compared with adjacent normal teeth. This condition usually occurs in teeth with incomplete root formation. Trauma results in the disruption of blood vessels entering the tooth, thus producing pulpal infarction. The wide periapical foramen allows connective tissue from the periodontal ligament to proliferate and replace the infarcted tissue, bringing with it cementoprogenitor and osteoprogenitor cells capable of differentiating into either cementoblasts or osteoblasts or both.

When calcific metamorphosis is noted on a patient's radiograph, it is sometimes suggested that the tooth be treated endodontically because the pulp is expected to be secondarily infected, and endodontic therapy should be performed while the pulp canal is still large enough to instrument. In a classic

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study of luxated teeth, Andreasen[7] found that only 7% of the pulps that underwent calcific metamorphosis exhibited secondary infection. Because the success rate for nonsurgical endodontic therapy, not only in general[406] but also for obliterated teeth,[68] is considered high, prophylactic intervention does not seem to be warranted.

Age changes

Continued formation of secondary dentin throughout life gradually reduces the size of the pulp chamber and root canals, although the width of the cementodentinal junction appears to stay relatively the same.[111,349] However, due to continuous cement application, the distance from the apical constriction to the radiographic apex may increase (Fig. 13.56).[388] The progressive sclerosis of dentin starting apically also has endodontic implications, as it makes dentine almost impermeable to bacterial invasion (see Fig. 13.56).[347] In addition, certain regressive changes in the pulp appear to be related to the aging process. There is a gradual decrease in the cellularity and a concomitant increase in the number and thickness of collagen fibers, particularly in the radicular pulp. The thick collagen fibers may serve as foci for pulpal calcification (see Fig. 13.53). The odontoblasts decrease in size and number, and they may disappear altogether in certain areas of the pulp, particularly on the pulpal floor over the bifurcation or trifurcation areas of multirooted teeth.

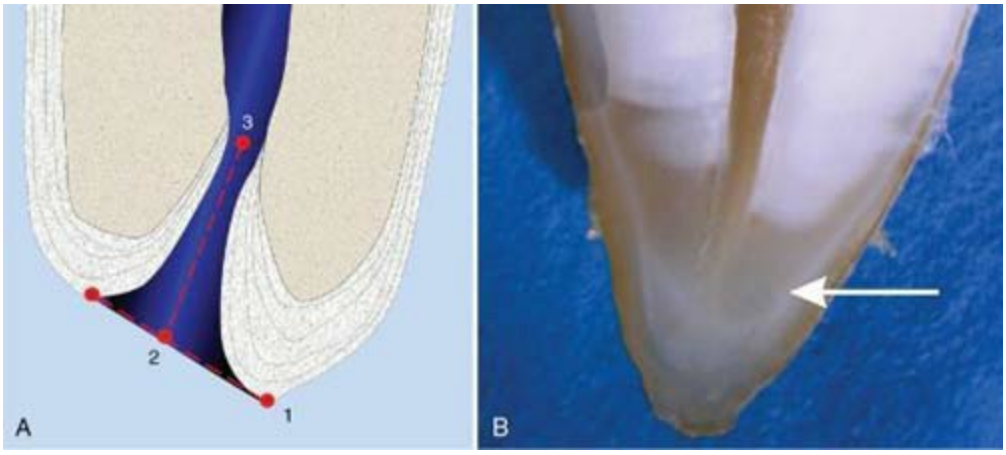

FIG. 13.56 Anatomical structures in the apical area. **A**, Cementum

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apposition during life will change the apical anatomical landmarks. Number *1* denotes the radiographic apex, *2* the apical foramen (major) and *3* the apical constriction (the minor apical foramen) representing the transition between the pulpal and periodontal tissues. The line that goes through *1* and *2* is often denoted the anatomical apex. **B**, Age-related sclerosis, making the dentinal tubules impermeable to bacteria, is a progressive event starting in the apical area *(white arrow)*.

A) Three points in the root canal near the apex are marked 1 through 3.

B) Root apex shows the loss of normal tissues at the apex comparatively rest of the structure in root.

With age there is a progressive reduction in the number of nerves[104] and blood vessels.[23,25] Evidence also suggests that aging results in an increase in the resistance of pulp tissue to the action of proteolytic enzymes,[427] hyaluronidase, and sialidase,[25] suggesting an alteration of both collagen and proteoglycans in the pulps of older teeth. The main changes in dentin associated with aging are an increase in peritubular dentin, dentinal sclerosis, and the number of dead tracts.[¶,349] Dentinal sclerosis produces a gradual decrease in dentinal permeability as the dentinal tubules become progressively reduced in diameter.[357]

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References

1. Aars H, Brodin P, Anderson E. *A study of cholinergic and b-adrenergic components in the regulation of blood flow in the tooth pulp and gingiva of man*, Acta Physiol Scand 1993;148: 441.
2. Aars H, Gazelius B, Edwall L, Olgart L. *Effects of autonomic reflexes on tooth pulp blood flow in man*, Acta Physiol Scand 1992;146: 423.
3. Accorinte ML, Loguercio AD, Reis A, et al: *Response of human dental pulp capped with MTA and calcium hydroxide powder*, Oper Dent 2008;33: 488.
4. Amess TR, Matthews B. *The effect of topical application of lidocaine to dentin in the cat on the response of intra-dental nerves to mechanical stimuli*, Shimono M Maeda T Suda H Takahashi K Proceedings of the international conference on dentin/pulp complex 1996; Quintessence Publishing Tokyo.
5. Andelin WE, Shabahang S, Wright K, Torabinejad M. *Identification of hard tissue after experimental pulp capping using dentin sialoprotein (DSP) as a marker*, J Endod 2003;29: 646.
6. Anderson LC, Vakoula A, Veinote R. *Inflammatory hypersensitivity in a rat model of trigeminal neuropathic pain*, Arch Oral Biol 2003;48: 161.
7. Andreasen JO. *Luxation of permanent teeth due to trauma: a clinical and radiographic follow-up study of 189 injured teeth*, Scand J Dent Res 1970;78: 273.
8. Anneroth G, Norberg KA. *Adrenergic vasoconstrictor innervation in the human dental pulp*, Acta Odontol Scand 1968;26: 89.

Telegram: @dental_k

www.konkur.in

9. Arbuckle JB, Docherty RJ. *Expression of tetrodotoxin-resistant sodium channels in capsaicin-sensitive dorsal root ganglion neurons of adult rats*, Neurosci Lett 1995;185: 70.
10. Artese L, Rubini C, Ferrero G, et al: *Vascular endothelial growth factor (VEGF) expression in healthy and inflamed human dental pulps*, J Endod 2002;28: 20.
11. Avery JK. *Structural elements of the young normal human pulp*, Oral Surg Oral Med Oral Pathol 1971;32: 113.
12. Awawdeh L, Lundy FT, Shaw C, et al: *Quantitative analysis of substance P, neurokinin A, and calcitonin gene-related peptide in pulp tissue from painful and healthy human teeth*, Int Endod J 2002;36: 30.
13. Banchs F, Trope M. *Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol?*, J Endod 2004;30: 196.
14. Basbaum AI, Fields HL. *Endogenous pain control systems: brainstem spinal pathways and endorphin circuitry*, Ann Rev Neurosci 1984;7: 309.
15. Baume LJ. *The biology of pulp and dentine*, Myers HM Monographs in oral science 1980;vol 8: S Karger AG Basel.
16. Beck P, Handwerker H. *Bradykinin and serotonin effects on various types of cutaneous nerve fibers*, Pflugers Arch 1974;347: 209.
17. Bender IB, Landau MA, Fonsecca S, et al: *The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth*, J Am Dent Assoc 1989;118: 305.
18. Bergenholtz G. *Evidence for bacterial causation of adverse pulpal responses in resin-based dental restorations*, Crit Rev Oral Biol Med 2000;11: 467.
19. Berggreen E, Haug SR, Mkony LE, et al:

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www.konkur.in

Characterization of the dental lymphatic system and identification of cells immunopositive to specific lymphatic markers, Eur J Oral Sci 2009;117: 34.

20. Berggreen E, Heyeraas KJ. *The role of sensory neuropeptides and nitric oxide on pulpal blood flow and tissue pressure in the ferret*, J Dent Res 1999;78: 1535.
21. Berggreen E, Heyeraas KJ. *Effect of the sensory neuropeptide antagonists h-CGRP(8) and SR 140.33 on pulpal and gingival blood flow in ferrets*, Arch Oral Biol 2000;45: 537.
22. Berggreen E, Heyeraas KJ. *Role of K1ATP channels, endothelin A receptors, and effect of angiotensin II on blood flow in oral tissues*, J Dent Res 2003;82: 33.
23. Bernick S, Nedelman C. *Effect of aging on the human pulp*, J Endod 1975;1: 88.
24. Bevilacqua MP, et al: *Interleukin-1 activation of vascular endothelium. Effects on procoagulant activity and leukocyte adhesion*, Am J Pathol 1985;121: 394.
25. Bhussary BR. *Modification of the dental pulp organ during development and aging*, Finn SB Biology of the dental pulp organ: a symposium 1968; University of Alabama Press Birmingham.
26. Biesterfeld RC, Taintor JF, Marsh CL. *The significance of alterations of pulpal respiration: a review of the literature*, J Oral Pathol 1979;8: 129.
27. Bisgaard H, Kristensen J. *Leukotriene B4 produces hyperalgesia in humans*, Prostaglandins 1985;30: 791.
28. Deleted in page review.
29. Bishop MA, Yoshida S. *A permeability barrier to lanthanum and the presence of collagen between odontoblasts in pig molars*, J Anat 1992;181: 29.
30. Bletsa A, Abdalla H, Loes S, et al: *Lymphatic growth*

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factors are expressed in human gingiva and upregulated in gingival fibroblasts after stimulation, J Periodontol 2018;89: 606.

31. Bletsa A, Berggreen E, Fristad I, et al: *Cytokine signalling in rat pulp interstitial fluid and transcapillary fluid exchange during lipopolysaccharide-induced acute inflammation*, J Physiol Pt 1, 2006;573: 225.
32. Bongenhielm U, Haegerstrand A, Theodorsson E, Fried K. *Effects of neuropeptides on growth of cultivated rat molar pulp fibroblasts*, Regul Pept 1995;60: 2391.
33. Borda E, Furlan C, Orman B, et al: *Nitric oxide synthase and PGE2 reciprocal interactions in rat dental pulp: cholinoceptor modulation*, J Endod 2007;33: 142.
34. Botero TM, Shelburne CE, Holland GR, et al: *TLR4 mediates LPS-induced VEGF expression in odontoblasts*, J Endod 2006;32: 951.
35. Bowles WR, Flores CM, Jackson DL, et al: *beta 2-Adrenoceptor regulation of CGRP release from capsaicin-sensitive neurons*, J Dent Res 2003;82: 308.
36. Brännström M. *The transmission and control of dentinal pain*, Grossman LJ Mechanisms and control of pain 1979; Masson Publishing USA New York.
37. Brännström M. *Communication between the oral cavity and the dental pulp associated with restorative treatment*, Oper Dent 1984;9: 57.
38. Brännström M, Aström A. *A study of the mechanism of pain elicited from the dentin*, J Dent Res 1964;43: 619.
39. Breschi L, Lopes M, Gobbi P, et al: *Dentin proteoglycans: an immunocytochemical FEISEM study*, J Biomed Mater Res 2002;61: 40.
40. Brown AC, Yankowitz D. *Tooth pulp tissue pressure and hydraulic permeability*, Circ Res 1964;15: 42.

Telegram: @dental_k

www.konkur.in

41. Butler WT, D'Sousa RN, Bronckers AL, et al: *Recent investigations on dentin specific proteins*, Proc Finn Dent Soc suppl 1, 1992;88: 369.
42. Byers MR. *Dynamic plasticity of dental sensory nerve structure and cytochemistry*, Arch Oral Biol Suppl, 1994;39: 13S.
43. Byers MR. *Neuropeptide immunoreactivity in dental sensory nerves: variations related to primary odontoblast function and survival*, Shimono M Takahashi K Dentin/pulp complex 1996; Quintessence Publishing Tokyo.
44. Byers MR, Chudler EH, Iadarola MJ. *Chronic tooth pulp inflammation causes transient and persistent expression of Fos in dynorphin-rich regions of rat brainstem*, Brain Res 2000;861: 191.
45. Byers MR, Narhi MV, Mecifi KB. *Acute and chronic reactions of dental sensory nerve fibers to cavities and desiccation in rat molars*, Anat Rec 1988;221: 872.
46. Byers MR, Narhi MVO. *Dental injury models: experimental tools for understanding neuroinflammatory interactions and polymodal nociceptor functions*, Crit Rev Oral Biol Med 1999;10: 4.
47. Byers MR, Närhi MVO. *Nerve supply of the pulpodentin complex and response to injury*, Hargreaves K Goodis H Seltzer and Bender's dental pulp 2002; Quintessence Publishing Chicago.
48. Byers MR, Schatteman GC, Bothwell MA. *Multiple functions for NGF-receptor in developing, aging and injured rat teeth are suggested by epithelial, mesenchymal and neural immunoreactivity*, Development 1990;109: 461.
49. Byers MR, Sugaya A. *Odontoblast process in dentin*

Telegram: @dental_k

www.konkur.in

revealed by fluorescent Di-I, J Histochem Cytochem 1995;43: 159.

50. Byers MR, Suzuki H, Maeda T. *Dental neuroplasticity, neuro-pulpal interactions and nerve regeneration*, Microsc Res Tech 2003;60: 503.
51. Byers MR, Taylor PE. *Effect of sensory denervation on the response of rat molar pulp to exposure injury*, J Dent Res 1993;72: 613.
52. Byers MR, Taylor PE, Khayat BG, et al: *Effects of injury and inflammation on pulpal and periapical nerves*, J Endod 1990;16: 78.
53. Byers MR, Westenbroek RE. *Odontoblasts in developing, mature and ageing rat teeth have multiple phenotypes that variably express all nine voltage-gated sodium channels*, Arch Oral Biol 2011;56: 1199.
54. Byers MR, Wheeler EF, Bothwell M. *Altered expression of NGF and P75 NGF-receptor by fibroblasts of injured teeth precedes sensory nerve sprouting*, Growth Factors 1992;6: 41.
55. Camps J, Pashley DH. *In vivo sensitivity to air blasts and scratching of human root dentin*, J Periodontol 2003;74: 1589.
56. Camps J, Salomon JP, Van Meerbeek B, et al: *Dentin deformation after scratching with clinically-relevant forces*, Arch Oral Biol 2003;48: 527.
57. Caraivan O, Manolea H, Corlan Puscu D, et al: *Microscopic aspects of pulpal changes in patients with chronic marginal periodontitis*, Rom J Morphol Embryol 2012;53: 725.
58. Casasco A, Calligaro A, Casasco M, et al: *Immunohistochemical localization of endothelin-like immunoreactivity in human tooth germ and mature dental*

Telegram: @dental_k

www.konkur.in

pulp, Anat Embryol (Berl) 1991;183: 515.

59. Chaudhary P, Martenson ME, Baumann TK. *Vanilloid receptor expression and capsaicin excitation of rat dental primary afferent neurons*, J Dent Res 2001;80: 1518.
60. Chiang CY, Park SJ, Kwan CL, et al: *NMDA receptor mechanisms contribute to neuroplasticity induced in caudalis nociceptive neurons by tooth pulp stimulation*, J Neurophysiol 1998;80: 2621.
61. Chu SC, Tsai CH, Yang SF, et al: *Induction of vascular endothelial growth factor gene expression by proinflammatory cytokines in human pulp and gingival fibroblasts*, J Endod 2004;30: 704.
62. Costos CAS, Hebling J, Hanks CT. *Current status of pulp capping with dentin adhesive systems: a review*, Dent Mater 2000;16: 188.
63. Coure E. *Ultrastructural changes during the life cycle of human odontoblasts*, Arch Oral Biol 1986;31: 643.
64. Cousins M, Power I. *Acute and postoperative pain*, Wall P Melzack R Textbook of pain 2002; Churchill Livingstone Edinburgh 456.
65. Cox CF, Bogen G, Kopel HM, et al: *Repair of pulpal injury by dental materials*, Hargreaves K Goodis H Seltzer and Bender's dental pulp 2002; Quintessence Publishing Chicago.
66. Cox CF, White KC, Ramus DL, et al: *Reparative dentin: factors affecting its deposition*, Quintessence Int 1992;23: 257.
67. Csillag M, Berggreen E, Fristad I, et al: *Effect of electrical tooth stimulation on blood flow and immunocompetent cells in rat dental pulp after sympathectomy*, Acta Odontol Scand 2004;62: 305.
68. Cvek M, Granath L, Lundberg M. *Failures and healing in*

Telegram: @dental_k

www.konkur.in

endodontically treated non-vital anterior teeth with posttraumatically reduced pulpal lumen, Acta Odontol Scand 1982;40: 223.

69. Deleted in page review.
70. Dahl T, Sabsay B, Veis A. *Type I collagen-phosphophoryn interactions: specificity of the monomer-monomer binding*, J Struct Biol 1998;123: 162.
71. Dallel R, Clavelou P, Woda A. *Effects of tractotomy on nociceptive reactions induced by tooth pulp stimulation in the rat*, Exp Neurol 1989;106: 78.
72. Davidson RM. *Neural form of voltage-dependent sodium current in human cultured dental pulp cells*, Arch Oral Biol 1994;39: 613.
73. Diamond J. *The effect of injecting acetylcholine into normal and regenerating nerves*, J Physiol (Lond) 1959;145: 611.
74. Diamond RD, Stanley HR, Swerdlow H. *Reparative dentin formation resulting from cavity preparation*, J Prosthet Dent 1966;16: 1127.
75. Diogenes A, Akopian AN, Hargreaves KM. *NGF upregulates TRPA1: implications for orofacial pain*, J Dent Res 2007;86: 550.
76. Dionne R. *Oral sedation*, Compend Contin Educ Dent 1998;19: 868.
77. Dray A. *Inflammatory mediators of pain*, Br J Anaesth 1995;75: 125.
78. D'Souza RN, Bachman T, Baumgardner KR, et al: *Characterization of cellular responses involved in reparative dentinogenesis in rat molars*, J Dent Res 1995;74: 702.
79. Dworkin SF. *Anxiety and performance in the dental environment: an experimental investigation*, J Am Soc

Telegram: @dental_k

www.konkur.in

Psychosom Dent Med 1967;14: 88.
80. Ebihara A, Tokita Y, Izawa T, et al: *Pulpal blood flow assessed by laser Doppler flowmetry in a tooth with a horizontal root fracture*, Oral Surg Oral Med Oral Path 1996;81: 229.
81. Eda S, Saito T. *Electron microscopy of cells displaced into the dentinal tubules due to dry cavity preparation*, J Oral Pathol 1978;7: 326.
82. Edwall B, Gazelius B, Fazekas A, et al: *Neuropeptide Y (NPY) and sympathetic control of blood flow in oral mucosa and dental pulp in the cat*, Acta Physiol Scand 1985;125: 253.
83. Edwall L, Kindlová M. *The effect of sympathetic nerve stimulation on the rate of disappearance of tracers from various oral tissues*, Acta Odontol Scand 1971;29: 387.
84. El Karim IA, Linden GJ, Curtis TM, et al: *Human odontoblasts express functional thermo-sensitive TRP channels: implications for dentin sensitivity*, Pain 2011;152: 2211.
85. Embery G. *Glycosaminoglycans of human dental pulp*, J Biol Buccale 1976;4: 229.
86. Embery G, Hall R, Waddington R, et al: *Proteoglycans in dentinogenesis*, Crit Rev Oral Biol Med 2001;12: 331.
87. Deleted in page review.
88. England S. *Molecular basis of peripheral hyperalgesia*, Wood J Molecular basis of pain induction 2000; Wiley-Liss, p 261 New York.
89. Engström C, Linde A, Persliden B. *Acid hydrolases in the odontoblast-predentin region of dentinogenically active teeth*, Scand J Dent Res 1976;84: 76.
90. Evans D, Reid T, Strang R, et al: *A comparison of laser Doppler flowmetry with other methods of assessing vitality*

Telegram: @dental_k

www.konkur.in

in traumatized anterior teeth, Endod Dent Traumatol 1999;15: 284.

91. Deleted in page review.
92. Fearnhead RW. *Innervation of dental tissues*, Miles AEW Structural and chemical organization of the teeth 1967;vol 1: Academic Press New York.
93. Ferrara N. *Vascular endothelial growth factor*, Eur J Cancer 1996;32A: 2413.
94. Finkelman RD, Mohan S, Jennings JC, et al: *Quantitation of growth factors IGF-1, SGF/IGF-11 and TGF-b in human dentin*, J Bone Miner Res 1990;5: 717.
95. Firestone AR, Wheatley AM, Thüer UW. *Measurement of blood perfusion in the dental pulp with laser Doppler flowmetry*, Int J Microcirc Clin Exp 1997;17: 298.
96. Fish WE. An experimental investigation of enamel, dentine and the dental pulp 1932; John Bale, Sons & Danielson London.
97. Fisher AK. *Respiratory variations within the normal dental pulp*, J Dent Res 1967;46: 424.
98. Fisher AK, Schumacher ER, Robinson NR, et al: *Effects of dental drugs and materials on the rate of oxygen consumption in bovine dental pulp*, J Dent Res 1957;36: 447.
99. Fisher AK, Walters VE. *Anaerobic glycolysis in bovine dental pulp*, J Dent Res 1968;47: 717.
100. Fitzgerald M, Chiego DJ, Heys DR. *Autoradiographic analysis of odontoblast replacement following pulp exposure in primate teeth*, Arch Oral Biol 1990;35: 707.
101. Follenfant R, Nakamura-Craig M, Henderson B, et al: *Inhibition by neuropeptides of interleukin-1ß-induced, prostaglandin-independent hyperalgesia*, Br J Pharmacol 1989;98: 41.

Telegram: @dental_k

www.konkur.in

102. Fouad A. *Molecular mediators of pulpal inflammation*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Chicago 247.
103. Fraser JR, Kimpton WG, Laurent TC, et al: *Uptake and degradation of hyaluronan in lymphatic tissue*, Biochem J 1988;256: 153.
104. Fried K. *Changes in pulp nerves with aging*, Proc Finn Dent Soc Suppl 1, 1992;88: 517.
105. Fried K, Sessle BJ, Devor M. *The paradox of pain from tooth pulp: low-threshold "algoneurons"?*, Pain 2011;152: 2685.
106. Fristad I, Heyeraas KJ, Kvinnsland I. *Nerve fibres and cells immunoreactive to neurochemical markers in developing rat molars and supporting tissues*, Arch Oral Biol 1994;39: 633.
107. Fristad I, Jacobsen EB, Kvinnsland IH. *Coexpression of vasoactive intestinal polypeptide and substance P in reinnervating pulpal nerves and in trigeminal ganglion neurones after axotomy of the inferior alveolar nerve in the rat*, Arch Oral Biol 1998;43: 183.
108. Fristad I, Kvinnsland IH, Jonsson R, et al: *Effect of intermittent long-lasting electrical tooth stimulation on pulpal blood flow and immunocompetent cells: a hemodynamic and immunohistochemical study in young rat molars*, Exp Neurol 1997;146: 230.
109. Fristad I, Vandevska-Radunovic V, Fjeld K, et al: *NK1, NK2, NK3 and CGRP1 receptors identified in rat oral soft tissues, and in bone and dental hard tissue cells*, Cell Tissue Res 2003;311: 383.
110. Fuss Z, Trowbridge H, Bender IB, et al: *Assessment of reliability of electrical and thermal pulp testing agents*, J Endod 1986;12: 301.

Telegram: @dental_k

www.konkur.in

111. Gani O, Visvisian C. *Apical canal diameter in the first upper molar at various ages*, J Endod 1999;10: 689.
112. Garant PR. *The organization of microtubules within rat odontoblast processes revealed by perfusion fixation with glutaraldehyde*, Arch Oral Biol 1972;17: 1047.
113. Garberoglio R, Brännström M. *Scanning electron microscopic investigation of human dentinal tubules*, Arch Oral Biol 1976;21: 355.
114. Gedney JJ, Logan H, Baron RS. *Predictors of short-term and long-term memory of sensory and affective dimensions of pain*, J Pain 2003;4: 47.
115. George CH, Kendall JM, Evans WH. *Intracellular trafficking pathways on assembly of connexins into tight junctions*, J Biol Chem 1999;274: 8678.
116. Gerli R, Secciani I, Sozio F, et al: *Absence of lymphatic vessels in human dental pulp: a morphological study*, Eur J Oral Sci 2010;118: 110.
117. Gibbs JL, Hargreaves KM. *Neuropeptide Y Y1 receptor effects on pulpal nociceptors*, J Dent Res 2008;87: 948.
118. Gilbert TM, Pashley DH, Anderson RW. *Response of pulpal blood flow to intra-arterial infusion of endothelin*, J Endod 1992;18: 228.
119. Gilron I, Max MB, Lee G, et al: *Effects of the 2-amino-3-hydroxy-5-methyl-4-isoxazole-proprionic acid/kainate antagonist LY293558 on spontaneous and evoked postoperative pain*, Clin Pharmacol Ther 2000;68: 320.
120. Gloe T, Pohl U. *Laminin binding conveys mechanosensing in endothelial cells*, News Physiol Sci 2002;17: 166.
121. Gold M. *Tetrodotoxin-resistant Na currents and inflammatory hyperalgesia*, Proc Natl Acad Sci U S A 1999;96: 7645.

Telegram: @dental_k

www.konkur.in

122. Gold MS, Reichling DB, Shuster MJ, et al: *Hyperalgesic agents increase a tetrodotoxin-resistant Na1 current in nociceptors*, Proc Natl Acad Sci U S A 1996;93: 1108.
123. Goldberg M, Farges J-C, Lacerda-Pinheiro S, et al: *Inflammatory and immunological aspects of dental pulp repair*, Pharmacol Res 2008;58: 137.
124. Goldberg M, Six N, Decup F, et al: *Bioactive molecules and the future of pulp therapy*, Am J Dent 2003;16: 66.
125. Goldberg M, Takagi M. *Dentine proteoglycans: composition, ultrastructure and functions*, Histochem J 1993;25: 781.
126. Goodis H, Bowles W, Hargreaves K. *Prostaglandin E2 enhances bradykinin-evoked iCGRP release in bovine dental pulp*, J Dent Res 2000;79: 1604.
127. Gordon SM, Dionne RA, Brahim J, et al: *Blockade of peripheral neuronal barrage reduces postoperative pain*, Pain 1997;70: 209.
128. Gotjamanos T. *Cellular organization in the subodontoblastic zone of the dental pulp. II. Period and mode of development of the cell-rich layer in rat molar pulps*, Arch Oral Biol 1969;14: 1011.
129. Gould HJ, England JD, Soignier RD, et al: *Ibuprofen blocks changes in Na v 1.7 and 1.8 sodium channels associated with complete Freund's adjuvant-induced inflammation in rat*, J Pain 2004;5: 270.
130. Gould HJ 3rd, Gould TN, Paul D, et al: *Development of inflammatory hypersensitivity and augmentation of sodium channels in rat dorsal root ganglia*, Brain Res 1999;824: 296.
131. Gregg JM, Dixon AD. *Somatotopic organization of the trigeminal ganglion*, Arch Oral Biol 1973;18: 487.
132. Grossman ES, Austin JC. *Scanning electron microscope*

Telegram: @dental_k

www.konkur.in

observations on the tubule content of freeze-fractured peripheral vervet monkey dentine (Cercopithecus pygerythrus), Arch Oral Biol 1983;28: 279.

133. Gunji T. *Morphological research on the sensitivity of dentin*, Arch Histol Jpn 1982;45: 45.
134. Hagermark O, Hokfelt T, Pernow B. *Flare and itch produced by substance P in human skin*, J Invest Dermatol 1979;71: 233.
135. Hahn C-L, Falkler WA Jr, Siegel MA. *A study of T cells and B cells in pulpal pathosis*, J Endod 1989;15: 20.
136. Hals E, Tonder KJ. *Elastic pseudoelastic tissue in arterioles of the human and dog dental pulp*, Scand J Dent Res 1981;89: 218.
137. Hamersky PA, Weimer AD, Taintor JF. *The effect of orthodontic force application on the pulpal tissue respiration rate in the human premolar*, Am J Orthod 1980;77: 368.
138. Han SS. *The fine structure of cells and intercellular substances of the dental pulp*, Finn SB Biology of the dental pulp organ 1968; University of Alabama Press Birmingham 103.
139. Hargreaves KM. *Pain mechanisms of the pulpodentin complex*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Company Chicago 181.
140. Hargreaves KM, Bowles WR, Jackson DL. *Intrinsic regulation of CGRP release by dental pulp sympathetic fibers*, J Dent Res 2003;82: 398.
141. Hargreaves KM, Dionne RA, Mueller GP, et al: *Naloxone, fentanyl, and diazepam modify plasma beta-endorphin levels during surgery*, Clin Pharmacol Ther 1986;40: 165.

Telegram: @dental_k

www.konkur.in

142. Hargreaves KM, Keiser K. *Local anesthetic failure in endodontics: mechanisms and management*, Endod Topics 2002;1: 26.
143. Hargreaves KM, Milam SB. *Mechanisms of pain and analgesia*, Dionne R Phero J Management of pain and anxiety in dental practice 2001; Elsevier New York 18.
144. Hargreaves KM, Swift JQ, Roszkowski MT, et al: *Pharmacology of peripheral neuropeptide and inflammatory mediator release*, Oral Surg Oral Med Oral Pathol 1994;78: 503.
145. Harris R, Griffin CJ. *Fine structure of nerve endings in the human dental pulp*, Arch Oral Biol 1968;13: 773.
146. Hartmann A, Azerad J, Boucher Y. *Environmental effects on laser Doppler pulpal blood-flow measurements in man*, Arch Oral Biol 1996;41: 333.
147. Hashioka K, Suzuki K, Yoshida T, et al: *Relationship between clinical symptoms and enzyme-producing bacteria isolated from infected root canals*, J Endod 1994;20: 75.
148. Hattler AB, Listgarten MA. *Pulpal response to root planing in a rat model*, J Endod 1984;10: 471.
149. Haug SR, Heyeraas KJ. *Effects of sympathectomy on experimentally induced pulpal inflammation and periapical lesions in rats*, Neuroscience 2003;120: 827.
150. Haug SR, Heyeraas KJ. *Modulation of dental inflammation by the sympathetic nervous system*, J Dent Res 2006;85: 488.
151. Hermanstyne TO, Markowitz K, Fan L, et al: *Mechanotransducers in rat pulpal afferents*, J Dent Res 2008;87: 834.
152. Heyeraas KJ. *Pulpal hemodynamics and interstitial fluid pressure: balance of transmicrovascular fluid transport*, J Endod 1989;15: 468.

Telegram: @dental_k

www.konkur.in

153. Heyeraas KJ, Berggreen E. *Interstitial fluid pressure in normal and inflamed pulp*, Crit Rev Oral Biol Med 1999;10: 328.
154. Heyeraas KJ, Jacobsen EB, Fristad I. *Vascular and immunoreactive nerve fiber reactions in the pulp after stimulation and denervation: proceedings of the International Conference*, Shimono M Maeda T Suda H. Dentin/pulp complex 1996; Quintessence Publishing Tokyo 162.
155. Heyeraas KJ, Kim S, Raab WH, et al: *Effect of electrical tooth stimulation on blood flow, interstitial fluid pressure and substance P and CGRP-immunoreactive nerve fibers in the low compliant cat dental pulp*, Microvasc Res 1994;47: 329.
156. Heyeraas KJ, Kvinnsland I. *Tissue pressure and blood flow in pulpal inflammation*, Proc Finn Dent Soc Suppl 1, 1992;88: 393.
157. Hikiji A, Yamamoto H, Sunakawa M, Suda H. *Increased blood flow and nerve firing in the cat canine tooth in response to stimulation of the second premolar pulp*, Arch Oral Biol 2000;45: 53.
158. Hill R. *NK1 (substance P) receptor antagonists—why are they not analgesic in humans? [see comment]*, Trends Pharmacol Sci 2000;21: 244.
159. Hirvonen T, Närhi M, Hakumäki M. *The excitability of dog pulp nerves in relation to the condition of dentine surface*, J Endod 1984;10: 294.
160. Holland GR. *The extent of the odontoblast process in the cat*, Arch Anat 1976;121: 133.
161. Deleted in page review.
162. Holland GR. *Morphological features of dentine and pulp related to dentine sensitivity*, Arch Oral Biol Suppl,

Telegram: @dental_k

www.konkur.in

1994;39: 3S.

163. Ianiro SR, Jeansonne BG, McNeal SF, et al: *The effect of preoperative acetaminophen or a combination of acetaminophen and ibuprofen on the success of inferior alveolar nerve block for teeth with irreversible pulpitis*, J Endod 2007;33: 11.
164. Ibricevic H, Heyeraas KJ, Pasic Juhas E, et al: *Identification of alpha 2 adrenoceptors in the blood vessels of the dental pulp*, Int Endod J 1991;24: 279.
165. Ikeda H, Tokita Y, Suda H. *Capsaicin-sensitive A fibers in cat tooth pulp*, J Dent Res 1997;76: 1341.
166. Inoue H, Kurosaka Y, Abe K. *Autonomic nerve endings in the odontoblast/predentin border and predentin of the canine teeth of dogs*, J Endod 1992;18: 149.
167. Jacobsen EB, Heyeraas KJ. *Pulp interstitial fluid pressure and blood flow after denervation and electrical tooth stimulation in the ferret*, Arch Oral Biol 1997;42: 407.
168. Janig W, Kollman W. *The involvement of the sympathetic nervous system in pain*, Fortschr Arzneimittelforsch 1984;34: 1066.
169. Jebeles JA, Reilly JS, Gutierrez JF, et al: *Tonsillectomy and adenoidectomy pain reduction by local bupivacaine infiltration in children*, Int J Ped Otorhinolaryngology 1993;25: 149.
170. Jeske NA, Diogenes A, Ruparel NB, et al: *A-kinase anchoring protein mediates TRPV1 thermal hyperalgesia through PKA phosphorylation of TRPV1*, Pain 2008;138: 604.
171. Johnsen D, Johns S. *Quantitation of nerve fibers in the primary and permanent canine and incisor teeth in man*, Arch Oral Biol 1978;23: 825.

Telegram: @dental_k

www.konkur.in

172. Johnsen DC, Harshbarger J, Rymer HD. *Quantitative assessment of neural development in human premolars*, Anat Rec 1983;205: 421.
173. Johnson G, Brännström M. *The sensitivity of dentin: changes in relation to conditions at exposed tubule apertures*, Acta Odontol Scand 1974;32: 29.
174. Jones PA, Taintor JF, Adams AB. *Comparative dental material cytotoxicity measured by depression of rat incisor pulp respiration*, J Endod 1979;5: 48.
175. Jontell M, Okiji T, Dahlgren U, et al: *Immune defense mechanisms of the dental pulp*, Crit Rev Oral Biol Med 1998;9: 179.
176. Juan H, Lembeck F. *Action of peptides and other analgesic agents on paravascular pain receptors of the isolated perfused rabbit ear*, Naunyn Schmiedebergs Arch Pharmacol 1974;283: 151.
177. Katoh Y, Yamaguchi R, Shinkai K, et al: *Clinicopathological study on pulp-irritation of adhesive resinous materials (report 3). Direct capping effects on exposed pulp of Macaca fascicularis*, Jpn J Conserv Dent 1997;40: 163.
178. Kaufman E, Hargreaves KM, Dionne RA. *Comparison of oral triazolam and nitrous oxide with placebo and intravenous diazepam for outpatient premedication*, Oral Surg Oral Med Oral Pathol 1993;75: 156.
179. Kayaoglu G, Orstavik D. *Virulence factors of Enterococcus faecalis: relationship to endodontic disease*, Crit Rev Oral Biol Med 2004;15: 308.
180. Kaye H, Herold RC. *Structure of human dentine. I. Phase contrast, polarization, interference, and bright field microscopic observations on the lateral branch system*, Arch Oral Biol 1966;11: 355.

Telegram: @dental_k

www.konkur.in

181. Kelley KW, Bergenholtz G, Cox CF. *The extent of the odontoblast process in rhesus monkeys (Macaca mulatta) as observed by scanning electron microscopy*, Arch Oral Biol 1981;26: 893.
182. Kerezoudis NP, Funato A, Edwall L, et al: *Activation of sympathetic nerves exerts an inhibitory influence on afferent nerve-induced vasodilation unrelated to vasoconstriction in rat dental pulp*, Acta Physiol Scand 1993;147: 27.
183. Kerezoudis NP, Olgart L, Edwall L. *CGRP(8) reduces the duration but not the maximal increase of antidromic vasodilation in dental pulp and lip of the rat*, Acta Physiol Scand 1994;151: 73.
184. Kerezoudis NP, Olgart L, Edwall L. *Involvement of substance P but not nitric oxide or calcitonin gene-related peptide in neurogenic plasma extravasation in rat incisor pulp and lip*, Arch Oral Biol 1994;39: 769.
185. Khan A, Diogenes A, Jeske N, et al: *Tumor necrosis factor alpha enhances the sensitivity of trigeminal ganglion neurons to capsaicin*, Neuroscience 2008;155: 503.
186. Khan AA, McCreary B, Owatz CB, et al: *The development of a diagnostic instrument for the measurement of mechanical allodynia*, J Endod 2007;33: 663.
187. Khan AA, Owatz CB, Schindler WG, et al: *Measurement of mechanical allodynia and local anesthetic efficacy in patients with irreversible pulpitis and acute periradicular periodontitis*, J Endod 2007;33: 796.
188. Khullar SM, Fristad I, Brodin P, Kvinnsland IH. *Upregulation of growth associated protein 43 expression and neuronal co-expression with neuropeptide Y following*

Telegram: @dental_k

www.konkur.in

inferior alveolar nerve axotomy in the rat, J Peripher Nerv Syst 1998;3: 79.

189. Kim D, Park SH. *Effects of age, sex, and blood pressure on the blood flow velocity in dental pulp measured by Doppler ultrasound technique*, Microcirculation 2016;23: 523.
190. Kim S. *Regulation of pulpal blood flow*, J Dent Res Spec No, 1985;64: 590.
191. Kim S, Dorscher-Kim JE, Liu M. *Microcirculation of the dental pulp and its autonomic control*, Proc Finn Dent Soc 1989;85: 279.
192. Deleted in page review.
193. Kim S, Edwall L, Trowbridge H, Chien S. *Effects of local anesthetics on pulpal blood flow in dogs*, J Dent Res 1984;63: 650.
194. Kim S, Liu M, Simchon S, et al: *Effects of selected inflammatory mediators on blood flow and vascular permeability in the dental pulp*, Proc Finn Dent Soc Suppl 1, 1992;88: 387.
195. Kim S, Schuessler G, Chien S. *Measurement of blood flow in the dental pulp of dogs with the 133xenon washout method*, Arch Oral Biol 1983;28: 501.
196. Kim S, Trowbridge HO, Dorscher-Kim JE. *The influence of 5-hydroxytryptamine (serotonin) on blood flow in the dog pulp*, J Dent Res 1986;65: 682.
197. Kim SK, Ang L, Hsu YY, et al: *Antagonistic effect of D-myo-inositol-1,2,6-trisphosphate (PP56) on neuropeptide Y-induced vasoconstriction in the feline dental pulp*, Arch Oral Biol 1996;41: 791.
198. Kimberly CL, Byers BR. *Inflammation of rat molar pulp and periodontium causes increased calcitonin-gene-related peptide and axonal sprouting*, Anat Rec 1988;222:

Telegram: @dental_k

www.konkur.in

289.
199. Komorowski RC, Torneck CD, Hu JW. *Neurogenic inflammation and tooth pulp innervation pattern in sympathectomized rats*, J Endod 1996;22: 414.
200. Kramer IRH. *The distribution of blood vessels in the human dental pulp*, Finn SB Biology of the dental pulp organ 1968; University of Alabama Press Birmingham 361.
201. Kress M, Sommer C. *Neuroimmunology and pain: peripheral effects of proinflammatory cytokines*, Brune K Handwerker H Hyperalgesia: molecular mechanisms and clinical implications 2003; IASP Press Seattle 57.
202. Kroeger DC, Gonzales F, Krivoy W. *Transmembrane potentials of cultured mouse dental pulp cells*, Proc Soc Exp Biol Med 1961;108: 134.
203. Kumazawa T, Mizumura K. *Thin-fiber receptors responding to mechanical, chemical and thermal stimulation in the skeletal muscle of the dog*, Arch Physiol 1977;273: 179.
204. Kvinnsland IH, Luukko K, Fristad I, et al: *Glial cell line-derived neurotrophic factor (GDNF) from adult rat tooth serves a distinct population of large-sized trigeminal neurons*, Eur J Neurosci 2004;19: 2089.
205. Langeland K, Langeland LK. *Histologic study of 155 impacted teeth*, Odontol Tidskr 1965;73: 527.
206. Langeland K, Langeland LK. *Pulp reactions to cavity and crown preparations*, Aust Dent J 1970;15: 261.
207. Lantelme RL, Handleman SL, Herbison RJ. *Dentin formation in periodontally diseased teeth*, J Dent Res 1976;55: 48.
208. Latremoliere A, Woolf CJ. *Central sensitization: a generator of pain hypersensitivity by central neural*

Telegram: @dental_k

www.konkur.in

plasticity, J Pain 2009;10: 895.
209. Laurent TC, et al: *The catabolic fate of hyaluronic acid*, Connect Tissue Res 1986;15: 33.
210. Lechner JH, Kalnitsky G. *The presence of large amounts of type III collagen in bovine dental pulp and its significance with regard to the mechanism of dentinogenesis*, Arch Oral Biol 1981;26: 265.
211. Lepinski AM, Hargreaves KM, Goodis HE, et al: *Bradykinin levels in dental pulp by microdialysis*, J Endod 2000;26: 744.
212. Lesot H, Osman M, Ruch JV. *Immunofluorescent localization of collagens, fibronectin and laminin during terminal differentiation of odontoblasts*, Dev Biol 1981;82: 371.
213. Levi-Montalcini R. *The nerve growth factor: its mode of action on sensory and sympathetic nerve cells*, Harvey Lect 1966;60: 217.
214. Levine J, Moskowitz M, Basbaum A. *The contribution of neurogenic inflammation in experimental arthritis*, J Immunol 1985;135: 843.
215. Levine J, Taiwo Y. *Inflammatory pain*, Wall P Melzack R Textbook of pain 1994; Churchill Livingstone Edinburgh.
216. Levine JD, Gordon NC, Fields HL. *The mechanism of placebo analgesia*, Lancet 1978;2: 654.
217. Lewin G, Rueff A, Mendell L. *Peripheral and central mechanisms of NGF-induced hyperalgesia*, Eur J Neurosci 1994;6: 1903.
218. Light A. *Karger*, The initial processing of pain and its descending control: spinal and trigeminal systems 1992; Basel.
219. Lilja J. *Innervation of different parts of the predentin and*

Telegram: @dental_k

www.konkur.in

dentin in a young human premolar, Acta Odontol Scand 1979;37: 339.

220. Lilja J, Noredenvall K-J, Brännström M. *Dentin sensitivity, odontoblasts and nerves under desiccated or infected experimental cavities*, Swed Dent J 1982;6: 93.
221. Lilja JJ, Kivisto KT, Backman JT, Neuvonen PJ. *Effect of grapefruit juice dose on grapefruit juice-triazolam interaction: repeated consumption prolongs triazolam half-life*, Eur J Clin Pharmacol 2000;56: 411.
222. Lindahl O. *Pain-a chemical explanation*, Acta Rheumatol Scand 1962;8: 161.
223. Deleted in page review.
224. Linde A. *The extracellular matrix of the dental pulp and dentin*, J Dent Res special issue, 1985;64: 523.
225. Liu L, Simon SA. *Capsaicin, acid and heat-evoked currents in rat trigeminal ganglion neurons: relationship to functional VR1 receptors*, Physiol Behav 2000;69: 363.
226. Lohinai Z, Balla I, Marczis J, et al: *Evidence for the role of nitric oxide in the circulation of the dental pulp*, J Dent Res 1995;74: 1501.
227. Lohinai Z, Szekely AD, Benedek P, Csillag A. *Nitric oxide synthetase containing nerves in the cat and dog dental pulps and gingiva*, Neurosci Lett 1997;227: 91.
228. Lohrberg M, Wilting J. *The lymphatic vascular system of the mouse head*, Cell Tissue Res 2016;366: 667.
229. Lundberg JM, nggård A, Fahrenkrug J, et al: *Vasoactive intestinal polypeptide in cholinergic neurons of exocrine glands: functional significance of coexisting transmitters for vasodilation and secretion*, Proc Natl Acad Sci U S A 1980;77: 1651.
230. Lundberg JM, Fried G, Fahrenkrug J, et al: *Subcellular fractionation of cat submandibular gland: comparative*

Telegram: @dental_k

www.konkur.in

studies on the distribution of acetylcholine and vasoactive intestinal polypeptide (VIP), Neuroscience 1981;6: 1001.

231. Lundgren T, Nannmark U, Linde A. *Calcium ion activity and pH in the odontoblast-predentin region: ion-selective microelectrode measurements*, Calcif Tissue Int 1992;50: 134.
232. Luo A H, Wang C, Liu M, Yin B, A, Huang D, et al: *Inhibition of SOX9 promotes inflammatory and immune responses of dental pulp*, J Endod 2018;44: 792.
233. Luthman J, Luthman D, Hökfelt T. *Occurrence and distribution of different neurochemical markers in the human dental pulp*, Arch Oral Biol 1992;37: 193.
234. Madison S, Whitsel EA, Suarez-Roca H, Maixner W. *Sensitizing effects of leukotriene B4 on intradental primary afferents*, Pain 1992;49: 99.
235. Maeda T, Honma S, Takano Y. *Dense innervation of radicular human dental pulp as revealed by immunocytochemistry for protein gene-product 9.5*, Arch Oral Biol 1994;39: 563.
236. Magloire H, Couble ML, Thivichon-Prince B, et al: *Odontoblast: a mechano-sensory cell*, J Exp Zool (Mol Dev Evol) 2009;312B: 416.
237. Magloire H, Maurin JC, Couble ML, et al: *Topical review. Dental pain and odontoblasts: facts and hypotheses*, J Orofacial Pain 2010;24: 335.
238. Maixner W, Dubner R, Kenshalo DR Jr, et al: *Responses of monkey medullary dorsal horn neurons during the detection of noxious heat stimuli*, J Neurophysiol 1989;62: 437.
239. Malmberg AB, Yaksh TL. *Spinal nitric oxide synthesis inhibition blocks NMDA-induced thermal hyperalgesia and produces antinociception in the formalin test in rats*,

Telegram: @dental_k

www.konkur.in

Pain 1993;54: 291.

240. Malmberg AB, Yaksh TL. *Cyclooxygenase inhibition and the spinal release of prostaglandin E2 and amino acids evoked by paw formalin injection: a microdialysis study in unanesthetized rats*, J Neurosci 1995;15: 2768.
241. Maltos KL, Menezes GB, Caliari MV, et al: *Vascular and cellular responses to pro-inflammatory stimuli in rat dental pulp*, Arch Oral Biol 2004;49: 443.
242. Mangkornkarn C, Steiner JC. *In vivo and in vitro glycosaminoglycans from human dental pulp*, J Endod 1992;18: 327.
243. Marbach JJ, Raphael KG. *Phantom tooth pain: a new look at an old dilemma*, Pain Med 2000;1: 68.
244. Marfurt CF, Zaleski EM, Adams CE, Welther CL. *Sympathetic nerve fibers in rat orofacial and cerebral tissues as revealed by the HRP-WGA tracing technique: a light and electron microscopic study*, Brain Res 1986;366: 373.
245. Marion D, Jean A, Hamel H, et al: *Scanning electron microscopic study of odontoblasts and circumferential dentin in a human tooth*, Oral Surg Oral Med Oral Pathol 1991;72: 473.
246. Marriott D, Wilkin GP, Coote PR, et al: *Eicosanoid synthesis by spinal cord astrocytes is evoked by substance P; possible implications for nociception and pain*, Adv Prostaglandin Thromboxane Leukot Res 1991;21B: 739.
247. Martin A, Gasse H, Staszyk C. *Absence of lymphatic vessels in the dog dental pulp: an immunohistochemical study*, J Anat 2010;217: 609.
248. Martin H, Basbaum AI, Kwiat GC, et al: *Leukotriene and prostaglandin sensitization of cutaneous high-threshold C- and A-delta mechanoreceptors in the hairy skin of rat*

Telegram: @dental_k

www.konkur.in

hindlimbs, Neuroscience 1987;22: 651.

249. Matsuo S, Ichikawa H, Henderson TA, et al: *trkA modulation of developing somatosensory neurons in oro-facial tissues: tooth pulp fibers are absent in trkA knockout mice*, Neuroscience 2001;105: 747.
250. Matthews B, Andrew D. *Microvascular architecture and exchange in teeth*, Microcirculation 1995;2: 305.
251. Matthews B, Vongsavan N. *Interactions between neural and hydrodynamic mechanisms in dentine and pulp*, Arch Oral Biol Suppl 1, 1994;39: 87S.
252. McGrath PA, Gracely RH, Dubner R, Heft MW. *Non-pain and pain sensations evoked by tooth pulp stimulation*, Pain 1983;15: 377.
253. Meller ST, Dykstra C, Gebhart GF. *Production of endogenous nitric oxide and activation of soluble guanylate cyclase are required for N-methyl-D-aspartate-produced facilitation of the nociceptive tail-flick reflex*, Eur J Pharmacol 1992;214: 93.
254. Meyer MW, Path MG. *Blood flow in the dental pulp of dogs determined by hydrogen polarography and radioactive microsphere methods*, Arch Oral Biol 1979;24: 601.
255. Meyer R, Campbell J. *Myelinated nociceptive afferents account for the hyperalgesia that follows a burn to the hand*, Science 1981;213: 1527.
256. Mirza AF, Mo J, Holt JL, et al: *Is there a relationship between throbbing pain and arterial pulsations?*, J Neurosci 2012;32: 7572.
257. Mitsiadis TA, De Bari C, About I. *Apoptosis in developmental and repair-related human tooth remodeling: a view from the inside*, Exp Cell Res 2008;314: 869.

Telegram: @dental_k

www.konkur.in

258. Mjör IA, Nordahl I. *The density and branching of dentinal tubules in human teeth,* Arch Oral Biol 1996;41: 401.
259. Modaresi J, Dianat O, Mozayeni MA. *The efficacy comparison of ibuprofen, acetaminophen-codeine, and placebo premedication therapy on the depth of anesthesia during treatment of inflamed teeth*, Oral Surg Oral Med Oral Pathol 2006;102: 399.
260. Mullaney TP, Howell RM, Petrich JD. *Resistance of nerve fibers to pulpal necrosis*, Oral Surg 1970;30: 690.
261. Murray PE, About I, Lumley PJ, et al: *Human odontoblast cell numbers after dental injury*, J Dent 2000;28: 277.
262. Murray PE, Hafez AA, Windsor LJ, et al: *Comparison of pulp responses following restoration of exposed and non-exposed cavities*, J Dent 2002;30: 213.
263. Murray PE, Lumley PJ, Ross HF, Smith AJ. *Tooth slice organ culture for cytotoxicity assessment of dental materials*, Biomaterials 2000;21: 1711.
264. Naftel JP, et al: *Course and composition of the nerves that supply the mandibular teeth of the rat*, Anat Rec 1999;256: 433.
265. Nagaoka S, Miyazaki Y, Liu HJ, et al: *Bacterial invasion into dentinal tubules of human vital and nonvital teeth*, J Endod 1995;21: 70.
266. Nair PN, et al: *Histological, ultrastructural and quantitative investigations on the response of healthy human pulps to experimental capping with mineral trioxide aggregate: a randomized controlled trial*, Int Endod J 2008;41: 128.
267. Nakanishi T, Shimizu H, Hosokawa Y, et al: *An immunohistological study on cyclooxygenase-2 in human*

Telegram: @dental_k

www.konkur.in

dental pulp, J Endod 2001;27: 385.

268. Närhi M. *Activation of dental pulp nerves of the cat and the dog with hydrostatic pressure*, Proc Finn Dent Soc Suppl 5, 1978;74: 1.
269. Narhi M. *The characteristics of intradental sensory units and their responses to stimulation*, J Dent Res 1985;64: 564.
270. Narhi M, Jyväsjärvi E, Virtanen A, et al: *Role of intradental A and C type nerve fibers in dental pain mechanisms*, Proc Finn Dent Soc 1992;88: 507.
271. Närhi M, Jyväsjärvi E, Hirronen T. *Activation of heat-sensitive nerve fibers in the dental pulp of the cat*, Pain 1982;14: 317.
272. Närhi M, Virtanen A, Kuhta J, Huopaniemi T. *Electrical stimulation of teeth with a pulp tester in the cat*, Scand J Dent Res 1979;87: 32.
273. Närhi M, Yamamoto H, Ngassapa D. *Function of intradental nociceptors in normal and inflamed teeth*, Shimono M Maeda T Suda H. Dentin/pulp complex 1996; Quintessence Publishing Tokyo 136.
274. Närhi M, Yamamoto H, Ngassapa D, Hirvonen T. *The neurophysiological basis and the role of inflammatory reactions in dentine hypersensitivity*, Arch Oral Biol Suppl, 1994;39: 23S.
275. Nassar M, Stirling L, Forlani G, et al: *Nociceptor-specific gene deletion reveals a major role for NAv 1.7 (PN1) in acute and inflammatory pain*, Proc Natl Acad Sci U S A 2004;101: 12706.
276. Nawroth PP, Stern DM. *Modulation of endothelial cell hemostatic properties by tumor necrosis factor*, J Exp Med 1986;163: 740.
277. Neumann S, Doubell TP, Leslie T, et al: *Inflammatory*

Telegram: @dental_k

www.konkur.in

pain hypersensitivity mediated by phenotype switch in myelinated primary sensory neurons, Nature 1996;384: 360.

278. Ngassapa D, Närhi M, Hirvonen T. *The effect of serotonin (5-HT) and calcitonin gene-related peptide (CGRP) on the function of intradental nerves in the dog*, Proc Finn Dent Soc Suppl 1, 1992;88: 143.
279. Nicol GD, Vasko MR. *Unraveling the story of NGF-mediated sensitization of nociceptive sensory neurons: ON or OFF the Trks?*, Mol Interv 2007;7: 26.
280. Nishioka M, Shiiya T, Ueno K, et al: *Tooth replantation in germ-free and conventional rats*, Endod Dent Traumatol 1998;14: 163.
281. Nitzan DW, Michaeli Y, Weinreb M, et al: *The effect of aging on tooth morphology: a study on impacted teeth*, Oral Surg Oral Med Oral Pathol 1986;61: 54.
282. Nuti N, Corallo C, Chan BM, et al: *Multipotent differentiation of human dental pulp stem cells: a literature review*, Stem Cell Rev 2016;12: 511.
283. O'Neil RG, Brown RC. *The vanilloid receptor family of calcium-permeable channels: molecular integrators of microenvironmental stimuli*, News Physiol Sci 2003;18: 226.
284. Ochoa JL, Torebjork E, Marchettini P, Sivak M. *Mechanism of neuropathic pain: cumulative observations, new experiments, and further speculation*, Fields HL Dubner R Cervero F Advances in pain research and therapy 1985; Raven Press New York 431.
285. Okeson JP. *Assessment of orofacial pain disorders*, Okeson JP Orofacial pain: guidelines for assessment, diagnosis, and management 1996; Quintessence Publishing Chicago 35.

Telegram: @dental_k

www.konkur.in

286. Okiji T, Kawashima N, Kosaka T, et al: *An immunohistochemical study of the distribution of immunocompetent cells, especially macrophages and Ia antigen-presenting cells of heterogeneous populations, in normal rat molar pulp*, J Dent Res 1992;71: 1196.
287. Okiji T, Morita I, Sunada I, et al: *Involvement of arachidonic acid metabolites in increases in vascular permeability in experimental dental pulpal inflammation in the rat*, Arch Oral Biol 1989;34: 523.
288. Olgart L, Kerezoudis NP. *Nerve-pulp interactions*, Arch Oral Biol Suppl, 1994;39: 47S.
289. Olgart LM, Edwall L, Gazelius B. *Neurogenic mediators in control of pulpal blood flow*, J Endod 1989;15: 409.
290. Olgart LM, Edwall L, Gazelius B. *Involvement of afferent nerves in pulpal blood-flow reactions in response to clinical and experimental procedures in the cat*, Arch Oral Biol 1991;36: 575.
291. Olgart LM, Gazelius B, Brodin E, Nilsson G. *Release of substance P-like immunoreactivity from the dental pulp*, Acta Physiol Scand 1977;101: 510.
292. Orchardson R, Cadden SW. *An update on the physiology of the dentine-pulp complex*, Dent Update 2001;28: 200-208.
293. Orchardson R, Gillam DG. *Managing dentin hypersensitivity*, J Am Dent Assoc 2006;137: 990- quiz 1028.
294. Owatz CB, Khan AA, Schindler WG, et al: *The incidence of mechanical allodynia in patients with irreversible pulpitis*, J Endod 2007;33: 552.
295. Oxlund H, Manschot J, Viidik A. *The role of elastin in the mechanical properties of skin*, J Biomech 1988;21: 213.

Telegram: @dental_k

www.konkur.in

296. Paine MF, Widmer WW, Hart HL, et al: *A furanocoumarin-free grapefruit juice establishes furanocoumarins as the mediators of the grapefruit juice-felodipine interaction [erratum appears in Am J Clin Nutr 84:264, 2006]*, Am J Clin Nutr 2006;83: 1097.
297. Pashley DH. *Dynamics of the pulpodentin complex*, Crit Rev Oral Biol Med 1996;7: 104.
298. Pashley DH. *Potential treatment modalities for dentin hypersensitivity—in office products*, Addy M Orchardson R Tooth wear and sensitivity 2000; Martin-Dunitz Publishers London 351.
299. Patwardhan A, Cerka K, Vela J, et al: *Trigeminal nociceptors express prostaglandin receptor subtypes EP2 and EP3*, J Dent Res 2008;87: 262.
300. Penfield W, Rasmussen G. The cerebral cortex of man 1950; Macmillan New York.
301. Perl E. *Causalgia, pathological pain and adrenergic receptors*, Proc Natl Acad Sci U S A 1999;96: 7664.
302. Perl ER. *Ideas about pain, a historical view [review]*, Nat Rev Neurosci 2007;8: 71.
303. Petty B, Cornblath DR, Adornato BT, et al: *The effect of systemically administered recombinant human nerve growth factor in healthy human subjects*, Ann Neurol 1994;36: 244.
304. Poggi P, Casasco A, Marchetti C, et al: *Ultrastructural localization of elastin-like immunoreactivity in the extracellular matrix around human small lymphatic vessels*, Lymphology 1995;28: 189.
305. Pohto P. *Sympathetic adrenergic innervation of permanent teeth in the monkey (Macaca irus)*, Acta Odontol Scand 1972;30: 117.
306. Pohto P, Antila R. *Demonstration of adrenergic nerve*

Telegram: @dental_k

www.konkur.in

fibres in human dental pulp by histochemical fluorescence method, Acta Odontol Scand 1968;26: 137.

307. Pohto P, Antila R. *Innervation of blood vessels in the dental pulp*, Int Dent J 1972;22: 228.
308. Pomonis JD, Rogers SD, Peters CM, et al: *Expression and localization of endothelin receptors: implications for the involvement of peripheral glia in nociception*, J Neurosci 2001;21: 999.
309. Prati C, Cervellati F, Sanasi V, Montebugnoli L. *Treatment of cervical dentin hypersensitivity with resin adhesives: 4 week evaluation*, Arch Dent 2001;14: 378.
310. Prescott RS, Alsanea R, Fayad MI, et al: *In vivo generation of dental pulp-like tissue by using dental pulp stem cells, a collagen scaffold, and dentin matrix protein 1 after subcutaneous transplantation in mice*, J Endod 2008;34: 421.
311. Qian XB, Naftel JP. *Effects of neonatal exposure to anti-nerve growth factor on the number and size distribution of trigeminal neurones projecting to the molar dental pulp in rats*, Arch Oral Biol 1996;41: 359.
312. Rapp R, el-Labban NG, Kramer IR, et al: *Ultrastructure of fenestrated capillaries in human dental pulps*, Arch Oral Biol 1977;22: 317.
313. Reader A, Foreman DW. *An ultrastructural qualitative investigation of human intradental innervation*, J Endod 1981;7: 493.
314. Renton T, Yiangou Y, Plumpton C, et al: *Sodium channel Nav1.8 immunoreactivity in painful human dental pulp*, BMC Oral Health 2005;5: 5.
315. Roberts-Clark D, Smith AJ. *Angiogenic growth factors in human dentine matrix*, Arch Oral Biol 2000;42: 1013.
316. Rodd HD, Boissonade FM. *Innervation of human tooth*

Telegram: @dental_k

www.konkur.in

pulp in relation to caries and dentition type, J Dent Res 2001;80: 389.

317. Rodd HD, Boissonade FM. *Comparative immunohistochemical analysis of the peptidergic innervation of human primary and permanent tooth pulp*, Arch Oral Biol 2002;47: 375.
318. Rodd HD, Boissonade FM, Day PF. *Pulpal status of hypomineralized permanent molars*, Pediatr Dent 2007;29: 514.
319. Roy ML, Narahashi T. *Differential properties of tetrodotoxin-sensitive and tetrodotoxin-resistant sodium channels in rat dorsal root ganglion neurons*, J Neurosci 1992;12: 2104.
320. Ruparel NB, Patwardhan AM, Akopian A, et al: *Homologous and heterologous desensitization of capsaicin and mustard oil responses utilize different cellular pathways in nociceptors*, Pain 2008;135: 271.
321. Rutherford B. *BMP-7 gene transfer into inflamed ferret-dental pulps*, Eur J Oral Sci 2001;109: 422.
322. Rutherford B, Fitzgerald M. *A new biological approach to vital pulp therapy*, Crit Rev Oral Biol Med 1995;6: 218.
323. Rutherford RB, Spanberg L, Tucker M, et al: *The time-course of the induction of reparative dentine formation in moneys by recombinant human osteogenic protein-1*, Arch Oral Biol 1994;39: 833.
324. Sakamoto N, Nakajima T, Ikunaga K, et al: *Identification of hyaluronidase activity in rabbit dental pulp*, J Dent Res 1981;60: 850.
325. Sakurai K, Okiji T, Suda H. *Co-increase of nerve fibers and HLA-DR- and/or factor XIIIa-expressing dendritic cells in dentinal caries-affected regions of the human dental pulp: an immunohistochemical study*, J Dent Res

Telegram: @dental_k

www.konkur.in

1999;78: 1596.
326. Sasaki S. *Studies on the respiration of the dog tooth germ*, J Biochem (Tokyo) 1959;46: 269.
327. Sasano T, Kuriwada S, Sanjo D. *Arterial blood pressure regulation of pulpal blood flow as determined by laser Doppler*, J Dent Res 1989;68: 791.
328. Sasano T, Kuriwada S, Shoji N, et al: *Axon reflex vasodilatation in cat dental pulp elicited by noxious stimulation of the gingiva*, J Dent Res 1994;73: 1797.
329. Sasano T, Shoji N, Kuriwada S, et al: *Absence of parasympathetic vasodilatation in cat dental pulp*, J Dent Res 1995;74: 1665.
330. Schaible H, Schmidt R. *Discharge characteristics of receptors with fine afferents from normal and inflamed joints: influence of analgesics and prostaglandins*, Agents Actions 1986;19: 99.
331. Schüpbach P, Lutz F, Finger WT. *Closing of dentin tubules by Gluma desensitizer*, Eur J Oral Sci 1997;105: 414.
332. Scott JN, Weber DF. *Microscopy of the junctional region between human coronal primary and secondary dentin*, J Morphol 1977;154: 133.
333. Seltzer S, Bender IB, Ziontz M. *The interrelationship of pulp and periodontal disease*, Oral Surg 1963;16: 1474.
334. Senger DR, Galli SJ, Dvorak AM, et al: *Tumor cells secrete a vascular permeability factor that promotes accumulation of ascites fluid*, Science 1983;219: 983.
335. Sessle BJ. *Recent developments in pain research: central mechanisms of orofacial pain and its control*, J Endodon 1986;12: 435.
336. Sessle BJ. *The neurobiology of facial and dental pain: present knowledge, future directions*, J Dent Res 1987;66:

Telegram: @dental_k

www.konkur.in

962.

337. Sessle BJ. *Acute and chronic craniofacial pain: brainstem mechanisms of nociceptive transmission and neuroplasticity, and their clinical correlates*, Crit Rev Oral Biol Med 2000;11: 57.

338. Sessle BJ, Greenwood LF. *Inputs to trigeminal brain stem neurones from facial, oral, tooth pulp and pharyngolaryngeal tissues: I. Responses to innocuous and noxious stimuli*, Brain Res 1976;117: 211.

339. Sessle BJ, Hu JW, Amano N, et al: *Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurones in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain*, Pain 1986;27: 219.

340. Shortland PJ, Jacquin MF, De Maro JA, et al: *Central projections of identified trigeminal primary afferents after molar pulp differentiation in adult rats*, Somatosens Mot Res 1995;12: 227.

341. Shulman K, Rosen S, Tognazzi K, et al: *Expression of vascular permeability factor (VPF/VEGF) is altered in many glomerular diseases*, J Am Soc Nephrol 1996;7: 661.

342. Shuttleworth CA, Ward JL, Hirschmann PN. *The presence of type III collagen in the developing tooth*, Biochim Biophys Acta 1978;535: 348.

343. Sigal MJ, Pitaru S, Aubin JE, et al: *A combined scanning electron microscopy and immunofluorescence study demonstrating that the odontoblast process extends to the dentinoenamel junction in human teeth*, Anat Rec 1984;210: 453.

344. Smith AJ, Garde C, Cassidy N, et al: *Solubilization of dentin extracellular matrix by calcium hydroxide*, J Dent

Telegram: @dental_k

www.konkur.in

Res 1995;74: 829- (abstract).
345. Smith AJ, Sloan AJ, Matthews JB, et al: *Reparative processes in dentine and pulp*, Addy M Embery G Edger WM. Tooth wear and sensitivity: clinical advances in restorative dentistry 2000; Martin Dunitz London.
346. Smith AJ, Tobias RS, Cassidy N, et al: *Odontoblast stimulation in ferrets by dentine matrix components*, Arch Oral Biol 1994;39: 13.
347. Solheim T. *Dental root translucency as an indicator of age*, Scand J Dent Res 1989;97: 189.
348. Souza PP, Fukada SY, Cunha FQ, et al: *Regulation of angiotensin II receptors levels during rat induced pulpitis*, Regul Pept 2007;140: 27.
349. Stanley HR, White CL, McCray L. *The rate of tertiary (reparative) dentin formation in the human tooth*, Oral Surg 1966;21: 180.
350. Steen KH, Reeh PW, Anton F, Handwerker HO. *Protons selectively induce lasting excitation and sensitization to mechanical stimulation of nociceptors in rat skin in vitro*, J Neurosci 1992;21: 86.
351. Stenvik A, Iverson J, Mjör IA. *Tissue pressure and histology of normal and inflamed tooth pulps in Macaque monkeys*, Arch Oral Biol 1972;17: 1501.
352. Stern D, Nawroth P, Handley D, et al: *An endothelial cell-dependent pathway of coagulation*, Proc Natl Acad Sci U S A 1985;82: 2523.
353. Sunakawa M, Tokita Y, Suda H. *Pulsed Nd:YAG laser irradiation of the tooth pulp in the cat. II. Effect of scanning lasing*, Lasers Surg Med 2000;26: 477.
354. Sundqvist G, Lerner UH. *Bradykinin and thrombin synergistically potentiate interleukin 1 and tumour necrosis factor induced prostanoid biosynthesis in human*

Telegram: @dental_k

www.konkur.in

dental pulp fibroblasts, Cytokine 1996;8: 168.

355. Sweitzer SM, Schubert P, DeLeo JA. *Propentofylline, a glial modulating agent, exhibits antiallodynic properties in a rat model of neuropathic pain*, J Pharmacol Exp Ther 2001;297: 1210.
356. Swift ML, Byers MR. *Effects of aging responses of nerve fibers to pulpal inflammation in rat molars analyzed by quantitative immunohistochemistry*, Arch Oral Biol 1992;37: 901.
357. Tagami J, Hosoda H, Burrow MF, Nakajima M. *Effect of aging and caries on dentin permeability*, Proc Finn Dent Soc Suppl 1, 1992;88: 149.
358. Takahashi K, Kishi Y, Kim S. *A scanning electron microscope study of the blood vessels of dog pulp using corrosion resin casts*, J Endod 1982;8: 131.
359. Tanaka T. *The origin and localization of dentinal fluid in developing rat molar teeth studied with lanthanum as a tracer*, Arch Oral Biol 1980;25: 153.
360. Telles PD, Hanks CT, Machado MA, Nör JE. *Lipoteichoic acid up-regulates VEGF expression in macrophages and pulp cells*, J Dent Res 2003;82: 466.
361. Thomas HF. *The extent of the odontoblast process in human dentin*, J Dent Res 1979;58(D): 2207.
362. Thomas HF, Payne RC. *The ultrastructure of dentinal tubules from erupted human premolar teeth*, J Dent Res 1983;62: 532.
363. Tokita Y, Sunakawa M, Suda H. *Pulsed ND: YAG laser irradiation of the tooth pulp in the cat. I. Effect of spot lasing*, Lasers Surg Med 2000;26: 477.
364. Tominaga M, Numazaki M, Iida T, et al: Molecular mechanisms of TRPV1-mediated thermal hypersensitivity (vol 30) 2003; IASP Press Seattle 37.

Telegram: @dental_k

www.konkur.in

365. Tönder KH, Naess G. *Nervous control of blood flow in the dental pulp in dogs*, Acta Physiol Scand 1978;104: 13.
366. Tönder KJ. *Effect of vasodilating drugs on external carotid and pulpal blood flow in dogs: "stealing" of dental perfusion pressure*, Acta Physiol Scand 1976;97: 75.
367. Tönder KJH, Kvininsland I. *Micropuncture measurements of interstitial fluid pressure in normal and inflamed dental pulp in cats*, J Endod 1983;9: 105.
368. Tonioli M, Patel T, Diogenes A, et al: *Effect of neurotrophic factors on bradykinin expression in rat trigeminal sensory neurons determined by real-time polymerase chain reaction*, J Endod 2004;30: 263.
369. Torabinejad M, Cymerman JJ, Frankson M, et al: *Effectiveness of various medications on postoperative pain following complete instrumentation*, J Endod 1994;20: 345.
370. Torebjörk HE, Hanin RG. *Perceptual changes accompanying controlled preferential blocking of A and C fiber responses in intact human skin nerves*, Exp Brain Res 1973;16: 321.
371. Torneck CD. *Dentin-pulp complex*, Ten Cate AR Oral histology: development, structure, and function ed 5 1998; Mosby St. Louis 150.
372. Torneck CD, Kwan CL, Hu JW. *Inflammatory lesions of the tooth pulp induce changes in brainstem neurons of the rat trigeminal subnucleus oralis*, J Dent Res 1996;75: 553.
373. Tran XV, Gorin C, Willig C, et al: *Effect of a calcium-silicate-based restorative cement on pulp repair*, J Dent Res 2012;91: 1166.
374. Trantor IR, Messer HH, Birner R. *The effects of neuropeptides (calcitonin-gene-related peptide and*

Telegram: @dental_k

www.konkur.in

substance P) on cultured human pulpal cells, J Dent Res 1995;74: 1066.

375. Trowbridge HO. *Pathogenesis of pulpitis resulting from dental caries*, J Endod 1981;7: 52.
376. Trowbridge HO. *Review of dental pain–histology and physiology*, J Endod 1986;12: 445.
377. Trowbridge HO, Franks M, Korostoff E, et al: *Sensory response to thermal stimulation in human teeth*, J Endod 1980;6: 405.
378. Trowbridge HO, Silver DR. *Review of current approaches to in-office management of tooth hypersensitivity*, Dent Clin North Am 1990;16: 561.
379. Trowbridge HO, Stewart JCB, Shapiro IM. *Assessment of indurated, diffusely calcified human dental pulps*, Proceedings of the International Conference on Dentin/Pulp Complex 1996; Quintessence Publishing Tokyo 297.
380. Turner D, Marfurt C, Sattelburg C. *Demonstration of physiological barrier between pulpal odontoblasts and its perturbation following routine restorative procedures: a horseradish peroxidase tracing study in the rat*, J Dent Res 1989;68: 1262.
381. Turner DF. *Immediate physiological response of odontoblasts*, Proc Finn Dent Soc Suppl 1, 1992;88: 55.
382. Uddman R, et al: *Occurrence of VIP nerves in mammalian dental pulps*, Acta Odontol Scand 1980;38: 325.
383. van Amerongen JP, Lemmens IG, Tonino GJ. *The concentration, extractability and characterization of collagen in human dental pulp*, Arch Oral Biol 1983;28: 339.
384. Van Hassel HJ. *Physiology of the human dental pulp*,

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Oral Surg Oral Med Oral Path 1971;32: 126.
385. Van Hassel HJ, Brown AC. *Effect of temperature changes on intrapulpal pressure and hydraulic permeability in dogs*, Arch Oral Biol 1969;14: 301.
386. van Wijk AJ, Hoogstraten J. *Reducing fear of pain associated with endodontic therapy*, Int Endod J 2006;39: 384.
387. Veerayutthwilai O, Byers MR, Pham TT, et al: *Differential regulation of immune responses by odontoblasts*, Oral Microbiol Immunol 2007;22: 5.
388. Vertucci F. *Root canal morphology and its relationship to endodontic procedures*, Endod Top 2005;10: 3.
389. Vickers ER, Cousins MJ. *Neuropathic orofacial pain part 1: prevalence and pathophysiology*, Aust Endod J 2000;26: 19.
390. Virtej A, Loes S, Iden O, Bletsa A, Berggreen E. *Vascular endothelial growth factors signalling in normal human dental pulp: a study of gene and protein expression*, Eur J Oral Sci 2013;121: 92-100.
391. Vongsavan N, Matthews B. *The permeability of cat dentine in vivo and in vitro*, Arch Oral Biol 1991;36: 641.
392. Vongsavan N, Matthews B. *Fluid flow through cat dentine in vivo*, Arch Oral Biol 1992;37: 175.
393. Vongsavan N, Matthews B. *The vascularity of dental pulp in cats*, J Dent Res, 1992;71: 1913.
394. Vongsavan N, Matthews B. *The relation between fluid flow through dentine and the discharge of intradental nerves*, Arch Oral Biol Suppl, 1994;39: 140S.
395. Vongsavan N, Matthews B. *The relationship between the discharge of intradental nerves and the rate of fluid flow through dentine in the cat*, Arch Oral Biol 2007;52: 640.
396. Wakisaka S. *Neuropeptides in the dental pulp: their*

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www.konkur.in

distribution, origins and correlation, J Endod 1990;16: 67.

397. Wakisaka S, Ichikawa H, Akai M. *Distribution and origins of peptide- and catecholamine-containing nerve fibres in the feline dental pulp and effects of cavity preparation on these nerve fibres*, J Osaka Univ Dent Sch 1986;26: 17.
398. Wakisaka S, Sasaki Y, Ichikawa H, Matsuo S. *Increase in c-fos-like immunoreactivity in the trigeminal nucleus complex after dental treatment*, Proc Finn Dent Soc Suppl 1, 1992;88: 551.
399. Wall PD. *Alterations in the central nervous system after deafferentation: connectivity control*, Bonica JJ Lindblom U Iggo A Advances in pain research and therapy 1983;vol 5: Raven Press New York 677.
400. Walton R, Fouad A. *Endodontic interappointment flare-ups: a prospective study of incidence and related factors*, J Endod 1992;18: 172.
401. Wang C, Li GW, Huang LY. *Prostaglandin E2 potentiation of P2X3 receptor mediated currents in dorsal root ganglion neurons*, Mol Pain 2007;3: 22.
402. Warfvinge J, Dahlen G, Bergenholtz G. *Dental pulp response to bacterial cell wall material*, J Dent Res 1985;64: 1046.
403. Warren C, Mok L, Gordon S, et al: *Quantification of neural protein in extirpated tooth pulp*, J Endod 2008;34: 7.
404. Watkins LR, Milligan ED, Maier SF. *Glial activation: a driving force for pathological pain*, Trends Neurosci 2001;24: 450.
405. Weber DK, Zaki AL. *Scanning and transmission electron microscopy of tubular structure presumed to be human odontoblast processes*, J Dent Res 1986;65: 982.

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www.konkur.in

406. Weiger R, Axmann-Kremar D, Lost C. *Prognosis of conventional root canal treatment reconsidered*, Endodon Dent Traumatol 1998;14: 1.
407. Weinstock A, Weinstock M, Leblond CP. *Autoradiographic detection of 3H-fucose incorporation into glycoprotein by odontoblasts and its deposition at the site of the calcification front in dentin*, Calcif Tissue Res 1972;8: 181.
408. Weinstock M, Leblond CP. *Synthesis, migration and release of precursor collagen by odontoblasts as visualized by radioautography after 3H-proline administration*, J Cell Biol 1974;60: 92.
409. Wells J, Bingham V, Rowland K, Hatton J. *Expression of Na v 1.9 channels in human dental pulp and trigeminal ganglion*, J Endod 2007;33: 1172.
410. Wiig H, Aukland K, Tenstad O. *Isolation of interstitial fluid from rat mammary tumors by a centrifugation method*, Arch Physiol Heart Circ Physiol 2003;284: H416-H424.
411. Wiig H, Gyenge C, Iversen PO, et al: *The role of the extracellular matrix in tissue distribution of macromolecules in normal and pathological tissues: potential therapeutic consequences*, Microcirculation 2008;15: 283.
412. Willingale HL, Gardiner NJ, McLymont N, et al: *Prostanoids synthesized by cyclo-oxygenase isoforms in rat spinal cord and their contribution to the development of neuronal hyperexcitability*, Br J Pharmacol 1997;122: 1593.
413. Winter HF, Bishop JG, Dorman HL. *Transmembrane potentials of odontoblasts*, J Dent Res 1963;42: 594.
414. Wong M, Lytle WR. *A comparison of anxiety levels*

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www.konkur.in

associated with root canal therapy and oral surgery treatment, J Endod 1991;17: 461.

415. Woodnutt DA, Wager-Miller J, O'Neill PC, et al: *Neurotrophin receptors and nerve growth factor are differentially expressed in adjacent nonneuronal cells of normal and injured tooth pulp*, Cell Tissue Res 2000;299: 225.
416. Woolf C. *Transcriptional and posttranslational plasticity and the generation of inflammatory pain*, Proc Natl Acad Sci U S A 1999;96: 7723.
417. Wright EF. *Referred craniofacial pain patterns in patients with temporomandibular disorder [see comment] [erratum appears in J Am Dent Assoc 131:1553, 2000]*, J Am Dent Assoc 2000;131: 1307.
418. Yaksh TL. *Central pharmacology of nociceptive transmission*, Wall P Melzack R Textbook of pain 2002; Churchill Livingstone Edinburgh 285.
419. Yamada T, Nakamura K, Iwaku M, Fusayama T. *The extent of the odontoblast process in normal and carious human dentin*, J Dent Res 1983;62: 798.
420. Yamaguchi M, Kojima T, Kanekawa M, et al: *Neuropeptides stimulate production of interleukin-1 beta, interleukin-6, and tumor necrosis factor-alpha in human dental pulp cells*, Inflamm Res 2004;53: 199.
421. Yamamura T. *Differentiation of pulpal wound healing*, J Dent Res special issue, 1985;64: 530.
422. Yang BH, Piao ZG, Kim Y-B. *Activation of vanilloid receptor 1 (VR1) by eugenol*, J Dent Res 2003;82: 781.
423. Yonehara N, Amano K, Kamisaki Y. *Involvement of the NMDA-nitric oxide pathway in the development of hypersensitivity to tactile stimulation in dental injured rats*, Jpn J Pharmacol 2002;90: 145.

Telegram: @dental_k

www.konkur.in

424. Yu CY, Boyd NM, Cringle SJ. *An in vivo and in vitro comparison of the effects of vasoactive mediators on pulpal blood vessels in rat incisors*, Arch Oral Biol 2002;47: 723.
425. Yu CY, Boyd NM, Cringle SJ, et al: *Oxygen distribution and consumption in rat lower incisor pulp*, Arch Oral Biol 2002;47: 529.
426. Zerari-Mailly F, Braud A, Davido N, et al: *Glutamate control of pulpal blood flow in the incisor dental pulp of the rat*, Eur J Oral Sci 2012;120: 402.
427. Zerlotti E. *Histochemical study of the connective tissue of the dental pulp*, Arch Oral Biol 1964;9: 149.
428. Zhang J, Kawashima N, Suda H, et al: *The existence of CD11c1 sentinel and F4/801 interstitial dendritic cells in dental pulp and their dynamics and functional properties*, Int Immunol 2006;18: 1375.

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*The authors acknowledge the outstanding work of Drs. Henry Trowbridge, Syngcuk Kim, Hideaki Suda, David H. Pashley, and Fredrick R. Liewehr in previous editions of this text. The present chapter is built on their foundational work.

†A receptor cell is a non–nerve cell capable of exciting adjacent afferent nerve fibers. Synaptic junctions connect receptor cells to afferent nerves.

‡To appreciate fully the dimensions of dentin tubules, understand that the diameter of the tubules (about 1 μm) is much smaller than that of red blood cells (about 7 μm). The thickness of coronal dentin is about 3 mm, so each tubule is 3000 μm long but only 1 μm in diameter. Thus each tubule is 3000 diameters long.

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[§]A force of 44 cM (44 g) applied to an explorer having a tip 40 μm in diameter would produce a pressure of 2437 MPa on the dentin.[55] This is far in excess of the compressive strength of dentin, listed as 245 MPa, as evidenced by the shallow grooves lined by smear layers created in dentin using this force.[56]

[¶]The term *dead tract* refers to a group of dentinal tubules in which odontoblast processes are absent. Dead tracts are easily recognized in ground sections because the empty tubules refract transmitted light, and the tract appears black in contrast to the light color of normal dentin.

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14: Pulp reactions to caries and dental procedures

Ashraf F. Fouad, Linda G. Levin

CHAPTER OUTLINE

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The dental pulp is a dynamic tissue that responds to external stimuli in a variety of ways. However, there are certain unique features of the dental pulp response that distinguish it from other connective tissues in the body. These include the pulp's exposure to dental caries, a very prevalent chronic infectious disease, its encasement in an unyielding environment after complete tooth maturation, and the paucity of collateral circulation render it susceptible to injury and complicate its regeneration. Moreover, the pulp is endowed with a rich neurovascular supply that promotes the effects of inflammation and may lead to rapid degeneration and necrosis. The treatment of dental caries and other tooth pathosis often involves the cleaning and shaping of the enamel and dentin, the hardest tissues in the body, thus adding to the irritation of the pulp. In this chapter, the response of the pulp to all these variables will be discussed and recent advances in our understanding of dental procedures and their effects on the pulp will be presented.

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Pulp reaction to caries

Dental caries is a localized, destructive, and progressive infection of dentin, which, if left unchecked, can result in pulp necrosis and potential tooth loss. Both bacterial by-products and products from the dissolution of the organic and inorganic constituents of dentin mediate the effects of dental caries on the pulp. Three basic reactions tend to protect the pulp against caries: (1) decreases in dentin permeability due to dentin sclerosis, (2) tertiary dentin formation, and (3) inflammatory and immune reactions in the pulp.[143] These responses occur concomitantly and their robustness is highly dependent on the aggressive nature of the advancing lesion.

Bacterial proteolytic enzymes, toxins, and metabolic by-products have been thought to be initiators of pulp reactions, yet the buffering capacity of dentin and dentinal fluid likely attenuate these stimulatory effects. This protective function is significantly reduced when the remaining dentin thickness (RDT) is minimal.[267] When the pulp is exposed, bacterial metabolites, toxins, and cell wall components induce inflammation. In initial to moderate lesions, current evidence suggests that acidic by-products of the carious process act indirectly by degrading the dentin matrix, thereby liberating bioactive molecules previously sequestered during dentinogenesis. Once liberated, these molecules resume their role in dentin formation, this time being stimulatory for *tertiary* dentinogenesis.[268] This theory is supported by the findings that demineralized dentin matrix implanted at the site of pulp exposure can induce dentinogenesis.[300] Furthermore, placement of purified dentin matrix proteins on exposed dentin or exposed pulp stimulates tertiary dentin formation, indicating that these molecules can act directly or across intact dentin.[269,299]

Recent evidence suggests several candidate molecules for stimulation of reparative dentinogenesis. Transforming growth factor-β1 (TGF-β1), TGF-β3, insulin-like growth factors I (IGF-I) and II, platelet-derived growth factor (PDGF), bone morphogenetic protein-2 (BMP-2), and angiogenic growth factors such as vascular endothelial growth factor (VEGF) have been shown to be stimulatory for dentinogenesis in vitro.[259] The TGF-β superfamily in particular seems to be important in the signaling process for odontoblast differentiation as well as primary and tertiary dentinogenesis. As the

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predominant isoform, TGF-β1 is equally distributed in the soluble and insoluble fractions of dentin matrix.[43] During the carious dissolution of dentin, it is believed that the soluble pool of TGF-β1 can diffuse across intact dentin while the insoluble pool is immobilized on insoluble dentin matrix and serves to stimulate odontoblasts, much like membrane-bound TGF-βs during odontogenesis.[267,270]

Despite the research interest in tertiary dentinogenesis, it is neither the first nor necessarily the most effective defense against invading pathogens. A combination of an increased deposition of intratubular dentin and the direct deposition of mineral crystals into the narrowed dentin tubules to decrease dentin permeability is the first defense to caries and is called *dentin sclerosis*. The combination of increased deposition of intratubular dentin and tubule occlusion by precipitated crystals results in an effective decrease in dentin permeability underneath the advancing carious lesion.[229] In vitro studies with cultured tooth slices implicate TGF-β1 as a central player in the increased deposition of intratubular dentin.[266] The deposition of whitlockite crystals in the tubular lumen most likely results from a similar stimulation of vital associated odontoblasts, possibly in combination with precipitation of mineral released during the demineralization process (Fig. 14.1).[171,296]

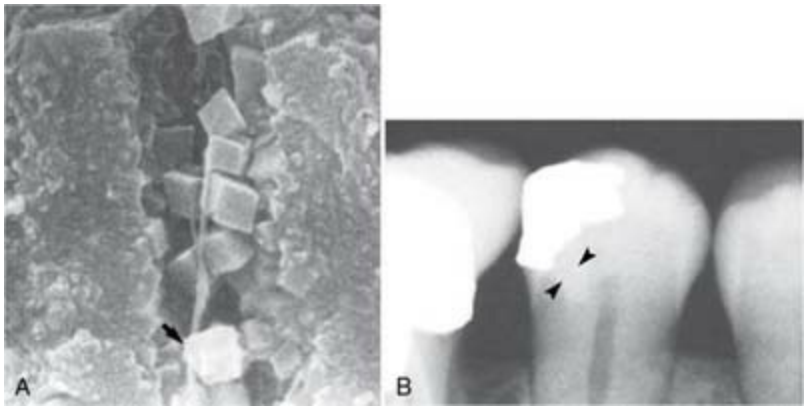

FIG. 14.1 **A,** Whitlockite crystals occlude the dentinal tubules in sclerotic dentin. **B,** Dentinal sclerosis is radiographically apparent beneath a deep class II lesion.

A) Micrograph shows a deep canal at the center with cube-like structures.

B) Radiograph shows a white spot of sclerosis at the top right of root canal below an extremely radiopaque patch.

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The formation of tertiary dentin occurs over a longer period than does that of sclerotic dentin, and its resultant character is highly dependent on the stimulus. Mild stimuli activate resident quiescent odontoblasts whereupon they elaborate the organic matrix of dentin. This type of tertiary dentin is referred to as *reactionary dentin* and can be observed when initial dentin demineralization occurs beneath the noncavitated enamel lesion.[164] Mediators present during the carious process induce a focal upregulation of matrix production by resident odontoblasts. The resultant dentin is similar in morphology to physiologic dentin and may only be apparent due to a change in the direction of the new dentinal tubules (Fig. 14.2). In the aggressive lesion, the carious process may prove cytocidal to subjacent odontoblasts and require repopulation of the disrupted odontoblast layer with differentiating progenitors. The organization and composition of the resultant matrix are a direct reflection of the differentiation state of the secretory cells. This accounts for the heterogeneity of *reparative dentin*, where the morphology can range from organized tubular dentin to more disorganized irregular *fibrodentin*. Fibrodentin, due to its irregular configuration and tissue inclusions, is more permeable than physiologic dentin (Fig. 14.3).[310]

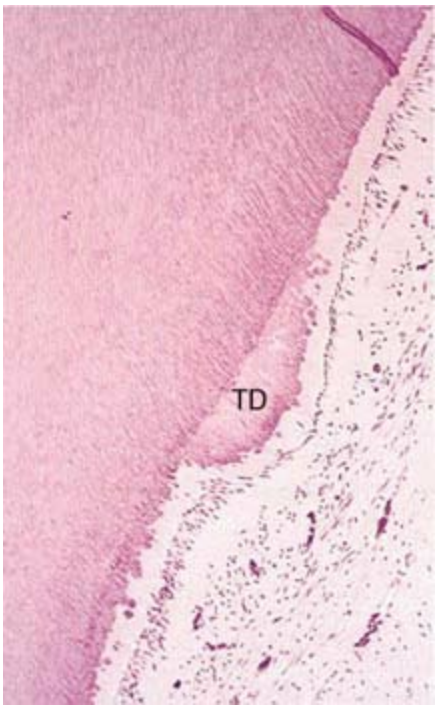

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FIG. 14.2 Reactionary dentinogenesis *(TD)*. Note the tubular morphology and the discontinuity of the tubules at the interface of secondary and reactionary dentin. Resident odontoblasts are still present.

Stained micrograph shows a protuberance of dentin tubules, marked TD, at the center extending toward pulp.

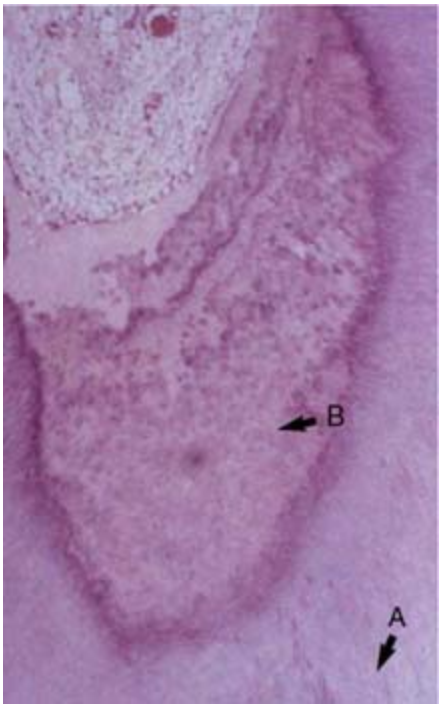

FIG. 14.3 Tertiary dentin. The strong stimulus of the impinging infection is cytocidal for odontoblasts. The resultant dentin is irregular with soft-tissue inclusions. Intact secondary dentin is depicted by *arrow A,* and tertiary dentin is designated by *arrow B*.

Stained micrograph shows a magnified view of reparative dentin consisting of the matrix of disorganized cells.

While dentin can provide a physical barrier against noxious stimuli, the pulp immune response provides humoral and cellular challenges to invading pathogens. In the progressing carious lesion, the host immune response increases in intensity as the infection advances. It has been shown that titers of T helper cells, B-lineage cells, neutrophils, and macrophages are directly proportional to lesion depth in human teeth.[126] The disintegration of large

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amounts of dentin, however, is not necessary to elicit a pulp immune response. This is supported by the observation that a pulp inflammatory response can be seen beneath noncavitated lesions and noncoalesced pits and fissures.[36]

The early inflammatory response to caries is characterized by the focal accumulation of chronic inflammatory cells (Fig. 14.4). This is mediated initially by odontoblasts and later by dendritic cells. As the most peripheral cell in the pulp, the odontoblast is positioned to encounter foreign antigens first and initiate the innate immune response. Pathogen detection in general is accomplished via specific receptors called pattern recognition receptors (PRRs).[128] These receptors recognize pathogen associated molecular patterns (PAMPs) on invading organisms and initiate a host defense through the activation of the NF-κB pathway.[106] One class of the PAMP recognition molecules is the Toll-like receptor (TLR) family. Odontoblasts have been shown to have an increased expression of certain TLRs in response to bacterial products. Under experimental conditions, odontoblast expression of TLR3, 5, and 9 was increased in response to lipoteichoic acid while lipopolysaccharide increased TLR2 and 4 expression.[71,114,204] It was also shown that TGF-β1 inhibits the expression of TLR2 and 4 by odontoblasts in response to gram-positive and -negative bacteria.[114] Once the odontoblast TLR is stimulated by a pathogen, proinflammatory cytokines, chemokines, and antimicrobial peptides are elaborated by the odontoblast resulting in recruitment and stimulation of immune effector cells as well as direct bacterial killing.[80,82,138]

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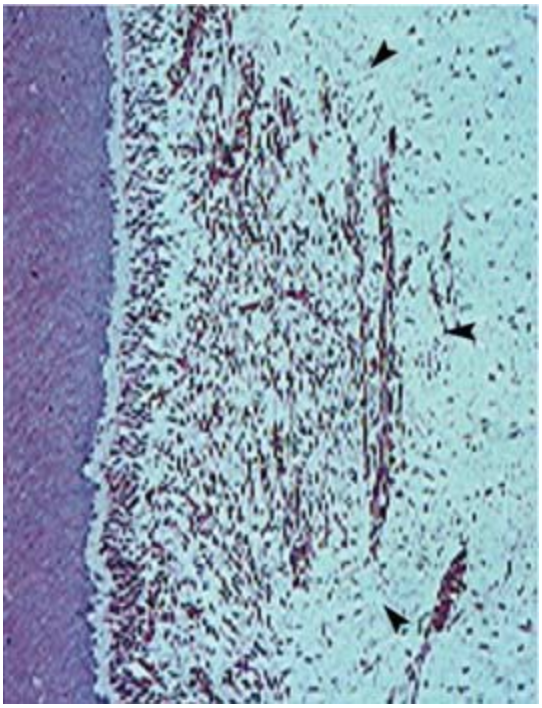

FIG. 14.4 The early pulpal response to caries is represented by a focal accumulation of chronic inflammatory cells *(arrowheads)*. Note that peripheral to the inflammation the pulpal parenchyma is relatively unaffected.

Stained micrograph shows pulp layer with high density of cells toward the odontoblasts.

Many cells produce chemokines at low levels constitutively. Unstimulated odontoblasts express genes coding for CCl2, CXCL12, and CXCL14, three genes known to code for factors chemotactic for immature dendritic cells.[44] They also produce CCL26—a natural antagonist for CCR1, CCR2, and CCR5—which are chemokines normally produced by monocytes and dendritic cells.[324] Stimulation with bacterial cell wall constituents has been shown to upregulate the expression of multiple chemokine genes, including CXCL12, CCL2, CXCL9, CX3CL1, CCL8, CXCL10, CCL16, CCL5, CXCL2, CCL4, CXCL11, and CCL3, as well as 9 chemokine receptor genes, including CXCR4, CCR1, CCR5, CX3CR1, CCR10, and CXCR3, suggesting that odontoblasts sense pathogens and express factors that recruit immune effector cells (Fig. 14.5).[44,81,113,165] These data suggest a scenario whereby stimulated odontoblasts express high levels of chemokines, such as IL-8 (CXCL8), that act in concert with the release of formerly sequestered growth

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factors from carious dentin, which induce a focal increase in dendritic cell numbers with additional release of chemotactic mediators.[83,270] The subsequent influx of immune effector cells is composed of lymphocytes, macrophages, and plasma cells. This cellular infiltrate is accompanied by localized capillary sprouting in response to angiogenic factors as well as coaggregation of nerve fibers and HLA-DR-positive dendritic cells.[316,317]

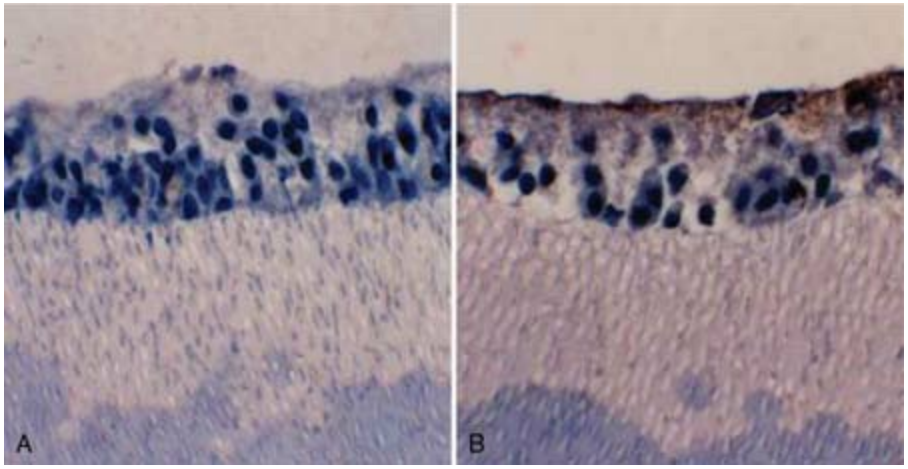

FIG. 14.5 Odontoblasts exposed to lipopolysaccharide (LPS) in an in vitro culture model express IL-8 as evidenced by immunostaining with anti-IL-8 antibodies. **A,** Control cultures not exposed to LPS (negative control). **B,** Immunostaining for IL-8 in cultures exposed to LPS.

Set of two stained micrographs marked A and B shows the changes in odontoblasts with abnormal periphery in B micrograph.

As the carious lesion progresses, the density of the chronic inflammatory infiltrate and well as that of dendritic cells in the odontoblast region increases. Pulp dendritic cells are responsible for antigen presentation and stimulation of T lymphocytes. In the uninflamed pulp, they are scattered throughout the pulp. With caries progression, they aggregate initially in the pulp and subodontoblastic regions then extend into the odontoblast layer and eventually migrate into the entrance to tubules beside the odontoblast process (Fig. 14.6).[318] There are two distinct populations of dendritic cells that have been identified in the dental pulp. CD11c+ is found in the pulp/dentin border and subjacent to pits and fissures. F4/80+ dendritic cells are concentrated in the perivascular spaces in the subodontoblastic zone and inner pulp.[324]

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CD11c+ dendritic cells express TLRs 2 and 4 and are CD205 positive. F4/80+ dendritic cells have migratory ability. As they migrate from the central pulp, they increase in size and become CD86 positive. In vitro studies have suggested that the secretion of GM-CSF and osteopontin by dendritic cells and macrophages represents a mechanism whereby they contribute to odontoblast differentiation.[255]

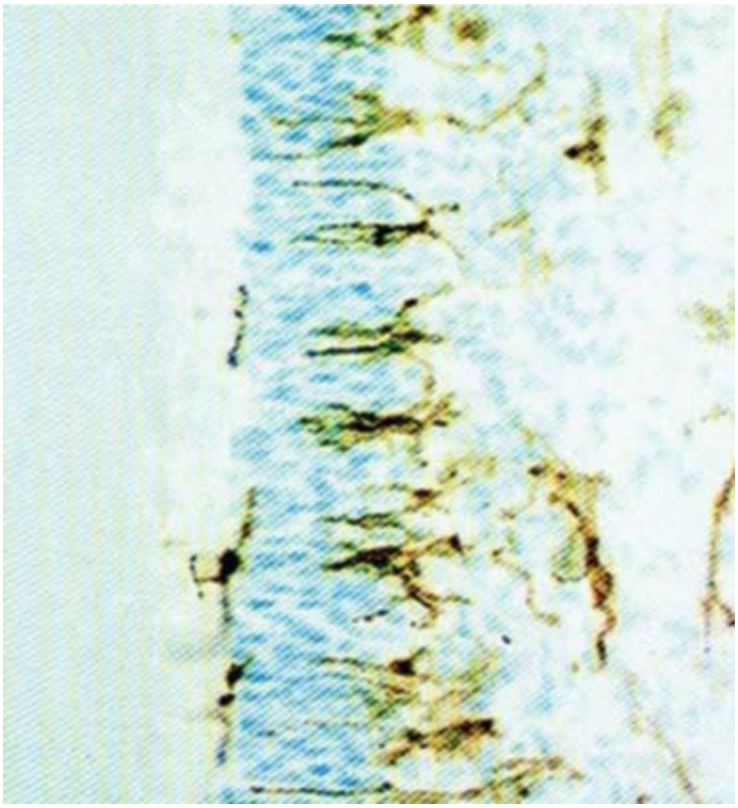

FIG. 14.6 Dental caries stimulates the accumulation of pulpal dendritic cells in and around the odontoblastic layer.

Micrograph shows several branched dendritic cells in odontoblastic layer extending toward the dentin layer.

Source: (Reprinted with permission from Mats Jontell.)

Evidence suggests that odontoblasts also play a role in the humoral immune response to caries. IgG, IgM, and IgA have been localized in the cytoplasm and cell processes of odontoblasts in human carious dentin, suggesting that these cells actively transport antibodies to the infection front.[211] In the incipient lesion antibodies accumulate in the odontoblast layer

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and with lesion progression can be seen in the dentinal tubules. Eventually, this leads to a focal concentration of antibodies beneath the advancing lesion.[210]

In the most advanced phase of carious destruction, the humoral immune response is accompanied by immunopathologic destruction of pulp tissue. In animal studies where monkeys were hyperimmunized to BSA, there was an observed increase in pulp tissue destruction subsequent to antigenic challenge across freshly cut dentin.[22] Odontoblasts also appear to be involved in the production of innate antimicrobial molecules such as human beta defensin-2 (HBD2). IL-1 and TNF-α as well as bacterial LPS were responsible for significant increases in HBD2 in response to caries.[113] In sum, it appears that the odontoblasts play a central role in orchestrating local and chemotactic inflammatory responses to dental caries (Fig. 14.7).

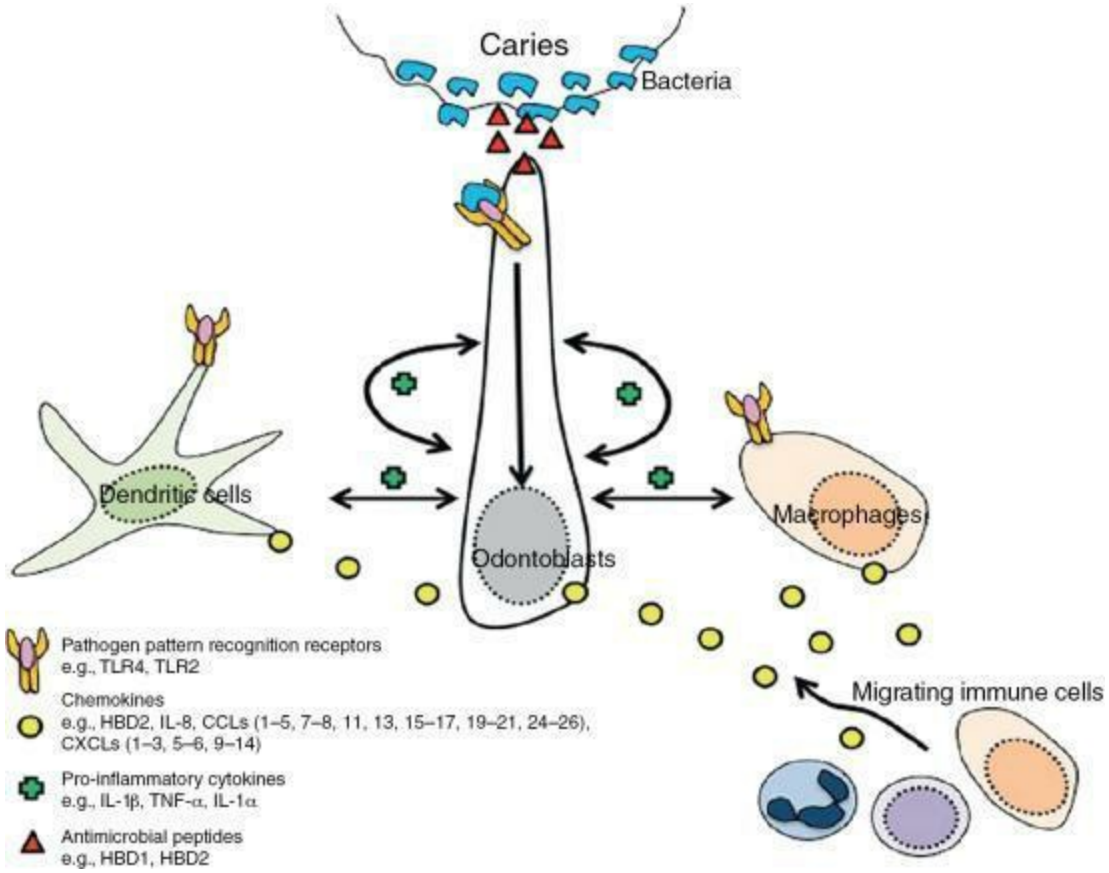

FIG. 14.7 Innate immunity in the odontoblast layer (ODL). Bacterial components from caries activate cytokine/chemokine release from odontoblasts, dendritic cells, and/or macrophages via Toll-like

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receptors *(TLRs)*. Proinflammatory cytokines released from these cells act as autocrine and paracrine signals to amplify cytokine responses including antimicrobial peptide, cytokine, and chemokine production. The release of chemokines creates a migration gradient for immune cells to ODL while antimicrobial peptides reduce bacterial load.

Diagram explains innate immunity in the odontoblast layer. Labels are as follows: Odontoblasts, macrophages, dendritic cells, and migrating immune cells. Examples of pathogen pattern recognition receptors are TLR4, TLR2. Examples of chemokines are HBD2, IL-8, CCLs (1–5, 7–8, 11, 13, 15–17, 19–21, 24–26), CXCLs (1–3, 5–6, 9–14). Examples of pro-inflammatory cytokines are IL-1 beta, TNF-alpha, IL-1 alpha. Examples of antimicrobial peptides are HBD1, HBD2.

Source: (Reprinted with permission from Horst OV, Horst JA, Samudrala R, Dale BA. Caries induced cytokine network in the odontoblast layer of human teeth. *BMC Immunol* 12:9, 2011, Fig. 4C.)

Pulp exposure in primary and immature permanent teeth can lead to a proliferative response or *hyperplastic pulpitis*. Exuberant inflammatory tissue proliferates through the exposure and forms a “pulp polyp” (Fig. 14.8). It is presumed that a rich blood supply facilitates this proliferative response. Conventional root canal therapy or therapeutic pulpotomy would be indicated in these cases.

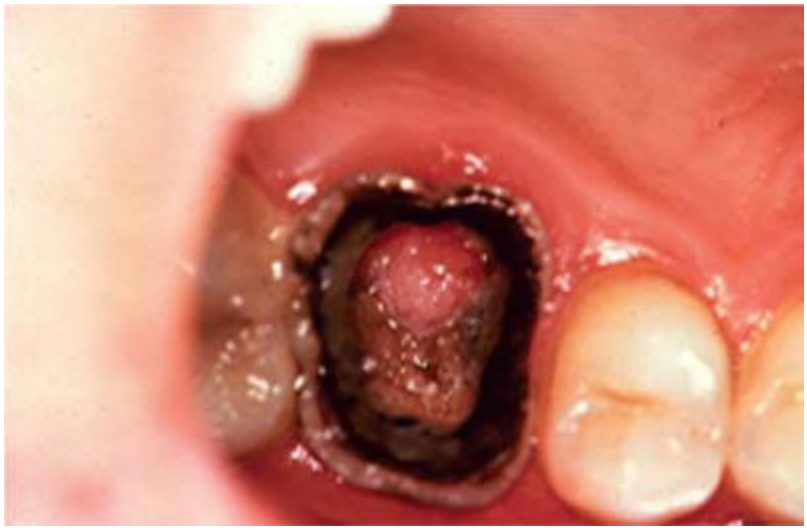

FIG. 14.8 A proliferative response to caries in a young tooth, typically referred to as proliferative pulpitis, hyperplastic pulpitis, or pulp polyp.

Close-up view shows abnormal growth of large mass surrounded by a cavity in place of the tooth.

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Source: (Courtesy Dr. Howard Strassler at the University of Maryland, with permission.)

Neurogenic mediators

Neurogenic mediators are involved in the pulp response to irritants and like immune components they can mediate pathology as well as the healing response. External stimulation of dentin causes the release of proinflammatory neuropeptides from pulp afferent nerves.[38,139] Substance P (SP), calcitonin gene-related peptide (CGRP), neurokinin A (NKA), neurokinin Y (NKY), and vasoactive intestinal peptide (VIP) are released and affect vascular events such as vasodilatation and increased vascular permeability. This results in a net increase in tissue pressure that can progress to necrosis in extreme and persistent circumstances. Stimulation of sympathetic nerves in response to the local release of mediators such as norepinephrine, neuropeptide Y, and ATP has been shown to alter pulp blood flow (PBF). Receptor field studies as well as anatomic studies have shown sprouting of afferent fibers in response to inflammation.[38]

Neuropeptides can act to modulate the pulp immune response. It has been demonstrated that SP acts as a chemotactic and stimulatory agent for macrophages and T lymphocytes. The result of this stimulation is increased production of arachidonic acid metabolites, stimulation of lymphocytic mitosis, and production of cytokines. CGRP demonstrates immunosuppressive activity, which is evidenced by a diminution of Class II antigen presentation and lymphocyte proliferation.

SP and CGRP are mitogenic for pulp and odontoblast-like cells, thereby initiating and propagating the pulp healing response.[294] CGRP has been shown to stimulate the production of bone morphogenic protein by human pulp cells. The result of such stimulation has been postulated to induce tertiary dentinogenesis.[40] SP appears to increase in the dental pulp and periodontal ligament as a result of acutely induced occlusal trauma,[47] which may be related to the pain associated with concussion traumatic injury.

It was shown that there may be gender differences in CGRP production in the dental pulp.[31,175] In one study, serotonin (a peripheral pronociceptive mediator) induced a significant increase in capsaicin-evoked CGRP release in

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dental pulps obtained from female and not male patients.[175] This interplay of inflammatory mediators may explain some of the gender differences in clinical presentation with dental pain.

It was also recently shown that these neurotropic factors are not only present in dentin matrix but may be released and may aid in trigeminal neurite growth during regenerative procedures of the dental pulp.[13,313]

Correlation between clinical symptoms and actual pulp inflammation

From a clinical perspective, it would be most helpful to the clinician to be able to diagnose pulp conditions from a profile of symptoms with which a patient presents. If symptoms are not conclusive, a number of objective tests should aid the clinician in reaching a definitive diagnosis of the pulp pathological status. In actuality, such combinations of subjective and objective findings are frequently insufficient in reaching definitive diagnosis of the status of the dental pulp. This is particularly true in cases of vital inflamed pulp, where it is difficult for the practitioner to determine clinically whether the inflammation is reversible or irreversible.

Many practitioners rely on painful symptoms to determine the status of the pulp. Several studies have examined this question in some detail. Several classic studies were performed in which the subjective and objective clinical findings related to carious teeth were recorded prior to extracting the teeth and examining them histologically. The underlying hypothesis in these studies was that the more severe the clinical symptoms, the more intense pulp inflammation and destruction was evident histologically. These studies showed that in the vital pulp, clinical symptoms generally did not correlate with histopathological findings.[105,189,262] Furthermore, carious pulp exposure was associated with severe inflammatory response or liquefactive necrosis, regardless of symptoms (Fig. 14.9). These histological changes ranged in extent from being present only at the site of the exposure to deep into the root canals.[262] In a few studies, prolonged or spontaneous severe symptoms were associated with chronic partial or total pulpitis, or pulp necrosis.[68,262] However, in these as well as in other studies, it was common to find cases with evidence of severe inflammatory responses including partial necrosis

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histologically, but with little or no clinical symptoms—the so-called "painless pulpitis."[68,105,189,262] Moreover, the frequency of nerve fibers,[252] and the vascularity[253] in inflamed pulp does not correlate with clinical symptoms in primary and permanent teeth. It has been reported that the incidence of painless pulpitis that leads to pulp necrosis and asymptomatic apical periodontitis is about 40% to 60% of all pulpitis cases.[188] More recently, one study[248] documented the clinical and histopathological findings and showed that the clinical diagnosis of normal pulp/reversible pulpitis match the histologic diagnosis in 96.6% of the time and of irreversible pulpitis 84.4%, respectively. They also noted that the inflammation and bacterial infection was often localized to the coronal pulp when the tooth responded as irreversible pulpitis. This is critical for vital pulp therapy (VPT), as the radicular pulp is maintained in that procedure. However, this study was retrospective, based on one dentist's subjective definitions of reversible and irreversible pulpitis with no standardization of testing. Asymptomatic irreversible pulpitis was not included. Moreover, cases with signs and symptoms of symptomatic irreversible pulpitis and normal periapex respond well to emergency pulpotomy[77] and to VPT with a tricalcium silicate.[166]

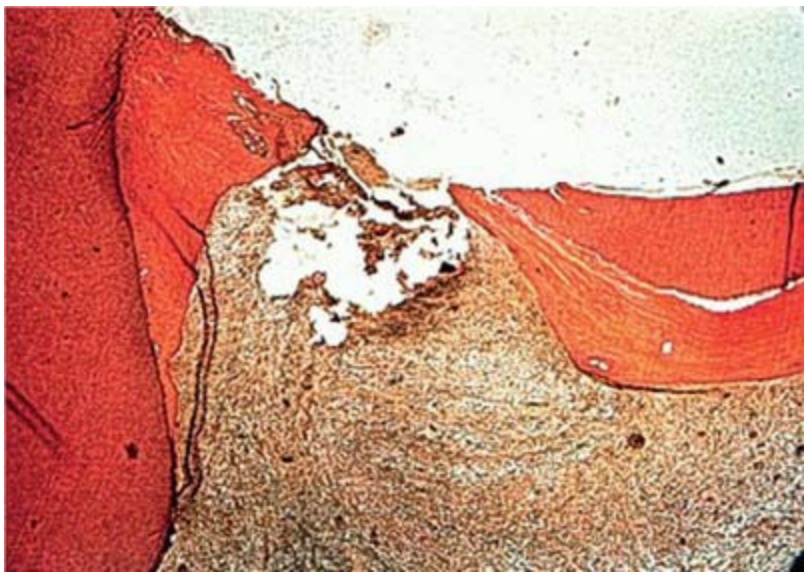

FIG. 14.9 Histological photomicrograph of a molar with carious pulp exposure. The exposure had been capped but had failed and the patient presented with symptoms. The photomicrograph shows an area

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of necrosis and extensive inflammation throughout the coronal pulp.

Stained micrograph shows dead tissue in the crown of tooth.

Source: (Courtesy Dr. Larz Spangberg at the University of Connecticut, with permission.)

Objective clinical findings are essential for determining the vitality of the pulp and whether the inflammation has extended into the periapical tissues. Lack of response to electric pulp testing is generally indicative that the pulp has become necrotic.[247,262] Thermal pulp testing is valuable in reproducing a symptom of thermal sensitivity, and in allowing the practitioner to assess the reaction of the patient to a stimulus and the duration of the response. However, pulp testing cannot determine the degree of pulp inflammation.[68,262] These studies show that irreversible pulp inflammation can be diagnosed with some certainty only in cases where, in addition to being responsive to pulp testing, the pulp develops severe spontaneous symptoms. Pulp necrosis could be predictably diagnosed by a consistent negative response to pulp tests, preferably to both cold and electrical tests to avoid false responses.[234,235] Recent studies show the accuracy of traditional pulp testing to be about 82% to 84%.[179]

The lack of correlation between the histological status of the pulp and clinical symptoms may be explained by recent advances in the science of pulp biology. In the last few decades, studies have shown that numerous molecular mediators may act in synchrony to initiate, promote, and/or modulate the inflammatory response in the dental pulp. The nature and quantity of these inflammatory mediators cannot be determined from histological analysis, without the use of very specialized staining techniques and molecular assays. Many of these molecular mediators tend to reduce the pain threshold, either directly by acting on peripheral nerve cells or through promoting the inflammatory process. Thus a number of these mediators were shown to be elevated in human pulp diagnosed with painful pulpitis. These mediators include prostaglandins,[53,251] the vasoactive amine bradykinin,[163] tumor necrosis factor alpha,[151] neuropeptides such as SP,[32] CGRP and neurokinin A,[14] and catecholamines.[207] In fact, it was even shown that when patients have painful pulpitis, the crevicular fluid related to the affected teeth has significantly increased neuropeptides compared to the levels in

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contralateral teeth.[14]

It has also been determined that peripheral opioid receptors are present in the dental pulp,[127] and these could play a role in helping us understand why many cases with irreversible pulpitis are asymptomatic. As noted before, carious teeth are frequently not associated with significant symptoms. However, they still have significant amount of inflammation. The pulp in teeth with mild to moderate caries has increased neuropeptide Y,[73] and its Y1 receptor,[74] compared to that in normal teeth. Neuropeptide Y is a neurotransmitter for sympathetic nervous system and is thought to act as a modulator of neurogenic inflammation. Likewise, the levels of VIP, although not its receptor VPAC1, seemed to increase in the pulp of moderately carious teeth.[72]

With recent advances in molecular biology, efficient detection of hundreds of molecular mediators simultaneously by their gene expression has become a reality. Current research seeks to examine which genes are specifically expressed or upregulated in the pulp, in response to the carious lesion. In this regard, preliminary studies have shown that various cytokines and other inflammatory mediators are upregulated underneath a carious lesion, in a manner that correlates with the depth of caries.[184] Gene microarrays have been used in several studies to obtain an accurate mapping of candidate genes that show elevated expression in inflamed pulp and the odontoblastic cell layer.[185,216,217] In addition, research has revealed the differential expression of microRNAs (miRNAs) in healthy and diseased dental pulp,[326] and the increase in viral microRNA in inflamed pulp, to correlate with levels of inflammatory mediators.[325] miRNAs are noncoding RNA molecules that regulate gene expression in complex inflammatory responses and may eventually assist in clinical predictions of pulp status. Therefore more accurate chair-side diagnostic methods are potentially feasible to develop; particularly, sampling can be made from crevicular fluid, dentinal fluid, or the pulp directly. For this reason, more research is needed to determine the key mediators that would predict survival or degeneration of the dental pulp in difficult diagnostic cases.

Dentin hypersensitivity and its management

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Dentin hypersensitivity is a special situation in which a significant, chronic, pulp pain arises, which does not seem to be associated with irreversible pulp pathosis, in most cases. Dentin hypersensitivity is characterized by brief sharp pain arising from exposed dentin in response to stimuli (typically thermal, evaporative, tactile, osmotic or chemical) that cannot be ascribed to any other form of dental defect or pathology.[111] Facial root surfaces in canines, premolars, and molars are particularly affected, especially in areas of periodontal attachment loss. Dentin hypersensitivity may be related to excessive abrasion during tooth brushing, periodontal disease, or erosion from dietary or gastric acids,[2,3,49] and may be increased following scaling and root planning.[49,308] The dentin is hypersensitive most likely due to the lack of protection by cementum, loss of smear layer by acidic dietary fluids, and the hydrodynamic movement of fluid in dentinal tubules.[5,35] The degree of inflammation in the pulp in cases of dentin hypersensitivity is not well characterized because the condition is usually not severe enough to warrant tooth extraction or endodontic therapy. However, patent dentinal tubules are present in areas of hypersensitivity (Fig. 14.10)[320] and may result in increased irritation and localized reversible inflammation of the pulp at the sites involved.

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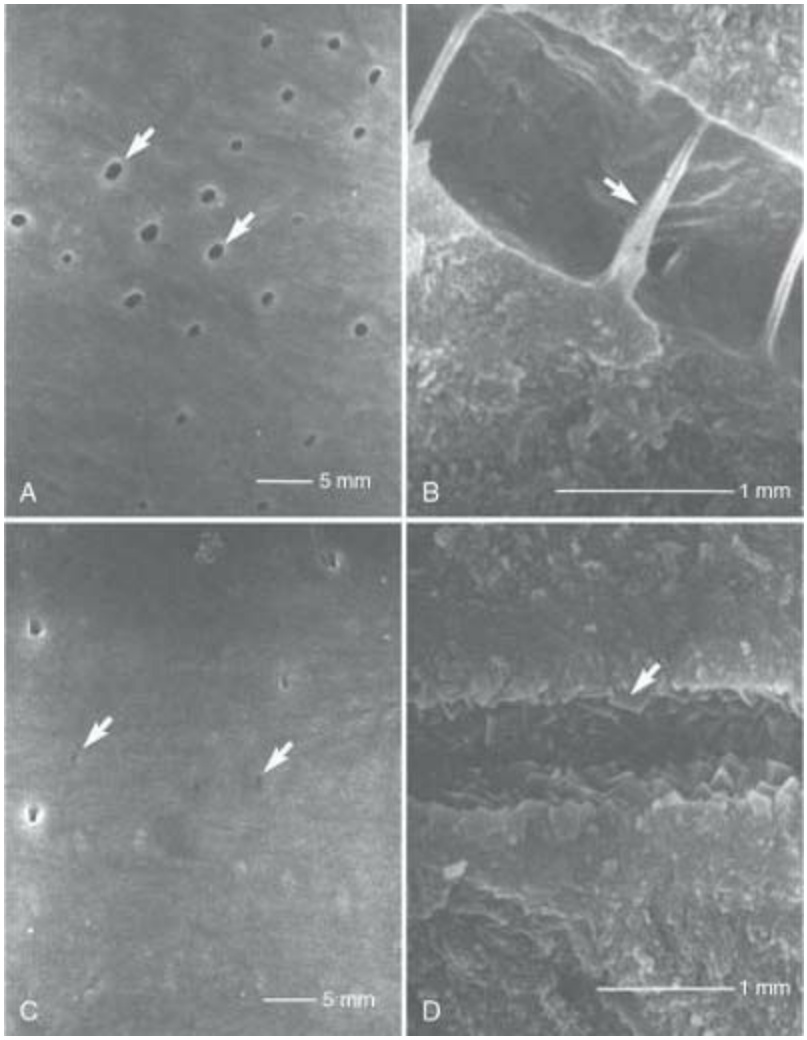

FIG. 14.10 Scanning electron microscope (SEM) image of an exposed dentin surface of a hypersensitive area. **A,** A large proportion of dentinal tubules *(arrows)* are seen to be open. **B,** SEM image of a fractured dentinal tubule of a hypersensitive area. The lumen of the dentinal tubule is partitioned by membranous structures *(arrow)*. **C,** SEM image of exposed dentin surface of a naturally desensitized area. The lumens of dentinal tubules *(arrows)* are mostly occluded, and the surface is extremely smooth. **D**, SEM image of a fractured dentinal tubule of a naturally-desensitized area. Rhombohedral platelike crystals of from 0.1 to 0.3 μm *(arrow)* are present.

Set of four SEM images marked A through D shows circular open dentins, a hollow chamber with septum, slightly blurred small dentins, and a long, horizontal channel with crystals, respectively.

Source: (Reprinted with permission from Yoshiyama M, Masada J, Uchida A, Ishida H. Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin. *J Dent Res* 68:1498–1502, 1989.)

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The application of neural modulating agents such as potassium nitrate,[183] or tubule blocking agents such as strontium chloride, oxalates, or dentin bonding agents (Fig. 14.11),[5,224] usually alleviates the condition, at least temporarily. However, the placement of passive molecules or crystals may provide only temporary relief, thus there has been the need to provide biocompatible materials that bond to the root surface, in order to provide a more lasting solution. One such material was a calcium sodium phosphosilicate bioactive glass,[172] which was developed into a commercial product (SootheRx, NovaMin Technology Inc., Alachua, FL). Another product uses a combination of a calcium oxalate and an acid-etched bonding material to seal the dentinal tubules (BisBlock, Bisco Inc., Schaumberg, IL). A concern has been raised that the acidic pH during etching may cause dissolution of the oxalate crystals, thus interfering with the effectiveness of the material,[315] However, one study found that BisBlock and two other products (Seal&Protect, Dentsply DeTrey GmbH, Konstanz, Germany, and Vivasens, Ivoclar Vivadent AG, Schaan, Liechtenstein) were effective compared to placebo, several weeks after treatment,[220] In the long term, the development of smear layer, such as from tooth brushing, dentin sclerosis, reactionary dentin, and the blockage of tubules with large endogenous macromolecules, are all thought to reduce the problem[225] (illustrated by Dentin Hypersensitivity animation at www.PathwaysofthePulp.com). A practice-based randomized clinical trial compared the effectiveness of non-desensitizing toothpaste (Colgate Cavity Protection Regular, Colgate-Palmolive, United States), with desensitizing toothpaste (Colgate Sensitive Fresh Stripe, Colgate-Palmolive, United States) and a professionally applied desensitizing agent (Seal and Protect, Dentsply, United States).[95] The findings showed a significant reduction of dentin hypersensitivity in the desensitizing therapies compared to the non-desensitizing group. In fact, the reduction was much more significant than that obtained by the professionally applied desensitizing agent over a 6-month period. Contemporary desensitizing agents that have been found to be effective include nano-hydroxyapatite,[66] laser treatment, HEMA and glutaraldehyde primer, 3% oxalate, or home products containing 8% arginine/NaF or 5% potassium nitrate.[93]

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FIG. 14.11 Smear layer treated with 30% dipotassium oxalate for 2 minutes plus 3% monopotassium and monohydrogen oxalate for 2 minutes. Dentin surface is completely covered with calcium oxalate crystals. (Original magnification ×1900.)

SEM image shows magnified view of octahedral, irregular, and ovoid shaped calcium oxalate crystals on dentin surface of tooth.

Source: (Reprinted with permission from Pashley DH, Galloway SE. The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. *Arch Oral Biol* 30:731–737, 1985.)

Pulp reactions to local anesthetics

An intact PBF is critical for maintaining the health of the dental pulp. Because the dental pulp is enclosed in a rigid chamber and is supplied by few arterioles through the apical foramina, it cannot benefit from collateral circulation or volumetric changes that compensate for changes in blood flow in other soft tissues. Furthermore, reduction in blood flow has the compounding effect of reducing the clearance of large molecular weight toxins or waste products,[223] resulting in irreversible pulp pathosis. Vasoconstrictors are added to local anesthetics to enhance the duration of anesthesia. However, vasoconstrictors in local anesthetics could negatively impact the health of the pulp if they reduce blood flow, particularly if the pulp is inflamed preoperatively. Earlier studies have documented that

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vasoconstrictors in local anesthetics do reduce PBF in experimental animals when administered by infiltration and nerve block (Fig. 14.12),[142] and that this effect was more severe with periodontal ligament injections (Fig. 14.13).[141] More recently, clinical trials were conducted in which subjects were given infiltration of different local anesthetics with or without epinephrine at a concentration of 1:100,000 and the PBF was measured by the laser Doppler flowmetry. In groups that received the epinephrine, there were consistently significant reductions in PBF,[6,51,198] even if the infiltration was palatal to maxillary premolars.[237] Interestingly, in one study the reduction in PBF with epinephrine infiltration was more that the reduction in gingival blood flow, and did not return to baseline values after 1 hour of injection.[6] Similar reductions in PBF were reported when inferior alveolar nerve block injections of lidocaine and 1:100,000 or 1:80,000 epinephrine were administered.[209] It is important to note a limitation of studies using laser Doppler flowmetry, which is that a large proportion of the signal measured may be from sources other than the dental pulp.[236,271] Thus the monitoring of minor changes in PBF should be interpreted with caution, particularly if the rubber dam or a similar barrier was not used.[104] Human studies showed that intraosseous anesthesia reduced PBF in molars by 60% after 1 minute, which returned to normal within 45 minutes. A similar reduction was not observed for the blood flow in canines.[309] Taken together, these findings suggest that local anesthesia with vasoconstrictors may compromise the inflamed pulp's ability to recover from inflammation, particularly if it is severely inflamed or if the tooth is subjected to extensive restorative procedures or acute trauma, and if the anesthetic is delivered via a periodontal ligament or an intraosseous route.

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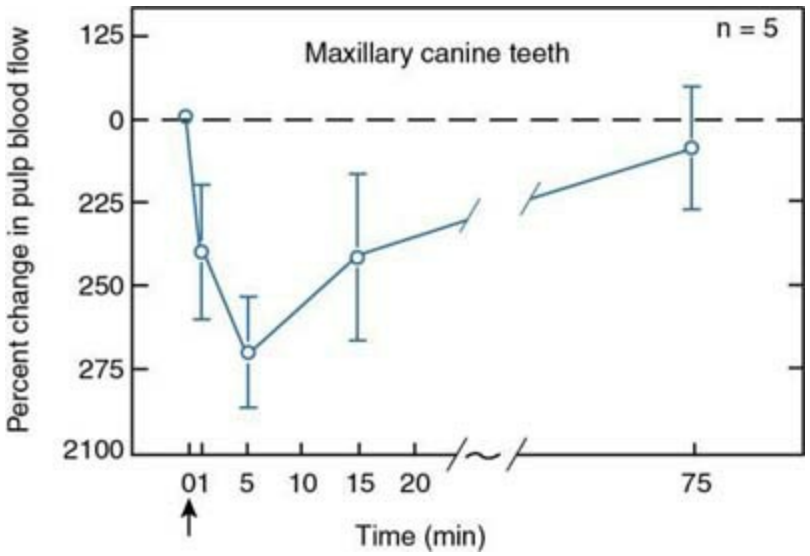

FIG. 14.12 Effects of infiltration anesthesia (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the maxillary canine teeth of dogs. There is a drastic decrease in pulpal blood flow soon after the injection. *Arrow* indicates the time of injection. *Bars* depict standard deviation.

Graph plots percent change in pulp blood flow in maxillary canine teeth against time in minutes. The vertical axis from top to bottom is marked as follows: 125, 0, 225, 250, 275, and 2100. The horizontal axis is marked at 01, 5, 10, 15, 10, and 75 minutes. The data points are as follows: (01, 0), (01, 240), (5, 272), (15, 240), (75, 100). All data are approximate.

Source: (Reprinted with permission from Kim S, Edwall L, Trowbridge H, Chien S. Effects of local anesthetics on pulpal blood flow in dogs. *J Dent Res* 63:650–652, 1984.)

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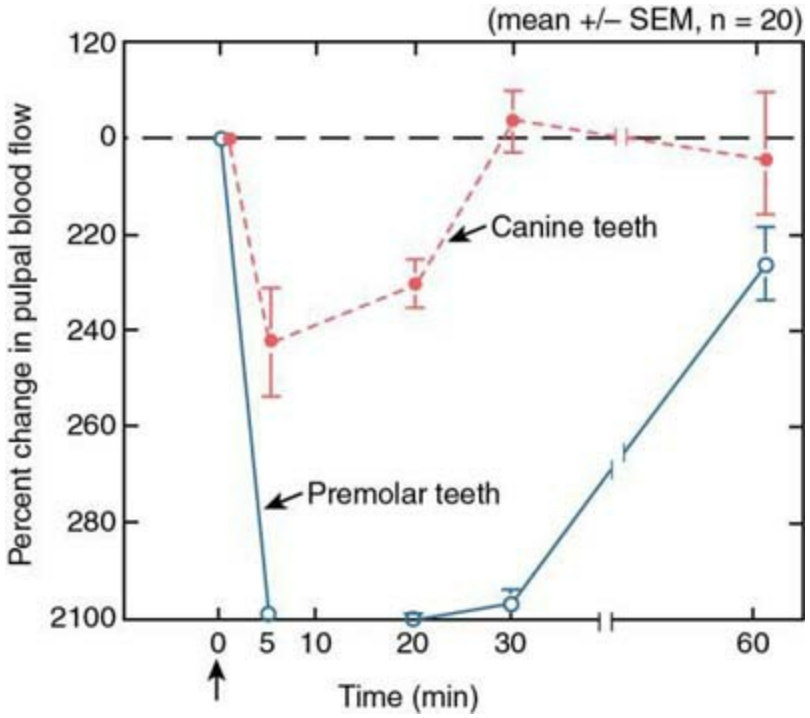

FIG. 14.13 Effects of ligamental injection (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the mandibular canine and premolar teeth of dogs. Injection was given in the mesial and distal sulcus of premolar teeth. Injection caused total cessation of pulpal blood flow which lasted about 30 minutes in the premolar teeth. *Arrow* indicates time of injection.

Graph plots percent change in pulpal blood flow in canine and premolar teeth against time in minutes. The vertical axis from top to bottom is marked as follows: 120, 0, 220, 240, 260, 280, and 2100. The horizontal axis is marked at 0, 5, 10, 20, 30, and 80 minutes. The data points are as follows. Canine teeth: (4, 0), (6, 242), (21, 235), (30, 20 above 0), and (60, 30 below 0). Premolar teeth: (5, 278), (20, 2100), (30, 2000), and (60, 228). Text above reads: mean 6 SEM, n 5 20). All data are approximate.

Source: (Reprinted with permission from Kim S. Ligamental injection: a physiological explanation of its efficacy. *J Endod* 12:486–491, 1986.)

Intrapulpal anesthesia is often used as a last resort, when pulp anesthesia is insufficient during root canal therapy. The effect of intrapulpal anesthesia on the pulp in these cases is not considered, as the pulp will be removed. However, occasionally VPT is indicated. One study has shown that intrapulpal anesthesia can be used in these cases, with no clinical differences on follow-up of over 24 weeks between the groups that did or did not receive intrapulpal anesthesia, and when given, in the groups where the anesthetic

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contained or did not contain epinephrine.[287] However, clearly this injection would have to be performed with caution.

Pulp reactions to restorative procedures

A large body of literature exists on the effects of restorative procedures on the dental pulp. This topic understandably, has been important for practicing dentists for many years. Restorative procedures are performed primarily to treat an infectious disease, dental caries, which itself causes significant irritation of the pulp. They may also be performed to help restore missing teeth, correct developmental anomalies, and address fractures, cracks, failures of previous restorations, or a myriad of other abnormalities. One key requirement of a successful restorative procedure is to cause minimal additional irritation of the pulp so as not to interfere with normal pulp healing. When pulp vitality is to be maintained during a restorative procedure, then a provisional diagnosis of reversible pulpitis rather than irreversible pulpitis must preexist. Therefore it would be most desirable to perform a minimally traumatic restorative procedure, which would not potentially convert the diagnosis to irreversible pulpitis. As was discussed previously, irreversible pulpitis may present clinically with severe spontaneous postoperative pain, but it may also be asymptomatic, leading to the asymptomatic demise of the pulp. The additive effects of restorative procedures are particularly critical in borderline cases, such as those of moderately symptomatic teeth with deep caries but no pulp exposure. There are still many factors whose influence on the response of the dental pulp to cumulative effects of caries, microleakage, restorative procedures, and materials is not well understood. It is generally accepted that the effects of pulp insults, be they from caries, restorative procedures, or trauma, are cumulative. That is, with each succeeding irritation, the pulp has a diminished capacity to remain vital. As a part of informed consent, the clinician is often faced with the task of outlining possible risks of restorative treatment. One study addressed the fate of pulps beneath single unit metal-ceramic (MC) crowns or MC bridge abutments.[50] Patients who had received either treatment were invited to attend a recall appointment that involved both clinical and radiographic examinations. One hundred and twenty-two teeth

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with preoperatively vital pulps treated with single unit MC crowns and 77 treated as bridge abutments were examined. The mean observation period was 14 years for the former and 15.6 years for the latter. Pulp necrosis had occurred in 15.6% of the teeth treated with single unit crowns, while 32.5% of the pulps in the bridge retainer groups had become necrotic. There was a significantly higher percentage of pulp necrosis in anterior teeth that served as bridge abutments (54.5% of anterior abutment teeth examined). In general, however, the available evidence indicates that the effects of dental procedures on the pulp depend on the following factors.

The degree of inflammation of the pulp preoperatively

As stated previously, the dental pulp is compromised in its ability to respond to external irritants because it is enclosed in a noncompliant environment, and because it lacks collateral circulation. Thus the more severe the pulp is inflamed, the less it will be able to respond to further irritation, such as in the form of restorative procedures.[160]

Most research studies designed to evaluate the effects of restorative procedures (or materials) on the pulp are conducted on human or experimental animal teeth with normal pulp tissue. Furthermore, many of the animal research projects have been performed on anesthetized animals without the use of local anesthesia, which as stated previously, reduces PBF. Therefore the results of these studies may not reveal the true clinical effects of these procedures when the pulp is already inflamed by the carious lesion and PBF is reduced by local anesthetic with vasoconstrictors. In a study that evaluated the response of the pulp to capping procedures as a function of duration of exposure, it was shown that the pulp responds favorably to exposures of up to 24 hours after exposure, but not as favorably after longer periods of exposure to oral environment.[60] It may be that the longer exposure periods lead to the formation of a bacterial biofilm, which is difficult for the pulp immune responses to eliminate, and/or the extension of the infection so deep into the pulp to preclude healing. This is relevant in cases of aseptic mechanical exposures or teeth where the pulp is exposed by traumatic injuries for a brief duration. In these cases, the pulp usually responds favorably to VPT procedures.[7] Models of standardized pulp inflammation with chronic caries are not commonly used in determining the effects of

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dental procedures. Older clinical studies show an unfavorable long-term outcome of capping cases with carious pulp exposures[18,115]; however, newer studies in which tricalcium silicates were used show more favorable results in these cases.[28,166,186,230]

In the absence of severe spontaneous symptoms or pulp exposure, as indicated previously, the clinician currently cannot determine accurately the degree of preoperative pulp inflammation. Thus every effort should be done to minimize added irritation during restorative procedures, as it is possible that excessive irritation could convert the inflammatory status of the pulp from a reversible to an irreversible condition. In addition, the patient should always be advised of the possibility of pulp degeneration and the importance of follow-up.

The amount of physical irritation caused by the procedure

Physical irritation during restorative procedures such as from heat, desiccation, or vibration may adversely affect the dental pulp.

Heat

Restorative procedures such as cavity or crown preparation or curing of resins during direct fabrication of provisional restorations[288] may cause significant increases in pulp temperatures. It has been shown using primate models that an intrapulpal temperature rise of 10°C causes irreversible pulp pathosis in 15%, and a 20°C rise caused pulp abscess formation in 60% of teeth evaluated.[322] Several other older studies documented burns or severe inflammation in the pulp when cavity or crown preparations were performed without coolants (Figs. 14.14–14.16). However, a more recent study in which gradual controlled heat application over a large area of the intact occlusal surface of human unanesthetized teeth was employed, failed to corroborate these earlier findings.[15] In this study, an increase of intrapulpal temperature of about 11°C followed by 2- to 3-month evaluation did not show any clinical or histological changes in the pulp of any of the teeth evaluated. A heat increase in rat pulp tissue to 42°C and in vitro raised heat shock protein-70, which are known to be tissue protective, caused changes in alkaline phosphatase and gap junction proteins, that were reversed to normalcy a few

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hours later.[9] By contrast, heat applied in deep cavity preparations, prepared atraumatically in human teeth, in another study, caused histologic changes that were dependent on the proximity of the heat source to the pulp.[208] It was common in that study to see a loss of odontoblasts or their aspiration into the dentinal tubules. In cases where the cavity floor was less than 0.5 mm from the pulp, areas of coagulation necrosis could be seen, although the patients remained asymptomatic for the 1-month duration of the study. The measurement of heat in the tooth being prepared in areas other than the site of tooth preparation, occasionally shows reduction in temperature,[96] presumably because of the poor conductive properties of dentin and the cooling effect of compressed air of the high-speed hand-piece. Furthermore, cavity and crown preparations include several other irritating stimuli, such as desiccation, severance of odontoblastic processes, vibration, and smearing of bacterial irritants onto the surface of dentin. Therefore taken together, these findings suggest that the transient increase in temperature to levels relevant to modern dental procedures on its own may not be the culprit in inducing pulp changes. Rather, the synergistic application of excessive heat with other irritation factors and its proximity to the pulp may induce pathological changes.

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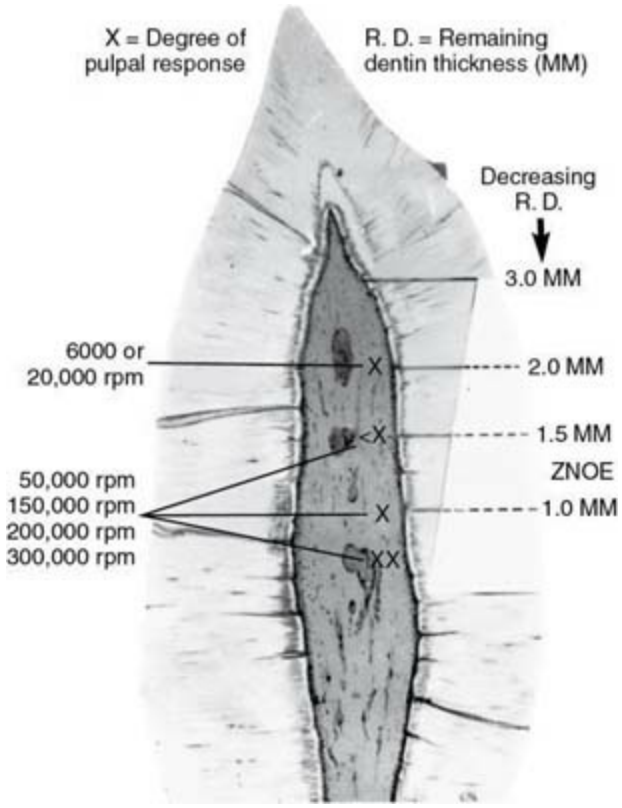

FIG. 14.14 With adequate water and air spray coolant, the same cutting tools, and a comparable remaining dentin thickness, the intensity of the pulpal response with high-speed techniques (i.e., decreasing force) is considerably less traumatic than with lower speed techniques (i.e., increasing force).

Tooth pulp with labels as follows: X5Degree of pulpal response, R. D.5Remaining dentin thickness (MM), decreasing R. D. leading to 3.0 MM, 2.0 MM, 1.5 MM ZNOE (from inner to outer layer), 1.0 MM, 300,000 rpm, 200,000 rpm, 150,000 rpm, 50,000 rpm, 6000 or 20,000 rpm (thickness of internal structures of pulp).

Source: (Reprinted with permission from Stanley HR, Swerdlow H. An approach to biologic variation in human pulpal studies. *J Prosthet Dent* 14:365, 1964.)

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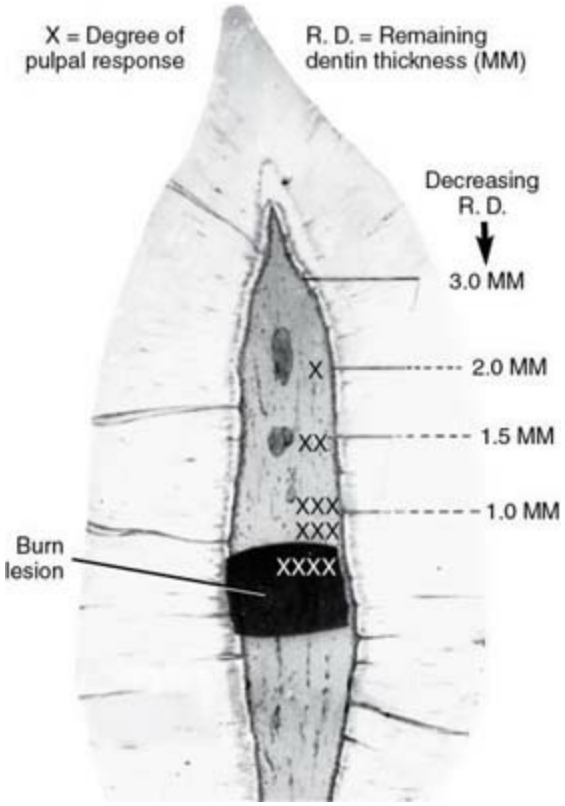

FIG. 14.15 Without adequate water coolant, larger cutting tools (e.g., no. 37 diamond point), create typical burn lesions within the pulp when the remaining dentin thickness becomes less than 1.5 mm.

Tooth pulp with labels as follows: X5Degree of pulpal response, R. D.5Remaining dentin thickness (MM), decreasing R. D. leading to 3.0 MM, 2.0 MM (X), 1.5 MM (XX), and 1.0 MM (XXX) (from inner to outer layer), and burn lesion.

Source: (Reprinted with permission from Stanley HR, Swerdlow H. An approach to biologic variation in human pulpal studies. *J Prosthet Dent* 14:365, 1964.)

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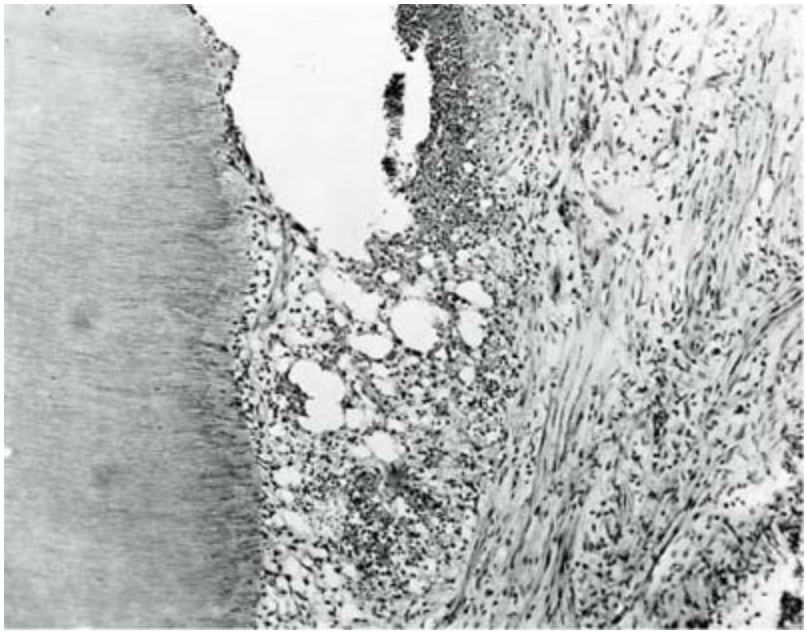

FIG. 14.16 Burn lesion with necrosis and expanding abscess formation in a 10-day specimen. Cavity preparation dry at 20,000 rpm, with remaining dentin thickness is 0.23 mm.

Micrograph shows a U-shaped cavity surrounded by dead cells and inflammation in pulp.

Source: (Reprinted with permission from Swerdlow H, Stanley HR, Jr. Reaction of the human dental pulp to cavity preparation. I. Effect of water spray at 20,000 rpm. *J Am Dent Assoc* 56:317–329, 1958.)

Desiccation

Desiccation during cavity and crown preparation has long been known to cause aspiration of odontoblastic nuclei into dentinal tubules and pulp inflammation.[33] One study showed that as little as 30 seconds of continuous air drying of class V cavities in human molars with uninflamed pulp caused significant displacement of odontoblastic nuclei, pulp inflammation and even areas of necrosis related to the areas that were dried.[59] However, another study showed that the effects of desiccation are transient and that within 7 to 30 days, there is autolysis of the aspirated cells and formation of reactionary dentin.[34] The pulp, in cases with aspirated odontoblasts following desiccation for 1 minute, was not sensitive to clinical scraping with an explorer. The sensitivity was restored with rehydration of the cavities and was increased in other cases where pulp inflammation was induced by microbial

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contamination.[168] In this study, despite the lack of sensitivity in desiccated cavities, neural elements were seen histologically to be pushed into the tubules, like the odontoblastic nuclei. The disruption of the odontoblastic layer and peripheral neural elements in the pulp with desiccation was also observed in a rat model using axonal transport of radioactive protein.[39] One clinical observational study showed that air cooling without water during crown and bridge preparations caused minimal damage to the pulp clinically or radiographically.[174]

Biological and chemical irritation

Dental caries is clearly a disease in which microorganisms and their virulence factors constantly irritate the pulp, even at the early stages, long before pulp exposure.[36] However, despite the elimination of visible caries during cavity preparation, the cavity floor is undoubtedly left with some contamination by caries bacteria. While the rubber dam should be used with any cavity preparation to prevent cavity contamination with salivary microorganisms, the use of water coolants allows the cavity to be contaminated with bacteria from water lines. Concerns about residual cavity contamination prompted some to use cavity disinfection with some caustic chemicals. Chemicals such as hydrogen peroxide, sodium hypochlorite, or calcium hydroxide solutions have been proposed for this purpose, although they may exert a toxic effect.[56] An earlier study showed that the amounts of residual bacteria following adequate restoration are not significant.[190] Once dentin is exposed, there is a constant outward flow of dentinal fluid that minimizes the inward flow of any noxious agents.[310] This may aid in the reduction of irritation from residual microbial factors in dentinal tubules.

In contemporary practice, most chemical irritation during restorative procedures results from the application of etching agents, especially strong acids, in the form of total dentin etch, particularly if capping of exposed pulp is performed.[98,219] Etching is performed to remove the smear layer, to promote physical adhesion of bonding agents to dentin by forming resin tags in the dentinal tubules, and to permeate the newer unfilled resin primers into the unmineralized surface layer of collagen to form the so-called “hybrid layer.”

If the cavity is relatively superficial and is adequately sealed with a

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restorative resin, then the etching of dentin is probably not detrimental to the pulp because of the narrow diameter of dentinal tubules and their low density in peripheral dentin.[30] In fact, one study documented that histological evidence of bacteria in human cavities restored with composite was significantly less if the cavity had been etched with phosphoric acid than if it was etched with 17% EDTA or nonetched.[197] Pulp inflammation in this study was not correlated with the etching treatments, but with bacterial presence; thus in cases of etching with phosphoric acid, if bacteria were also present, severe pulp inflammation and necrosis could be seen.

Self-etching formulations have become popular because they eliminate the separate etching step involved in total-etch procedures. Some have speculated that the bonding of self-etching systems may be poorer than total-etch systems because of the weaker acidity of the acidic primers of self-etching systems compared to total-etch systems.[30] However, studies have shown no significant differences between the two adhesive systems in long-term in vitro bond strength, postoperative sensitivity,[232] long-term in vivo degradation,[153] or long-term in vitro bond strength.[11] One clinical study showed no differences between the two systems with respect to bacterial leakage and the inflammatory response in the pulp.[202] The most important variable that affected the pulp in this study was the amount of bacterial leakage with either system.

Other factors that may contribute to pulp irritation during resin placement from chemical/biologic irritants include unpolymerized monomer and polymerization shrinkage. Higher concentrations of monomeric resin components were shown to exert an inhibitory effect on T-lymphocytes and spleen cells,[132] as well as monocytes/macrophages[162,240,241,261] in vitro. These components may leach directly into the pulp in deep cavities and cause chemical irritation.[118] Shrinkage during polymerization of composites may induce internal stresses on dentin and create voids that allow microleakage. Shrinkage of resins is estimated to range from 0.6% to 1.4% and should be minimized during placement by incremental curing and possibly starting the restoration with flowable resins.[30]

Recent studies have shown that salivary or dentin matrix-based metalloproteinases or salivary esterases may be released during etching and may degrade the collagen in the hybrid layer, thereby degrading the

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dentin/composite bone.[116,182] In vitro and in vivo research showed that this detrimental effect could be reduced and the stability of the hybrid layer maintained with the use of chlorhexidine, galardin, benzalkonium chloride, or quaternary ammonium methacrylates.[182]

In summary, the available evidence indicates that chemicals involved in modern restorative procedures may irritate the pulp if placed directly on an exposure, if there is microbial leakage along the tooth/restoration interface, or if there is degradation of the hybrid layer that seals the restoration dentin interface.

The proximity of the restorative procedures to the dental pulp and the surface area of dentin exposed

It has been known for several decades that, as the carious lesion progresses towards the pulp, particularly when the RDT is less than 0.5 mm, there is an increasingly severe pulp reaction, with a greater likelihood of the pulp undergoing irreversible pathosis.[245] The diameter and density of dentinal tubules increase closer to the pulp (Fig. 14.17). Based on the dentinal tubule density at the DEJ (about 65,000/mm^2) and the pulp (about 15,000/mm^2),[90,94] it was estimated that the area occupied by tubule lumina at the DEJ was 1% of the total surface area at the DEJ and 22% at the pulp.[225] Thus it is not surprising that several studies have shown that pulp inflammation in response to restorative procedures increases with the reduction in RDT.[1,61,196] One study examined the differential effects on the rat pulp of the preparation method, RDT, coolant, drill speed, conditioning with EDTA, and filling materials.[195] Subsequent to the cavity preparations a tooth slice was obtained and maintained ex vivo as an organ culture for up to 2 weeks. The results showed that the RDT was the most important factor on pulp injury.

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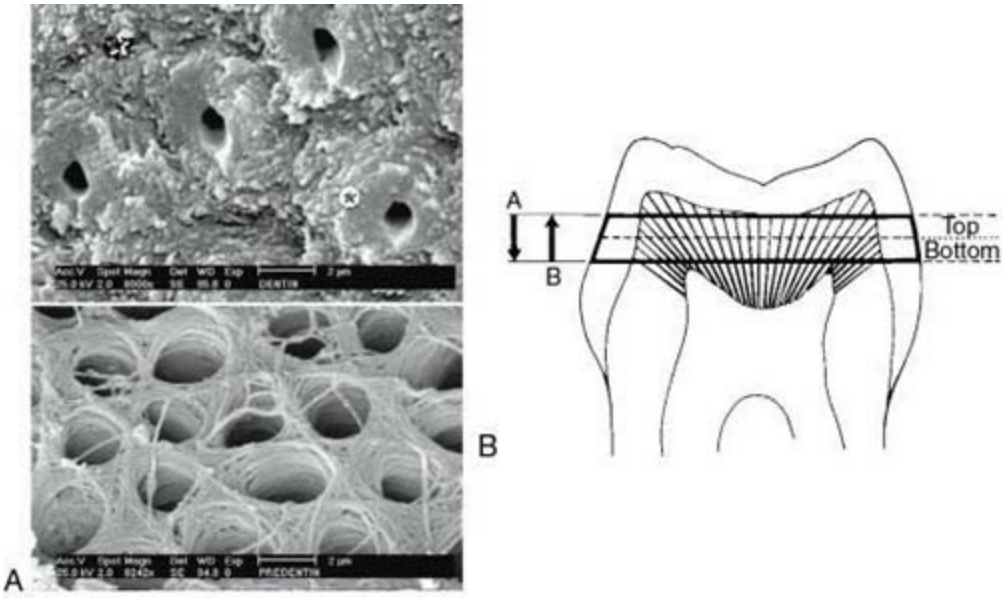

FIG. 14.17 Schematic of convergence of tubules toward the pulp. **A,** Periphery of the dentin. Most surface area is occupied by intertubular dentin (☆), with a few tubules surrounded by hypermineralized peritubular dentin (✪). **B,** Near the pulp, the increase in tubule diameter has occurred largely at the expense of the peritubular dentin. This substrate has high protein content. As the remaining dentin is made thinner (from A to B), the permeability of the dentin increases, because both diameter and density of dentinal tubules are increased.

A) Set of two SEM images shows the changes in dentin tubules.

B) Tooth crown highlight a layer marked A toward the bottom and B toward the top above pulp horns.

Source: (Reprinted with permission from Bouillaguet S. Biological risks of resin-based materials to the dentin-pulp complex. *Crit Rev Oral Biol Med* 15:47–60, 2004.)

With the passage of time following cavity preparation, there is a reduction in the permeability of RDT.[227] This may be due to the rapid deposition of reactionary dentin, the migration of large proteins into the tubules, and/or the diminution of tubule diameter as dentin becomes more sclerotic. Using a primate model, it was shown that the basic rate of secondary dentin deposition was about 0.8 µm/day and that this rate increased to an average of 2.9 µm/day following restorative procedures. Interestingly, in this study, dentin deposition was also more rapid next to shallow cavities than deep cavities[312]; however, another study showed that total reactionary dentin

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deposited was thicker in deeper and wider cavities.[194]

Clinically, it has been observed that postoperative sensitivity is common with many restorative procedures. Following resin composite restorations on patients, it was shown that postoperative sensitivity was related to the depth of the cavity but not to the presence or absence of liners or bases.[302]

In addition to the depth and/or the width of a large cavity preparation, a crown preparation exposes more dentinal tubules to microbial or chemical irritation. During crown fabrication, there are added irritation factors such as the length of time of preparation, impression techniques, and the imperfect adaptation of temporary restorations that cause microleakage during the temporization period. Due to the precise engineering requirements of some restorations, some providers may be inclined to reduce the coolant during crown preparations steps, such as finalizing the finishing lines. However, crown preparations without coolants have been shown to dramatically reduce PBF in an animal model (Fig. 14.18).[143] Air coolant appears to be adequate, at least in part.[174] In general, long-term outcome studies have documented that the incidence of pulp necrosis following crown placement ranges from 10% to 50%.[50,85,303]

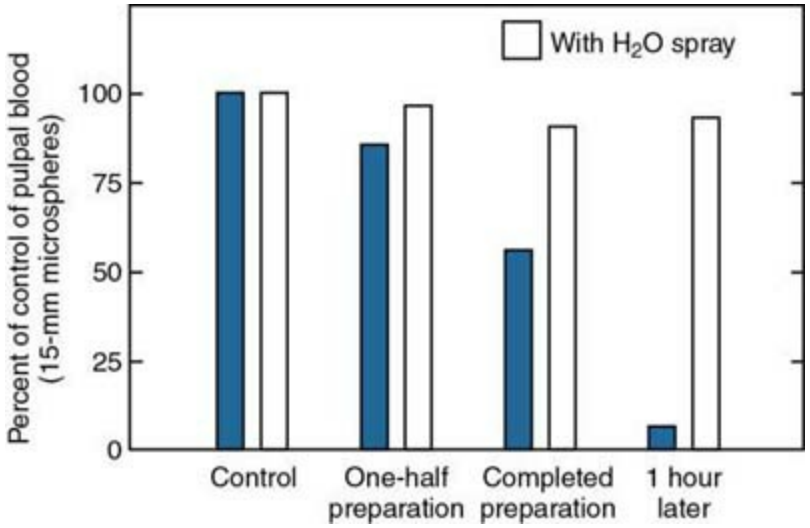

FIG. 14.18 Effects of crown preparation in dogs, with and without water and air spray (at 350,000 rpm) on pulpal blood flow. Tooth preparation without water and air spray caused substantial decrease in pulpal blood flow, whereas that with water and air spray caused insignificant changes in the flow

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Bar graph plots percent of control of pulpal blood (15-mm microspheres) with and without water spray and air spray. The vertical axis ranges from 0 to 100 in increments of 25. The data are as follows. Control (with water and air spray: 100, without water and air spray: 100); one-half preparation (with water and air spray: 85, without water and air spray: 98); completed preparation (with water and air spray: 55, without water and air spray: 95), and 1 hour later (with water and air spray: 10, without water and air spray: 96). All data are approximate.

Source: (Reprinted with permission from Kim S, Trowbridge H, Suda H. Pulpal reaction to caries and dental procedures. In: Cohen S, Burns RC, editors. *Pathways of the pulp,* ed 8, St. Louis, 2002, Mosby, pp 573–600.)

The permeability of dentin and the odontoblastic layer between the area being restored and the pulp

The permeability of dentin plays an important role in the ingress of potential irritants to the pulp. It is clear from research done in the last three decades that dentin is not uniformly permeable and that permeability depends on factors such as the location within the same tooth, the age of the patient, and the presence of pathological conditions such as dental caries. Fundamentally, the permeability of dentin depends on the collective sum of permeability of individual tubules at a specific site in the tooth. The tubular diameter increases from about 0.6 to 0.8 μm close to the DEJ to about 3 μm at the pulp.[222] Given that bacterial cells are about 0.5 to 1 μm in diameter, it is evident that in deep cavity preparations, particularly when total-etch procedures are employed, bacteria can migrate through the remaining dentin into the pulp.

With age, the width of peritubular dentin increases, causing reduction in tubular lumen or sclerosis. Caries causes demineralization in superficial dentin, which is associated with remineralization and the formation of caries crystals within the tubules of inner undemineralized dentin (Fig. 14.19). This causes a decrease in permeability in dentin subjacent to the carious lesion[224] and could be considered a protective mechanism, as it may delay the progress of the carious lesion.

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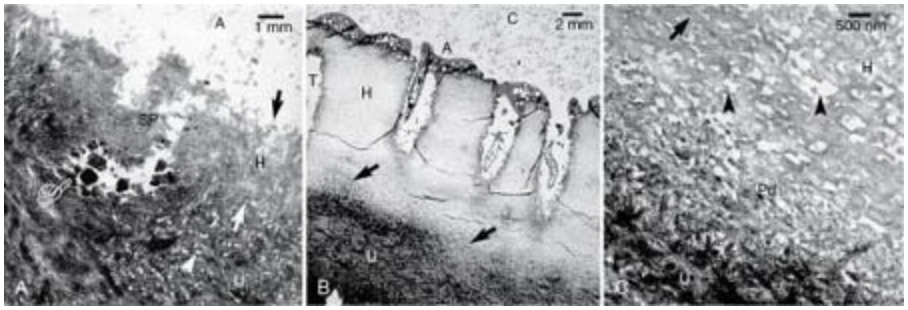

FIG. 14.19 Transmission electron microscope (TEM) images of undemineralized specimens of resin-bonded caries-affected dentin. **A,** Stained TEM image of undemineralized specimens following the application of the self-etch ABF system to caries-affected dentin. The hybrid layer (*H,* between *arrows*) was about 3 μm thick, and the underlying un-demineralized dentin *U* was highly porous *(arrowhead)*. The dentinal tubule was covered with a smear plug *(SP)* and was partially obliterated with large caries crystals (pointer). *A,* Filled adhesive. **B,** Stained section of the total etch Single Bond adhesive bonded to caries-affected dentin. A hybrid layer *H* between 15 and 19 μm thick could be seen, with a partially demineralized zone *(open arrows)* above the undemineralized caries affected dentin *U. C*, Composite; *T*, dentinal tubule. **C,** Higher magnification of the basal part of the unusually thick hybrid layer *H* shown in *A*. Banded collagen fibrils *(open arrow)* were separated by unusually wide and porous interfibrillar spaces *(open arrowheads)*. A partially demineralized zone *(Pd)* was present along the demineralization front. This zone was not seen in phosphoric-acid-etched sound dentin (B). *U*, Undemineralized caries-affected dentin.

Set of three TEM images marked A through C shows the cavities with cube-like structures affecting the dentin layer and resulting in the formation of hybrid layers on 1 mm scale, followed by the magnified views of hybrid layers on 2 mm and 500 nm scales, respectively.

Source: (Reprinted with permission from Yoshiyama M, Tay FR, Doi J, et al. Bonding of self-etch and total-etch adhesives to carious dentin. *J Dent Res* 81:556–560, 2002.)

It was shown that irritation from cavity preparation increased the permeability of the odontoblastic cell layer only at the site of the cavity preparation.[125,297] In addition to the physical barrier to permeability and the production of reactionary or reparative dentin, the odontoblastic layer may, in fact, contribute to the host response of the dental pulp by expressing important inflammatory mediators[165,218] or recognize bacteria through TLRs.[29,71,80,131]

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The age of the patient

Resting PBF, as well as the changes in PBF in response to cold application, decreases with age.[121] Age may also be associated with a reduction in pulp neuropeptides.[91] However, studies show no differences between young and old pulp in the regenerative capacity of odontoblast-like cells, nor in the presence of cells positive for class II major histocompatibility complex, heat shock protein 25 or nestin, when subjected to cavity preparation.[137] An examination of normal, young versus old human pulp showed that young pulp had an increased expression of biological factors related to cell differentiation, proliferation, and the immune response; however, older pulp had increased factors related to apoptosis.[293] Nevertheless, PBF in anterior teeth measured with laser Doppler flowmetry showed that it correlates with systolic blood pressure, but not with age, sex, or tooth type.[140] Taken together, these analyses cannot translate to the pulp's ability to deal with irritation and its sequelae. Thus the net result of the ability of the pulp to cope with external stimulation or irritation in humans with advancing age is not clear.

Pulp reactions to restorative materials

The effects of restorative materials on the dental pulp have been investigated and seem to relate directly to the permeability of the associated dentin. The degree of dentin permeability, however, is often variable and is governed by several factors, including age and caries status.[284] The most important variable in dentin permeability to restorative materials is the thickness of the dentin between the floor of the cavity preparation and the pulp.[1]

Given the importance of dentin permeability, there are direct effects of any given restorative material on the pulp that is governed by the composition of the material and associated eluted or degraded products. Unbound components of resin materials and preparative agents such as acid etchants can affect the subjacent pulp by inducing an inflammatory response.[93,98,239] The indirect effects of desiccation and/or demineralization of dentin as well as the direct effects of the material itself when in contact with pulp tissue mediate this inflammatory response. Studies have shown that the certain cytotoxic components of resin monomers (e.g., triethylene glycol

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dimethacrylate and 2-hydroxyethyl methacrylate) readily penetrate dentin.[87] Similarly, eugenol and components of Ledermix (triamcinolone and demeclocycline) have been shown to pass through dentin into the subjacent pulp.[117,120] In vivo data show that these chemicals have an effect on the pulp; however, the effect seems to be short lived and, in the absence of bacteria, reversible.[23]

The mechanisms whereby restorative materials exert an injurious effect on the dental pulp are varied. Evidence exists that supports direct and, in some instances, prolonged cytotoxicity, stimulation of hypersensitivity reactions, or impairment of the host immune response to bacteria.[258] Some of the components of resin restorations are released at cytotoxic levels after polymerization is completed, leading to chronic stimulation and a resultant prolonged inflammatory response.[86] Furthermore, even subtoxic concentrations of certain agents are capable of eliciting allergic reactions in humans.[119] Primates hyperimmunized with BSA showed significant pulp damage with repeated antigenic challenge in class V cavity preparations, suggesting a role for antigen-antibody complex–mediated hypersensitivity in tissue destruction.[26] In a separate study, exposure to dentin primers elicited a delayed-type hypersensitivity reaction in guinea pigs.[136] These studies, taken together, present a compelling argument for immune-mediated pulp tissue damage subsequent to exposure to restorative materials. Foreign body reactions have also been described in pulps containing extruded globules of resin material.[123,124] Histological examination of such pulps shows macrophages and giant cells surrounding the resin particles. Lastly, resin monomers have been shown to decrease the activity of immunocompetent cells in a dose-dependent manner in in vitro functional assays.[132] While all these effects are documented, their extent and therefore morbidity on the dental pulp is speculative and doubtless does not act solely to effect pulp demise. As previously noted, most restorative materials are placed adjacent to pulps that are previously compromised by bacterial insult and that disease, debridement, and restoration of the tooth have cumulative effects on the dental pulp.

Although pulp irritation is largely considered to be a negative sequela, the irritant potential of certain restorative materials is central to their usefulness in restorative dentistry. Calcium hydroxide is one of the oldest and most

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widely used medicaments for stimulation of dentinal bridge formation subsequent to microscopic or gross pulp exposure. The low-grade pulp irritation that it induces is important for dentinal bridge formation in exposures.[64,260] The degree of inflammation is dependent on the preparation of calcium hydroxide used. Aqueous suspensions of calcium hydroxide applied to exposed pulps cause superficial necrosis of pulp tissue followed by low-grade inflammatory changes. Within 30 days, the tissue subjacent to the necrotic zone has reorganized and resumed normal architecture. Hard setting calcium hydroxide preparations as well as mineral trioxide aggregates (MTAs) are effective in eliciting dentinal bridge formation with a much smaller to nonexistent necrotic zone.[108] This is preferable in vital pulp therapies such as the Cvek pulpotomy where maintenance of the maximum amount of vital pulp tissue is desirable and the extent of pulp inflammation is minimal (Fig. 14.20).[63] The irritation potential of calcium hydroxide across intact dentin is dependent on factors such as the RDT and permeability. Application of calcium hydroxide to intact dentin appears to induce sclerosis by promoting crystal precipitation within the tubules accompanied by reductions in permeability.[192] However, more recent findings show that calcium hydroxide over intact dentin does not exert a significant clinical advantage and may be lost due to the action of leakage or dentinal fluid.[62]

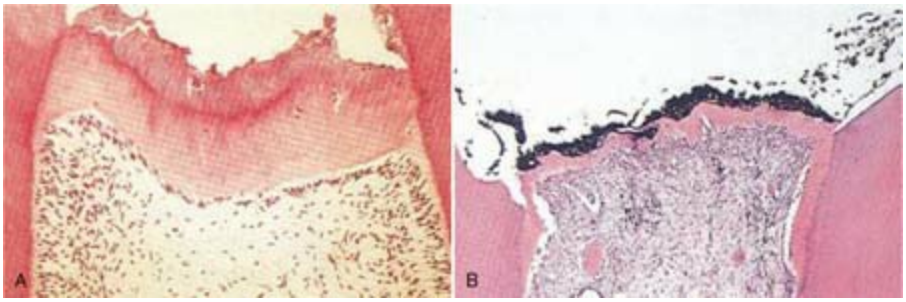

FIG. 14.20 Calcium hydroxide produces an inflammatory response that stimulates dentinal bridge formation. The dentin bridge forms lower in the tooth with calcium hydroxide paste **(A)** versus hard setting calcium hydroxide **(B)**.

Set of two stained micrographs marked A and B shows the formation of calcium hydroxide paste and calcium hydroxide hard tissue in tooth, respectively.

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While the irritation potential of calcium hydroxide plays a role in its effectiveness, the high pH of this material can act to liberate bioactive molecules from dentin. Numerous authors have demonstrated that dentin matrix proteins like TGF-β1 and adrenomedullin are liberated by both calcium hydroxide and MTA.[97,291] Once liberated, they are able to facilitate hard tissue formation yet again. This offers another explanation for the ability of these materials to induce hard tissue formation in vivo. Acid etching or the use of chelating agents such as EDTA represent a common step in the placement of bonded restorations. The etching process removes minerals and exposes collagen fibrils. As with a high pH, sequestered bioactive molecules are released and can act on pulp cells. These include growth factors and metalloproteinases, which can be inductive for healing but can also degrade the bond of the restoration.[228] EDTA has been shown to be even more effective at extracting noncollagenous proteins from dentin than either calcium hydroxide or MTA, but less so than citric acid.[97,291]

Investigations into the pulp response to glass ionomer materials have shown that both the lowered pH of the material and the release of high concentrations of fluoride can inflict damage to pulp tissues.[135] In direct contact with pulp cells, glass ionomers are toxic.[155] Direct pulp capping studies have shown it to be inferior to calcium hydroxide.[67,112] When applied to intact dentin and in the absence of bacteria, there is a transient inflammatory response and reparative dentin is formed.[57]

In addition to the direct chemical effects of restorative materials, there are indirect factors that contribute to pulp irritation. The technique sensitivity of certain materials predisposes them to faulty bonds to tooth structure that can translate to dentin hypersensitivity, recurrent disease, and pulp inflammation or necrosis. Much attention has been given to the interface created between resin-bonded materials and dentin. During the etching process, the more highly mineralized peritubular dentin is preferentially dissolved, leaving free collagen fibrils and opening lateral tubular branches.[98,99,191] Applied resin infiltrates the exposed collagen mesh creating a layer 5 to 10 μm thick referred to as the hybrid layer.[199] This layer, along with the resin permeating exposed tubules, forms the bond between the resin and dentin. If the preparation is too dry, the collagen fibrils collapse and the resin cannot effectively permeate the mesh, which results in a defective bond. As the

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optimal degree of hydration of the preparation surface can vary from material to material, resin restoration placement is technique-sensitive. This same principle is applicable to the practice of bonding fractured tooth fragments where the segment has become dehydrated while outside of the mouth. Current protocols recommend rehydration of the segment prior to bonding, thus increasing the mechanical and, presumably, the microbial seal.[84] This is particularly important with a complicated crown fracture where the pulp protection by intact dentin is absent.

Some restorative materials rely on their medicinal properties as well as their ability to seal a cavity preparation. Materials containing zinc oxide and eugenol (ZOE) fall into this category. ZOE is used for a variety of purposes in dentistry largely because of its anesthetic and antiseptic properties. It has been shown to block the transmission of action potentials in nerve fibers and to suppress nerve excitability in the pulp when applied to deep excavations.[295] In addition, ZOE has good adaptation to dentin and inhibits bacterial growth on cavity walls. These properties have made it a favored material for temporary fillings, but not long-term restorations, as ZOE temporaries have been shown to leak after only a few weeks in situ.[323]

Direct pulp capping with bioceramics

The direct capping of pulp exposures is indicated in pulps that were previously healthy and exposed by trauma or dental restorative procedures and, more recently, for pulp exposure where the diagnosis is reversible pulpitis.[4,279,283] This is particularly true in the cariously exposed immature permanent tooth, where maintenance of pulp vitality is crucial to further tooth development. Although calcium hydroxide has historically been the preferred dressing agent on mechanically exposed pulps, the use of MTA, Biodentine, and other bioceramic formulations has recently been proposed, even on carious pulp exposures.[17,28,109,186] Prospective animal studies and human case reports have evaluated the ability of MTA to allow for the formation of a reparative dentin bridge and to maintain continued pulp vitality.[79,89,148] Although the results are generally favorable, one concern is of tooth discoloration in cases where the gray MTA formulation is used on anterior teeth. Recent studies have indicated that newer bioceramic materials show

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less potential for discoloration when used as pulp capping materials.[149]

The bioceramic that has been most intensely studied for pulp capping is MTA. In one clinical study, MTA was used as a pulp capping material for carious pulp exposures.[28] Forty patients, ages 7 to 45, who were diagnosed with reversible pulpitis had caries removed using a caries detection dye and sodium hypochlorite for hemostasis. The treatment was performed in two visits to allow the MTA to set up and to confirm pulp sensibility to pulp tests in the second visit. Success was determined radiographically, with subjective symptoms, pulp testing with cold, and continued root formation on immature teeth. Outcomes were measured over a period of up to 9 years postoperatively and showed an overall success rate of 97%, with all the teeth in the immature root group showing success. In another clinical study in which dentists and students participated in the treatment, pulp capping of carious pulp exposures was compared between MTA and calcium hydroxide.[186] At follow-up, 122 cases of patients of mean age of 40 years were available. The success with MTA was 78% and, with calcium hydroxide, success was 60%, a difference that was statistically significant. Within the parameters of these studies, it seems that white MTA is a suitable capping agent for pulp exposures of healthy or reversibly inflamed pulps, including cases with asymptomatic carious exposures. Randomized trials and consensus reports have shown that tricalcium silicates are significantly better than calcium hydroxide as pulp capping agents in carious pulp exposure.[69,157] However, given that the prognosis of root canal therapy in this last category has been shown to be predictably high in many studies, the patient needs to be educated carefully of the options, and consent must be documented.

As previously mentioned, other calcium silicate-based materials are gaining popularity as pulp capping agents and appear to have similar properties and effects as MTA.[12,205] Biodentine deserves special reference because, in addition to the biocompatibility, it has physical properties that are very similar to dentin.[41,154] Studies show no differences between MTA, Biodentine, and other tricalcium silicates like NeoMTA Plus as pulp capping or pulpotomy agents.[134,206,290]

The use of hemostatic agents and

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disinfectants on direct pulp exposures

There is still debate as to whether the outcomes of pulp capping for exposure depends on the toxicity of medicaments and materials placed on vital pulp tissues, the ability of these materials to induce mineralization, or their ability to seal the cavity from further bacterial ingress. It is likely a combination of these factors, as was shown in the recent clinical trial with MTA mentioned before.[28] Another factor in the prognosis of direct pulp caps is the ability to control hemorrhage at the exposure site.[273] Given the difficulty in creating a bacteria-free operating environment during tooth preparation, the ideal hemostatic agent also would have the ability to kill bacteria.

One study compared the effects of two hemostatic/disinfectant agents on the healing of experimental pulp exposures created in human third molar teeth and capped with calcium hydroxide.[264] Pulp exposures were made in 45 maxillary wisdom teeth scheduled for extraction for orthodontic reasons. Teeth were randomly assigned to receive hard-setting calcium hydroxide pulp caps after a 30-second surface treatment with one of three agents: 0.9% saline, 2% chlorhexidine, or 5.25% sodium hypochlorite. Although the 7-day saline specimens showed slightly less inflammatory response, there were no statistically significant differences between the groups with respect to all dependent measures over the course of the study. Complete healing was seen in 88% of all specimens at 90 days.

The pulps in these teeth were previously uninjured, and the exposures were made in a clean environment. Carious pulp exposure frequently reveals an inflamed pulp in which it is difficult to stop the bleeding. The original study by Bogen et al. proposed applying a cotton pellet with 5.25% to 6% sodium hypochlorite for 1 to 10 minutes for hemostasis.[28]

Pulp reactions to laser procedures

Numerous studies have been published on the effect of using lasers on enamel, dentin, and pulp. Laser use on hard tissue has been a popular area of research because of the potential benefits of efficiency, reduced sensitivity, disinfection, and precision. There are several different types of laser technologies available that depend on the wavelength, active medium, emission mode, delivery system, power output, and duration of application.

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The main types available in dentistry today are shown in Fig. 14.21. The CO_2 laser is historically the oldest type used on soft tissues and thus has been the most studied. It has the longest wavelength (10,600 nm). It cannot be delivered in an optic fiber and thus must be used in a hollow tubelike wave guide in continuous gated-pulse mode. This means that the operator does not feel a solid resistance when using this laser. Er:YAG, Nd:YAG, or Ho:YAG lasers all have an active medium of a solid crystal of yttrium-aluminum-garnet, which is impregnated in erbium, neodymium, or holmium, respectively. Er:YAG has a wavelength of 2940 nm and is delivered using a solid optic fiber. It has a high affinity for water and hydroxyapatite, which means it can be used for removal of caries and cutting dentin with coolant. It can also be used on soft tissue. The Ho:YAG laser has a wavelength of 2120 nm, has a high affinity for water but not to tooth structure, and thus is used primarily for soft-tissue surgery. Nd:YAG laser is also delivered fiberoptically, has a wavelength of 1064 nm, and has been used extensively in dentistry because it has a high affinity for water and pigmented tissues and offers good hemostasis; therefore it is used extensively in surgery.[54] In addition, there are some low power output lasers such as HeNe or helium neon (632 nm), and GaAlAs or gallium-aluminum-arsenide (diode; semiconductor) (720 to 904 nm) lasers that have been used in laser Doppler flowmetry and in treating dentin hypersensitivity.[144]

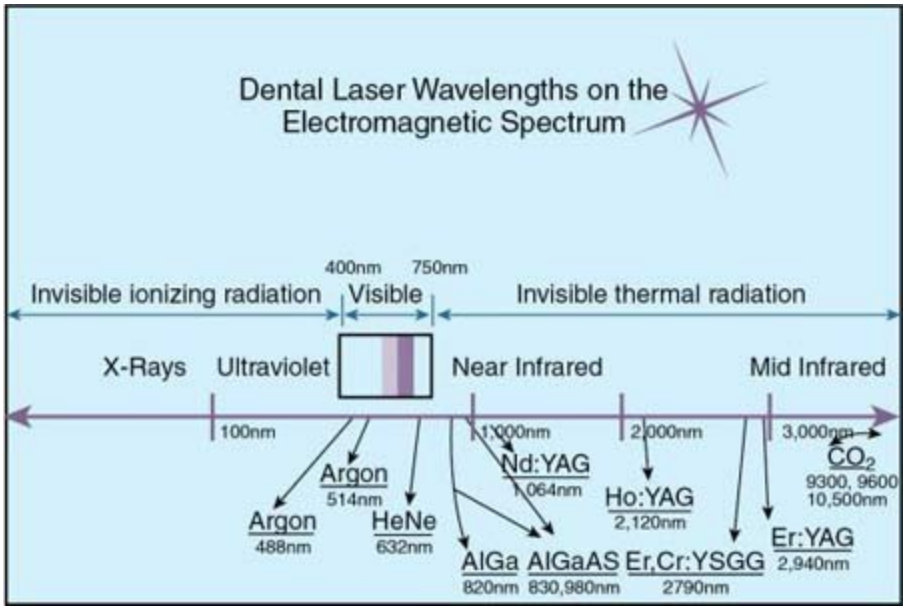

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FIG. 14.21 Currently available dental wavelengths on the electromagnetic spectrum. Note all of the wavelengths are nonionizing.

In dental laser wavelengths on the electromagnetic spectrum, the invisible ionizing and invisible thermal radiation extend toward the left and right of visible region (400 to 750 nanometers), respectively. The visible region marks argon at 488, argon at 514, HeNe at 632 nanometers. Region from near infrared to mid infrared shows AlGa at 820, AlGaAS at 830, 980, ND: YAG at 1,064, Ho: YAG at 2,120, Er,Cr: YSGG at 2790, Er:YAG at 2490, and CO2 at 9300, 9600, and 10, 500 nanometers.

Source: (Reprinted with permission from Coluzzi DJ. An overview of laser wavelengths used in dentistry. *Dent Clin North Am* 44:753–765, 2000.)

In order to summarize the available data, a discussion of the application of lasers for two specific purposes will follow.

Lasers in the prevention, diagnosis, and treatment of caries

Laser irradiation of deep susceptible pits and fissures may reduce the incidence of dental caries. Once caries develops, some lasers may be effective in removing the carious lesion and sparing undemineralized dentin because of their differential absorption by water and hydroxyapatite. Furthermore, if caries exposes the pulp in young teeth, particularly those with immature apex, lasers may be able to effectively excise coronal infected pulp in pulpotomy because of their hemostatic and antibacterial properties. All these potential uses prompted a large number of investigations on the effectiveness of lasers in these applications.

Some clinicians have proposed using lasers to enhance adhesion of pit and fissure sealants; however, this was shown to not add any advantage following acid-etching—a necessity for adhesion.[177] Laser fluorescence is used by the DIAGNOdent and DIAGNOdent pen, which are laser devices that have been introduced for the diagnosis of non-cavitated caries. Although these devices initially showed some promising results,[178] more recent work shows that they are best used as adjunctive devices to radiography and visual examination.[8,55,156]

Laser ablation of superficial carious lesions may be more conservative than bur preparation. A recent controlled clinical trial supports this tenet, when

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free-running pulsed Nd:YAG laser was used to ablate superficial pit and fissure caries in third molars scheduled for extraction (Fig. 14.22).[103] In this study, there were no histologic differences in the pulp response between the two groups.

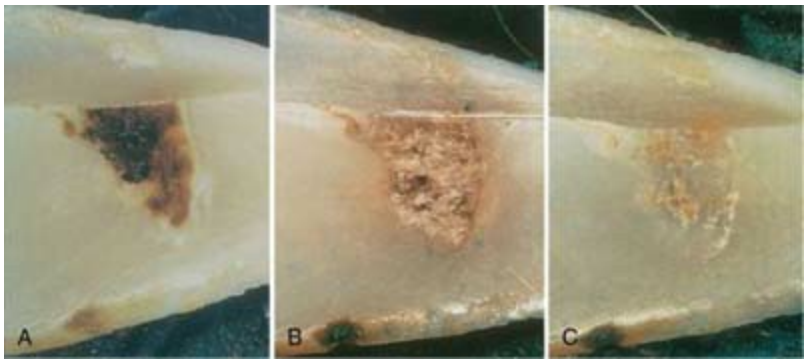

FIG. 14.22 **A**, Dental caries. **B**, Pulsed Nd:YAG ablation by-products (160 mJ, 10 Hz). **C**, Debris removed with acid etch and polishing. Enamel surface was faceted for reflection spectroscopy.

Set of three photographs marked A through C the removal of black tissue of dental carries using Nd: YAG laser.

Source: (Reprinted with permission from Harris DM, White JM, Goodis H, et al. Selective ablation of surface enamel caries with a pulsed Nd:YAG dental laser. *Lasers Surg Med* 30:342–350, 2002.)

From the perspective of the effects on the pulp, most laser applications that are employed in cutting or modifying cavities in dentin or acting directly on the pulp tissue are important. Earlier studies showed reduced permeability of dentin in vitro with an XeCl excimer laser (a laser with a relatively short wavelength of 308 nm in the ultraviolet range).[272] The apparent fusion of tubules in superficial layers of dentin was shown to occur with CO_2, Nd:YAG, and Er:YAG lasers in vitro.[314] The pulp responses to Nd:YAG and CO_2 lasers were not favorable. It was shown that Nd:YAG, and to a lesser degree CO_2 lasers, may be associated with charring and significant inflammation in the pulp compared with Er:YAG laser (Fig. 14.23).[289,314] More recently, it was reported that water cooling was necessary for laser ablation as it is for high-speed bur preparations.[45] Studies have shown that Er:YAG laser appears to induce similar responses in the pulp to those seen

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with high-speed bur preparations, at the level of light microscopy analysis.[78,285,286,314] However, the findings with electron microscopy were different: it was reported that while shallow cavities ablated in rat molars using Er:YAG lasers did not show changes from base-line using light microscopy and transmission electron microscopy showed disruption and degeneration of pulp peripheral nerve endings and of myelin sheath in the immediate postoperative period (Fig. 14.24).[122] This may explain the reduced sensitivity that accompanies laser cavity preparations. Thus in summary, it does not appear that the use of lasers provides predictable advantages in cavity preparation compared with traditional methods at this time.

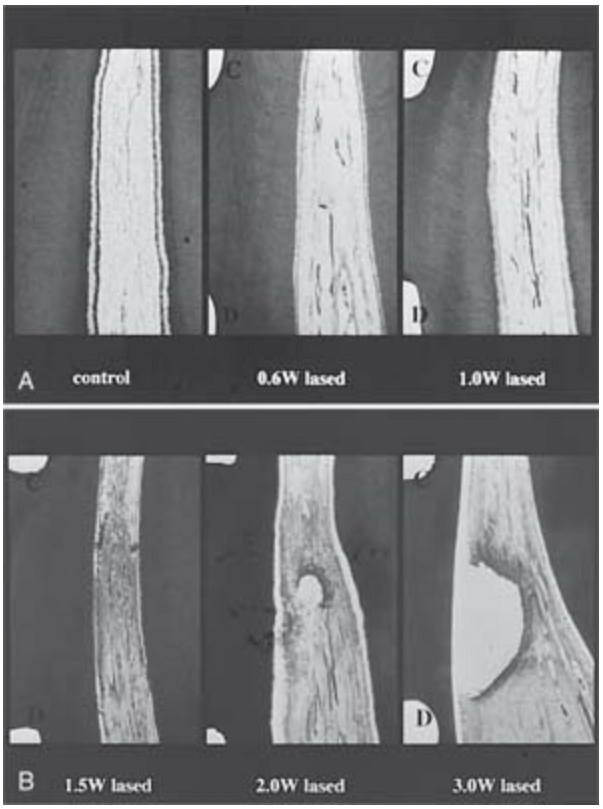

FIG. 14.23 **A** and **B,** Histopathologic picture of Nd:YAG laser specimen with an increasing power. There is direct relationship between the degree of pathologic changes and the increasing power of the laser. In fact, 1.5 W and the greater power cause permanent damage to the pulp.

A) Three images, marked control, 0.6 W lased, and 1.0 W lased show the moderate damage to pulp.

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B) Three images, marked 1.5 W lased, 2.0 W lased, and 3.0 W lased show the increasing damage to pulp that results in removal of tissue.

Source: (Reprinted with permission from Kim S, Trowbridge H, Suda H. Pulpal reaction to caries and dental procedures. In: Cohen S, Burns RC, editors. *Pathways of the pulp,* ed 8, St. Louis, 2002, Mosby, pp 573–600.)

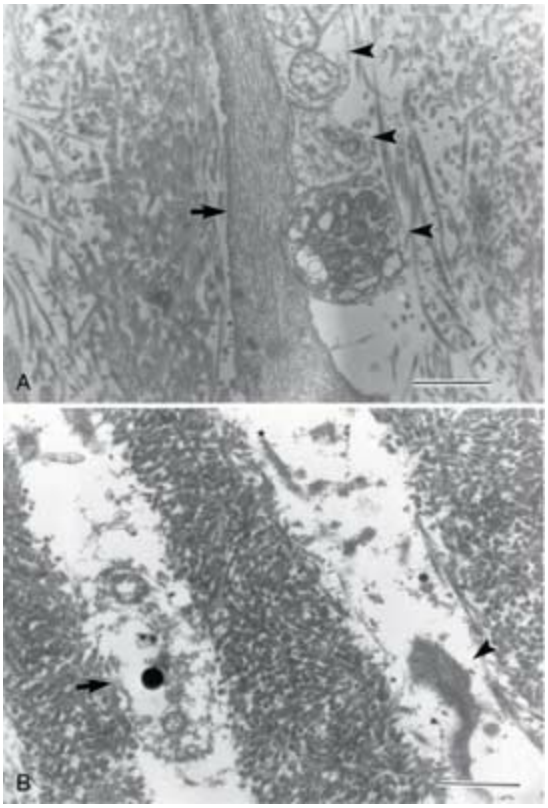

FIG. 14.24 **A,** Control; normal odontoblastic process *(arrow)* and a few nerve terminals *(arrowheads)* are seen in a dental tubule of rat upper first molar. **B,** Six hours after Er:YAG laser irradiation; disrupted cell membrane of a nerve terminal that contains some granular vesicles *(arrow)* and shrinkage of an odontoblastic process *(arrowhead)* are noted in dentinal tubules just under the ablated area. An asterisk indicates the irradiated side. *TEM,* ×13,700; *bar,* 1 μm.

Set of two TEM images marked A and B shows the changes in columnar odontoblasts and nerve terminals after using Er:YAG laser.

Source: (Reprinted with permission from Inoue H, Izumi T, Ishikawa H, Watanabe K. Short-term histomorphological effects of Er:YAG laser irradiation to rat coronal dentin-pulp complex. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:246–250, 2004.)

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For pulpotomy procedures, such as in primary teeth, permanent teeth with immature apex, or pulps that are exposed due to fracture and treated promptly, the lasers, particularly CO_2 lasers, may be useful in achieving precise surgical excision of coronal pulp and immediate hemostasis. A controlled clinical study showed that CO_2 laser pulpotomy was comparable to traditional methods in experimental pulpotomies of primary teeth scheduled for extraction for orthodontic reasons.[75] However, an animal study in which CO_2 and Nd:YAG lasers were compared revealed poor response with both lasers compared with calcium hydroxide.[133] An animal study using Er:YAG laser showed that the results are dependent on the power settings, in that lower energy delivered with this laser produced favorable results.[145]

Lasers in the treatment of dentin hypersensitivity

Earlier studies have shown effectiveness ranging from 5% to 100% of low output lasers on dentin hypersensitivity.[144] One author reported a reduction of hypersensitivity in 73% of mild cases, 19% of moderate cases, and 14% of severe cases after 4 months, using a GaAlAs laser.[144] Low output lasers do not have any effects on the morphology of enamel or dentin but are thought to cause transient reduction in action potential mediated by pulp C-fibers, but not Aδ-fibers,[311] although this finding was not consistent.[214] Nd:YAG lasers have also been used in dentin hypersensitivity. Because of the higher power output, these lasers cause superficial occlusion of dentinal tubules of up to 4 μm,[173] in addition to action potential blockage within the pulp in vitro or in experimental animals.[213,214] However, a placebo-controlled clinical trial has shown that both Nd:YAG and placebo caused significant reduction in dentin hypersensitivity for up to 4 months postoperatively, but were not different from each other.[167] More recent clinical trials show that while lasers are useful for managing hypersensitivity, they do not appear to be better than less costly and more readily available alternatives.[88,301]

Pulp reactions to vital bleaching techniques

Vital bleaching techniques employ the use of strong oxidizing agents,

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namely, 10% carbamide peroxide and hydrogen peroxide, to bleach the enamel of teeth with vital pulp. There have been concerns about the potential for pulp irritation during these procedures because of the long amount of time during which the chemicals are in contact with the teeth, particularly if dentin with open tubules or cracks are present. Histologic or histochemical analysis of the pulp following bleaching for up to 2 weeks showed minor inflammatory changes in the pulp of the bleached teeth that were reversible.[10,92] One clinical report documented that if 16% carbamide peroxide was used, gingival irritation was evident; however, no changes in pulp vitality or in symptoms were noted. Even in patients who develop symptoms post-operatively, these tend to be reversible and can be prevented by treating the teeth with fluorides and by correcting restorative deficiencies preoperatively.[201] Clinical symptoms are likely to be due to increases in neuropeptides, such as SP, in the pulp.[46] An earlier clinical trial showed that vital bleaching using 10% carbamide peroxide in a custom tray for 6 weeks was safe for the pulp health for up to 10 years postoperatively, although the bleaching effectiveness may decline with time.[250] In one recent clinical study premolars scheduled for orthodontic extraction had 38% H_2O_2 bleaching gel with and without a halogen light source applied. There were no histologic effects on the dental pulp at 2 to 15 days after bleaching.[146] When the study was performed on incisors with the same treatment for 45 minutes, areas of coagulation necrosis could be seen in the pulp.[58] Therefore caution should be exercised when this caustic agent is used for extended bleaching.

Light activation of bleaching agents is widely used. In conjunction with a source of peroxide moieties, the light emission results in both thermal and photo catalysis of the bleaching agent, thereby releasing hydroxyl radicals and facilitating the whitening process.[169,176] The light sources can be halogen light or diode lasers, and they are reported to speed the bleaching process, allowing shade improvement over a shorter period. Some of these techniques, however, have been shown to generate sufficient heat to result in a rise in pulp tissue temperature, increasing the risk of thermal tissue damage. To this point, comparisons of bleaching with and without light activation report a higher incidence of tooth sensitivity in the light-activated bleaching group.[147] Pretreatment with a desensitizing gel of 5% nitrate potassium/2% sodium fluoride was effective in one study in reducing postoperative sensitivity after

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light-activated bleaching.[246]

Pulp reactions to periodontal procedures

In an intact non-traumatized nontraumatized existence throughout life because it is insulated from microbial irritation in the oral cavity. Periodontal disease causes attachment loss, which exposes the root surface to the oral cavity. Occasionally, pulp inflammation secondary to severe periodontitis is observed.[161] Some reports have described bacterial infiltration through dentinal tubules of exposed root surface causing mild inflammatory changes in the pulp (Fig. 14.25).[159] However, in a case report of 25 teeth that were extracted in a patient with extensive periodontal disease, none of the pulp tissue had significant inflammatory changes.[292] It is much more common for pulp necrosis, or for failure of healing of periapical lesions, to present clinically with signs of periodontal disease than for periodontal disease to cause pulp pathosis. Primary endodontic, secondary periodontal pathosis is particularly evident if a perforation occurs in the pulp chamber or coronal third of the root during endodontic treatment and is not promptly treated, in cases of cracked teeth/vertical root fractures or in cases of congenital tooth defects such as palatal groove defects. Thus it is more likely for microbial irritants to move outward from a necrotic pulp to cause periodontal breakdown than for them to move inward from a periodontal pocket to cause irreversible pathosis in the vital pulp. The reasons for this observation are not fully understood. However, if the assumption is that bacteria may migrate through patent dentinal tubules in these situations, as is shown in Fig. 14.25, then it may be that the outward dentinal fluid flow in teeth with vital pulp contribute to the resistance to ingress of bacteria in sufficient amounts to cause a clinically significant disease process. Once the pulp degenerates, dentinal fluid flow no longer exists. Thus microbial irritants from the pulp may promote pocket formation and periodontal bone loss,[129] and the prognosis of endodontic and periodontal treatment may be related,[130] as the microbial factors may pass across dentin more readily.

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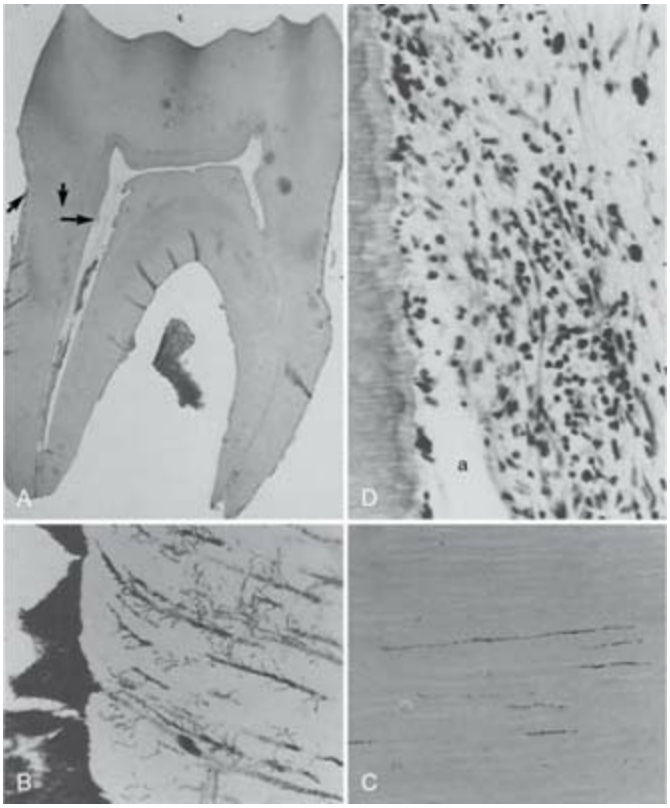

FIG. 14.25 Mandibular first molar of 49-year-old female, no pain. Clinically caries free. Calculus, periodontal disease, and bone loss from two thirds to three fourths of root. **A,** Note narrowness of pulp chamber seen throughout all serial sections (original magnification ×8.5). **B,** Bacterial plaque and adjacent dentinal tubules with bacteria (*oblique arrows* in **A**) (original magnification ×400). **C,** Farthest penetration of bacteria in dentinal tubules (*vertical arrow* in **A**) (original magnification ×400). **D,** Where dentinal tubules invaded by bacteria terminate in the pulp (*horizontal arrow* in **A**), a small but dense accumulation of lymphocytes and macrophages. *a,* Artifact; pulp tissue torn away from dentin during processing. (Original magnification ×400.)

Set of four micrographs marked A through D shows the removal of pulp canal after the penetration of bacteria through dentin in pulp.

Source: (Reprinted with permission from Langeland K. Tissue response to dental caries. *Endod Dent Traumatol* 3:149–171, 1987.)

Periodontal scaling and root planning may result in removal of cementum and exposure of dentin to the oral cavity. Frequently, this treatment results in dentin hypersensitivity, as discussed previously. In theory, periodontal disease and its treatment should be associated with increased incidence of pulp pathosis. In an older study, investigators induced periodontal disease in

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a primate model using ligatures and compared the effects on the pulp of periodontal disease with or without scaling.[24] In teeth with periodontal disease, mild chronic inflammatory changes were observed in 29% of the teeth and could be seen in areas of the pulp related to bone loss. One of 40 teeth developed pulp necrosis. In teeth that received scaling, a similar percentage of 32% developed the same mild inflammation and none developed pulp necrosis. In a later clinical study,[25] 52 periodontitis patients who had 672 teeth with vital pulp were followed up and maintained every 3 to 6 months for 4 to 13 years (mean 8.7 years). Of those teeth, 255 were bridge abutments. The results showed a significantly higher chance of pulp complications in teeth that were bridge abutments than teeth that were not abutments (15% vs. 3%; $P < .01$). Considering that both types of teeth had similar degrees of periodontal disease, the authors concluded that prosthodontic treatment is associated with pulp involvement more frequently than periodontal disease and its treatment. Another histologic analysis of 46 teeth with varying degrees of periodontal disease and coronal restorations reached a similar conclusion.[65] Furthermore, two comprehensive reviews of the topic concluded that while the potential exists for periodontal disease and its treatment to cause pulp pathosis, particularly if large lateral or accessory canals are exposed, this occurrence is rare.[102,254]

Mechanical irritants: Orthodontic movement

The most conspicuous pulp change observed in response to orthodontic forces is hemodynamic. Both human and animal studies have confirmed that both lateral and intrusive forces result in an increase in PBF.[158,203,212] Furthermore, blood flow alterations are not confined to the tooth in active movement. Observed increases in blood flow are seen in teeth adjacent to the focus of movement forces, implying that directed forces on one tooth could shunt blood to proximal vessels, supplying other oral structures including teeth. If orthodontic forces are extreme, circulatory interruptions can occur, resulting in pulp necrosis.[37]

Biochemical, biologic, and histological studies of the effects of orthodontic movement have confirmed that metabolic as well as inflammatory changes can result. The dental pulp tissue respiration rate is depressed after short-term

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application of orthodontic force.[100] Biochemical and molecular markers confirm that apoptosis and necrosis of pulp cells are also increased subsequent to movement.[233] However, it was shown that the inflammatory mediators IL-1α and TNF-α show minor increases in the pulp during orthodontic movement, compared to the increase in periodontal tissues.[27] Histological examination of pulps in teeth subjected to intrusive forces showed vascular congestion and dilatation as well as vacuolization of the odontoblastic layer.[276–278] Most, if not all, of these effects are due to circulatory changes, and the consensus is that they are transient provided that the movement forces are not excessive.[152] It was recently shown, however, that pulp necrosis of teeth that were undergoing orthodontic treatment and had also been traumatized prior to that is significantly increased compared to teeth that had either of these conditions but not the other, particularly in lateral incisors and if the traumatized tooth had pulp canal obliteration.[19,20] As noted previously, SP appears to increase in the dental pulp as a result of acutely induced occlusal trauma.[47] Also, CGRP increases under the influence of orthodontic forces.[48] One area of growing interest with respect to orthodontic forces is their role in the pathogenesis of cervical invasive root resorption (CIRR). Studies that have categorized the potential etiology of CIRR have documented that the history of orthodontic treatment is the most cited associated factor.[107,181] However, longitudinal observational or case control studies that show a stronger relationship between CIRR and orthodontic treatment are lacking.

Pulp reactions to orthodontic surgery

It has been known for decades that osteotomies in the maxilla or mandible may cause disruption in the blood supply to teeth in the area of the surgery, with resultant inflammation and/or necrosis.[16,150,200,231] Occasionally, the teeth affected show postoperative manifestations common with traumatic injuries, such as pulp canal obliteration.[305] Animal studies have shown that if a safe distance of 5 to 10 mm is maintained between the site of the surgery and the teeth, minimal disruption occurs.[70,319] A number of studies have documented, using laser Doppler flowmetry, actual reduction in PBF immediately following maxillary Le Fort I osteotomy,[215,242,257] particularly if

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segmental osteotomy is performed.[76] In most cases, the blood flow is regained within months of the surgery. A modification of the Le Fort I osteotomy technique has been described in which the Le Fort I sectioning is combined with a horseshoe palatal osteotomy to spare any disruption to the descending palatine artery.[101] An examination of PBF of maxillary teeth using laser Doppler flowmetry as well as the responsiveness to electric pulp testing showed significant differences between the two surgical techniques in the postoperative recovery values (Fig. 14.26). In cases where the surgery did not disrupt the palatine artery, the PBF in the anterior teeth consistently increased without disruption in the post-operative period. A recent systematic review concluded that following a Le Fort I osteotomy, there is a decrease in pulpal vascularity and neurosensory response but only in the early postoperative period (1 to 10 days), and that this is likely a temporary effect.[243]

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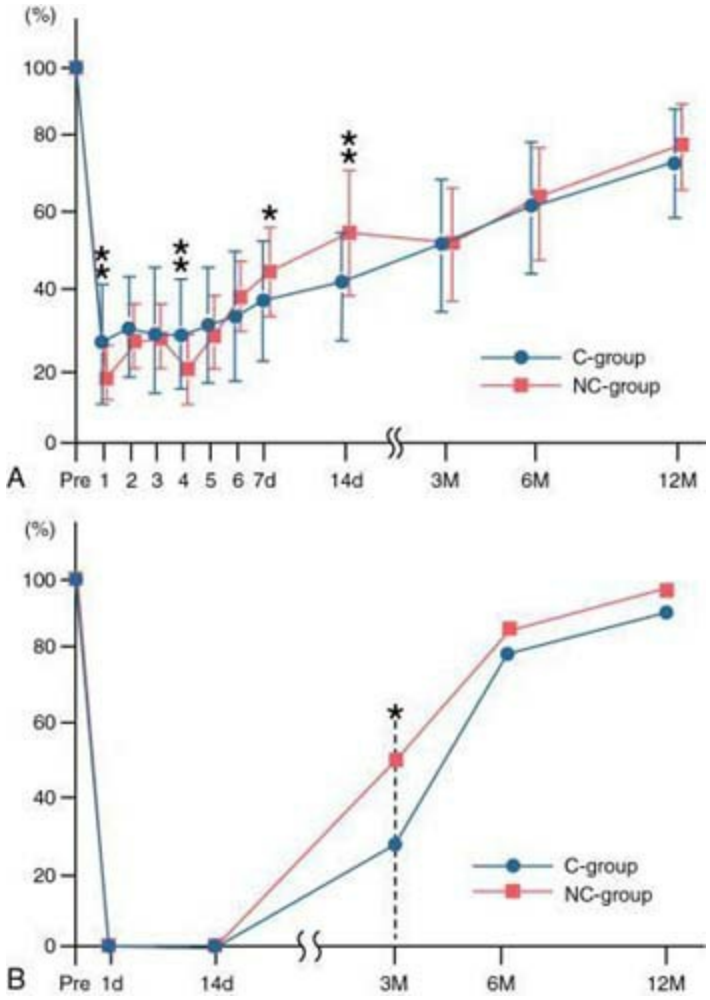

FIG. 14.26 **A,** The postoperative change of the mean pulpal blood flow in the upper incisors of the two groups. **B,** The postoperative change in the percentages of teeth (upper incisors) with positive pulpal sensibility in the two groups. *d*, Day(s) after the operation; *M*, months after the operation; *Pre*, before the operation; *bars,* SD; *, $P < .05$; **, $P < .01$.

A) Graph plots percent of pulpal blood flow against time in days and months. The vertical axis is marked 0 to 100 in increments of 20. The horizontal axis is marked at 1 through 7 days, fourteenth day, third month, sixth month, and twelfth month. The data are as follows: C-group: (1, 28), (2, 29), (3, 28), (4, 28), (5, (29), (6, 30), (7, 31), (14d, 35), (3M, 42), (6M, 58), and (12M, 62). NC-group: (1, 18), (2, 27), (3, 27), (4, 21), (5, 28), (6, 35), (7, 40), (14d, 45), (3M, 42), (6M, 59), and (12 M, 65). C-group is marked with two asterisks at first and fourth day. NC-group is marked with one asterisk at seventh day and two asterisks at fourteenth day. All data are approximate.

B) Graph plots percent of pulpal blood flow against time in days and months. The vertical axis is marked 0 to 100 in increments of 20. The horizontal axis is marked at first day, fourteenth day, third month, sixth month, and twelfth month. The data are as follows: C-group: (1, 0), (14, 0), (3, 21), (6, 79), and

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(12, 81). NC-group: (1, 0), (14, 0), (3, 42), (6, 82), and (12, 84). C- and NC-groups are marked at third month. All data are approximate.

Source: (Reprinted with permission from Harada K, Sato M, Omura K. Blood-flow and neurosensory changes in the maxillary dental pulp after differing Le Fort I osteotomies. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:12–17, 2004.)

It should also be noted that occasionally, teeth are traumatized during endotracheal intubation for surgery that requires general anesthesia, when the surgery itself is not related to the jaws or teeth.[265]

Biomechanical irritation: Parafunctional habits

Occlusal loading of teeth effects deformation to varying degrees.[187] While enamel is largely resistant to flexure, the underlying dentin demonstrates considerable elastic and viscoelastic characteristics. As a result, defects in enamel secondary to cracks, decay, and/or restorative preparation allow cusp flexure with subsequent pulp responses, presumably due to dentinal fluid flow secondary to compression and microleakage.[110] The magnitude of the pulp response is dictated by the degree and chronicity of dentinal deformation.

Multiple factors influence the degree of tooth deformation during occlusal loading. Investigators have noted that preparation geometry has a direct impact on cuspal flexure. The width of the occlusal isthmus relative to the facio-lingual dimension of the tooth as well as the ablation of marginal ridges directly impact the degree of cusp flexure.[221,244,304] MOD preparations have been shown to cause a 50% reduction in cusp stiffness and resistance to fracture. Physical properties of the restorative material can also play a part in cusp flexure. Studies have shown that polymerization shrinkage of certain resin composites can induce an inward deflection of cusps with resultant stresses on tooth structure.[306,307]

Symptomology from cusp flexure can result from two primary sources. It has been theorized that cusp flexure results in dentin deformation, thus promoting dentinal fluid flow that activates nerve endings in the odontoblast layer of the tooth. This is supported in part by an in vitro study, which found that dentinal fluid flow could be induced by occlusal loading of restored

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teeth.[110] A second source of pulp pain is bacterial microleakage created by a gap at the restoration/dentin interface that is repeatedly opened during cycles of occlusal loading. If repeated cuspal flexure gives rise to a crack, dentinal exposure to bacteria and their by-products is even greater. It is likely that in vivo, both dentinal fluid flow and bacterial access to dentinal surfaces work together to produce inflammation that is often manifested by thermal as well as biting sensitivity for the patient. In the instance of parafunctional habits, this is often combined with concussive periodontal forces to the periodontium that induce acute periradicular periodontitis, mobility, and radiographic changes.

On the cellular level, studies have shown increased levels of SP after experimentally induced occlusal trauma.[47] SP is a modulator of both sensory and immune function in the dental pulp. Alterations in physiologic levels of this mediator could result in disruptions in the pain response as well as secondary stimulation of prostaglandin E2. Furthermore, the immunostimulatory ability of neuropeptides could initiate and maintain a chronic pulp inflammation, possibly leading to necrosis. While animal studies have suggested that pulp necrosis is a possible sequela to chronic occlusal trauma, there are no controlled clinical studies that confirm this.

Dentinal cracks expose tubules that are not occluded by a smear layer and therefore offer a direct portal to the subjacent pulp. When dentinal tubules are freely exposed, there is an outward flow of dentinal fluid driven by relatively high pulp tissue pressures. Dentinal fluid is composed of proteins such as fibrinogen and serum albumin, which can coagulate and effectively block the tubule lumen, thereby limiting fluid egress and resultant dentin hypersensitivity. This phenomenon can occur within 2 days. As this serves as a short-term protective mechanism for the pulp, dentinal sclerosis and tertiary dentin formation can ultimately provide greater protection for the pulp and reduction of symptoms. Clinical interventions include the application of materials that occlude the tubules and extracoronal restorations to prevent the propagation of cracks. Cracks were also shown to harbor bacterial biofilms that can add to the irritation and inflammation of the pulp and induce periodontal pocket formation in cases with cracked teeth.[249]

Pulp reactions to implant placement and

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function

Osseointegrated implants are now a common option for the replacement of missing teeth. The placement of implants requires multifaceted preoperative radiographic techniques, including intraoral, tomographic, cephalometric, and panoramic imaging.[298] This ensures that implant placement fully rests in bone and does not compromise neighboring structures, including teeth. The lack of attention to the three-dimensional anatomy of the site of implant placement and the orientation of neighboring teeth may lead to the implant perforating the root and devitalizing the pulp.[180,281] The same could occur during procedures of maxillary sinus floor augmentation.[21]

It is usually recommended that implants not be placed directly at a site where a periradicular lesion, particularly one with signs of purulence, exists, as microbial irritants may interfere with osseointegration.[238] However, some data suggest that immediate implant placement in sites that have been adequately debrided is successful.[42] One systematic review corroborated this finding, provided that adequate preoperative, intraoperative, and postoperative antimicrobial measures are utilized.[52]

Case reports have also claimed that teeth with periradicular lesions may reduce the success of neighboring implants even if adequate endodontic treatment is performed.[280] To address this issue, a study was reported in which implants were placed to replace premolars in dogs and periradicular lesions were likewise induced, some of which were treated nonsurgically or with surgical and nonsurgical treatment.[263] The results showed that the presence of treated or untreated periradicular lesions did not affect the long-term osseointegration of implants that were already osseointegrated. Clinical cases with complete resolution of periapical lesions that also involve neighboring implants, following adequate endodontic treatment, have been published.[170,256,275]

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References

1. About I, Murray PE, Franquin JC, et al: *Pulpal inflammatory responses following non-carious class V restorations*, Oper Dent 2001;26: 336-342.
2. Absi EG, Addy M, Adams D. *Dentine hypersensitivity—the effect of toothbrushing and dietary compounds on dentine in vitro: an SEM study*, J Oral Rehabil 1992;19: 101-110.
3. Addy M, Pearce N. *Aetiological, predisposing and environmental factors in dentine hypersensitivity*, Arch Oral Biol Suppl, 1994;39: 33S- 38S.
4. Aguilar P, Linsuwanont P. *Vital pulp therapy in vital permanent teeth with cariously exposed pulp: a systematic review*, J Endod 2011;37: 581-587.
5. Ahlquist M, Franzen O, Coffey J, et al: *Dental pain evoked by hydrostatic pressures applied to exposed dentin in man: a test of the hydrodynamic theory of dentin sensitivity*, J Endod 1994;20: 130-134.
6. Ahn J, Pogrel MA. *The effects of 2% lidocaine with 1:100,000 epinephrine on pulpal and gingival blood flow*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 197-202.
7. Al-Hiyasat AS, Barrieshi-Nusair KM, Al-Omari MA. *The radiographic outcomes of direct pulp-capping procedures performed by dental students: a retrospective study*, J Am Dent Assoc 2006;137: 1699-1705.
8. Aljehani A, Yang L, Shi XQ. *In vitro quantification of smooth surface caries with DIAGNOdent and the DIAGNOdent pen*, Acta Odontol Scand 2007;65: 60-63.
9. Amano T, Muramatsu T, Amemiya K, et al: *Responses of*

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www.konkur.in

rat pulp cells to heat stress in vitro, J Dent Res 2006;85: 432-435.

10. Anderson DG, Chiego DJ Jr, Glickman GN, et al: *A clinical assessment of the effects of 10% carbamide peroxide gel on human pulp tissue*, J Endod 1999;25: 247-250.
11. Armstrong SR, Vargas MA, Fang Q, et al: *Microtensile bond strength of a total-etch 3-step, total-etch 2-step, self-etch 2-step, and a self-etch 1-step dentin bonding system through 15-month water storage*, J Adhes Dent 2003;5: 47-56.
12. Asgary S, Eghbal MJ, Parirokh M, et al: *A comparative study of histologic response to different pulp capping materials and a novel endodontic cement*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 609-614.
13. Austah O, Widbiller M, Tomson PL, et al: *Expression of neurotrophic factors in human dentin and their regulation of trigeminal neurite outgrowth*, J Endod 2019;45: 414-419.
14. Awawdeh L, Lundy FT, Shaw C, et al: *Quantitative analysis of substance P, neurokinin A and calcitonin gene-related peptide in pulp tissue from painful and healthy human teeth*, Int Endod J 2002;35: 30-36.
15. Baldissara P, Catapano S, Scotti R. *Clinical and histological evaluation of thermal injury thresholds in human teeth: a preliminary study*, J Oral Rehabil 1997;24: 791-801.
16. Banks P. *Pulp changes after anterior mandibular subapical osteotomy in a primate model*, J Oral Maxillofac Surg 1977;5: 39-48.
17. Barrieshi-Nusair KM, Qudeimat MA. *A prospective clinical study of mineral trioxide aggregate for partial*

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www.konkur.in

pulpotomy in cariously exposed permanent teeth, J Endod 2006;32: 731-735.

18. Barthel CR, Rosenkranz B, Leuenberg A, et al: *Pulp capping of carious exposures: treatment outcome after 5 and 10 years: a retrospective study*, J Endod 2000;26: 525-528.
19. Bauss O, Rohling J, Rahman A, et al: *The effect of pulp obliteration on pulpal vitality of orthodontically intruded traumatized teeth*, J Endod 2008;34: 417-420.
20. Bauss O, Rohling J, Sadat-Khonsari R, et al: *Influence of orthodontic intrusion on pulpal vitality of previously traumatized maxillary permanent incisors*, Am J Orthod Dentofacial Orthop 2008;134: 12-17.
21. Beck F, Lauterbrunner N, Lettner S, et al: *Devitalization of adjacent teeth following maxillary sinus floor augmentation: A retrospective radiographic study*, Clin Implant Dent Relat Res 2018;20: 763-769.
22. Bergenholtz G, Ahlstedt S, Lindhe J. *Experimental pulpitis in immunized monkeys*, Scand J Dent Res 1977;85: 396-406.
23. Bergenholtz G, Cox CF, Loesche WJ, et al: *Bacterial leakage around dental restorations: its effect on the dental pulp*, J Oral Pathol 1982;11: 439-450.
24. Bergenholtz G, Lindhe J. *Effect of experimentally induced marginal periodontitis and periodontal scaling on the dental pulp*, J Clin Periodontol 1978;5: 59-73.
25. Bergenholtz G, Nyman S. *Endodontic complications following periodontal and prosthetic treatment of patients with advanced periodontal disease*, J Periodontol 1984;55: 63-68.
26. Bergenholtz G, Warfvinge J. *Migration of leukocytes in dental pulp in response to plaque bacteria*, Scand J Dent

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Res 1982;90: 354-362.
27. Bletsa A, Berggreen E, Brudvik P. *Interleukin-1alpha and tumor necrosis factor-alpha expression during the early phases of orthodontic tooth movement in rats*, Eur J Oral Sci 2006;114: 423-429.
28. Bogen G, Kim JS, Bakland LK. *Direct pulp capping with mineral trioxide aggregate: an observational study*, J Am Dent Assoc 2008;139: 305-315; quiz 05-15.
29. Botero TM, Shelburne CE, Holland GR, et al: *TLR4 mediates LPS-induced VEGF expression in odontoblasts*, J Endod 2006;32: 951-955.
30. Bouillaguet S. *Biological risks of resin-based materials to the dentin-pulp complex*, Crit Rev Oral Biol Med 2004;15: 47-60.
31. Bowles WR, Burke R, Sabino M, et al: *Sex differences in neuropeptide content and release from rat dental pulp*, J Endod 2011;37: 1098-1101.
32. Bowles WR, Withrow JC, Lepinski AM, et al: *Tissue levels of immunoreactive substance P are increased in patients with irreversible pulpitis*, J Endod 2003;29: 265-267.
33. Brannstrom M. *Dental and pulpal response. III. Application of an air stream to exposed dentin. Long observation periods*, Acta Odontol Scand 1960;18: 234-252.
34. Brannstrom M. *The effect of dentin desiccation and aspirated odontoblasts on the pulp*, J Prosthet Dent 1968;20: 165-171.
35. Brannstrom M. *Smear layer: pathological and treatment considerations*, Oper Dent- Supplement 1984;3: 35-42.
36. Brannstrom M, Lind PO. *Pulpal response to early dental caries*, J Dent Res 1965;44: 1045-1050.

Telegram: @dental_k

www.konkur.in

37. Butcher EO, Taylor AC. *The effects of denervation and ischemia upon the teeth of the monkey*, J Dent Res 1951;30: 265-275.
38. Byers MR, Narhi MV. *Dental injury models: experimental tools for understanding neuroinflammatory interactions and polymodal nociceptor functions*, Crit Rev Oral Biol Med 1999;10: 4-39.
39. Byers MR, Narhi MV, Mecifi KB. *Acute and chronic reactions of dental sensory nerve fibers to cavities and desiccation in rat molars*, Anat Rec 1988;221: 872-883.
40. Calland JW, Harris SE, Carnes DL Jr. *Human pulp cells respond to calcitonin gene-related peptide in vitro*, J Endod 1997;23: 485-489.
41. Camilleri J. *Investigation of biodentine as dentine replacement material*, J Dent 2013;41: 600-610.
42. Casap N, Zeltser C, Wexler A, et al: *Immediate placement of dental implants into debrided infected dentoalveolar sockets*, J Oral Maxillofac Surg 2007;65: 384-392.
43. Cassidy N, Fahey M, Prime SS, et al: *Comparative analysis of transforming growth factor-beta isoforms 1-3 in human and rabbit dentine matrices*, Arch Oral Biol 1997;42: 219-223.
44. Caux C, Vanbervliet B, Massacrier C, et al: *Regulation of dendritic cell recruitment by chemokines*, Transplantation 2002;73: S7- S11.
45. Cavalcanti BN, Lage-Marques JL, Rode SM. *Pulpal temperature increases with Er:YAG laser and high-speed handpieces*, J Prosthet Dent 2003;90: 447-451.
46. Caviedes-Bucheli J, Ariza-Garcia G, Restrepo-Mendez S, et al: *The effect of tooth bleaching on substance P expression in human dental pulp*, J Endod 2008;34: 1462-1465.

Telegram: @dental_k

www.konkur.in

47. Caviedes-Bucheli J, Azuero-Holguin MM, Correa-Ortiz JA, et al: *Effect of experimentally induced occlusal trauma on substance p expression in human dental pulp and periodontal ligament*, J Endod 2011;37: 627-630.
48. Caviedes-Bucheli J, Moreno JO, Ardila-Pinto J, et al: *The effect of orthodontic forces on calcitonin gene-related peptide expression in human dental pulp*, J Endod 2011;37: 934-937.
49. Chabanski MB, Gillam DG. *Aetiology, prevalence and clinical features of cervical dentine sensitivity*, J Oral Rehabil 1997;24: 15-19.
50. Cheung GS, Lai SC, Ng RP. *Fate of vital pulps beneath a metal-ceramic crown or a bridge retainer*, Int Endod J 2005;38: 521-530.
51. Chng HS, Pitt Ford TR, McDonald F. *Effects of prilocaine local anaesthetic solutions on pulpal blood flow in maxillary canines*, Endod Dent Traumatol 1996;12: 89-95.
52. Chrcanovic BR, Martins MD, Wennerberg A. *Immediate placement of implants into infected sites: a systematic review*, Clin Implant Dent Relat Res 2013.
53. Cohen JS, Reader A, Fertel R, et al: *A radioimmunoassay determination of the concentrations of prostaglandins E2 and F2alpha in painful and asymptomatic human dental pulps*, J Endod 1985;11: 330-335.
54. Coluzzi DJ. *An overview of laser wavelengths used in dentistry*, Dent Clin North Am 2000;44: 753-765.
55. Costa AM, Bezzerra AC, Fuks AB. *Assessment of the accuracy of visual examination, bite-wing radiographs and DIAGNOdent on the diagnosis of occlusal caries*, Eur Arch Paediatr Dent 2007;8: 118-122.
56. Costa CA, Edwards CA, Hanks CT. *Cytotoxic effects of cleansing solutions recommended for chemical lavage of*

Telegram: @dental_k

www.konkur.in

pulp exposures, Am J Dent 2001;14: 25-30.

57. Costa CA, Giro EM, do Nascimento AB, et al: *Short-term evaluation of the pulpo-dentin complex response to a resin-modified glass-ionomer cement and a bonding agent applied in deep cavities*, Dent Mater 2003;19: 739-746.
58. Costa CA, Riehl H, Kina JF, et al: *Human pulp responses to in-office tooth bleaching*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: e59- e64.
59. Cotton WR. *Pulp response to an airstream directed into human cavity preparations*, Oral Surg Oral Med Oral Pathol 1967;24: 78-88.
60. Cox CF, Bergenholtz G, Fitzgerald M, et al: *Capping of the dental pulp mechanically exposed to the oral microflora — a 5 week observation of wound healing in the monkey*, J Oral Pathol 1982;11: 327-339.
61. Cox CF, Hafez AA, Akimoto N, et al: *Biocompatibility of primer, adhesive and resin composite systems on non-exposed and exposed pulps of non-human primate teeth*, Am J Dent 1998;11: S55- S63.
62. Cox CF, Suzuki S. *Re-evaluating pulp protection: calcium hydroxide liners vs. cohesive hybridization*, J Am Dent Assoc 1994;125: 823-831.
63. Cvek M. *A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture*, J Endod 1978;4: 232-237.
64. Cvek M, Granath L, Cleaton-Jones P, et al: *Hard tissue barrier formation in pulpotomized monkey teeth capped with cyanoacrylate or calcium hydroxide for 10 and 60 minutes*, J Dent Res 1987;66: 1166-1174.
65. Czarnecki RT, Schilder H. *A histological evaluation of the human pulp in teeth with varying degrees of periodontal*

Telegram: @dental_k

www.konkur.in

disease, J Endod 1979;5: 242-253.
66. de Melo Alencar C, de Paula BLF, Guanipa Ortiz MI, et al: *Clinical efficacy of nano-hydroxyapatite in dentin hypersensitivity: A systematic review and meta-analysis*, J Dent 2019;82: 11-21.
67. do Nascimento AB, Fontana UF, Teixeira HM, et al: *Biocompatibility of a resin-modified glass-ionomer cement applied as pulp capping in human teeth*, Am J Dent 2000;13: 28-34.
68. Dummer PM, Hicks R, Huws D. *Clinical signs and symptoms in pulp disease*, Int Endod J 1980;13: 27-35.
69. Duncan HF, Galler KM, Tomson PL, et al: *European society of endodontology position statement: management of deep caries and the exposed pulp*, Int Endod J 2019.
70. Duran S, Guven O, Gunhan O. *Pulpal and apical changes secondary to segmental osteotomy in the mandible—an experimental study*, J Craniomaxillofac Surg 1995;23: 256-260.
71. Durand SH, Flacher V, Romeas A, et al: *Lipoteichoic acid increases TLR and functional chemokine expression while reducing dentin formation in in vitro differentiated human odontoblasts*, J Immunol 2006;176: 2880-2887.
72. El Karim IA, Lamey PJ, Ardill J, et al: *Vasoactive intestinal polypeptide (VIP) and VPAC1 receptor in adult human dental pulp in relation to caries*, Arch Oral Biol 2006;51: 849-855.
73. El Karim IA, Lamey PJ, Linden GJ, et al: *Caries-induced changes in the expression of pulpal neuropeptide Y*, Eur J Oral Sci 2006;114: 133-137.
74. El Karim IA, Lamey PJ, Linden GJ, et al: *Neuropeptide Y Y1 receptor in human dental pulp cells of noncarious and carious teeth*, Int Endod J 2008;41: 850-855.

Telegram: @dental_k

www.konkur.in

75. Elliott RD, Roberts MW, Burkes J, et al: *Evaluation of the carbon dioxide laser on vital human primary pulp tissue*, Pediatr Dent 1999;21: 327-331.
76. Emshoff R, Kranewitter R, Brunold S, et al: *Characteristics of pulpal blood flow levels associated with non-segmented and segmented Le Fort I osteotomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: 379-384.
77. Eren B, Onay EO, Ungor M. *Assessment of alternative emergency treatments for symptomatic irreversible pulpitis: a randomized clinical trial*, Int Endod J 2017.
78. Eversole LR, Rizoiu I, Kimmel AI. *Pulpal response to cavity preparation by an erbium, chromium:YSGG laser-powered hydrokinetic system*, J Am Dent Assoc 1997;128: 1099-1106.
79. Faraco IM Jr, Holland R. *Response of the pulp of dogs to capping with mineral trioxide aggregate or a calcium hydroxide cement*, Dent Traumatol 2001;17: 163-166.
80. Farges JC, Keller JF, Carrouel F, et al: *Odontoblasts in the dental pulp immune response*, J Exp Zoolog B Mol Dev Evol 2008.
81. Farges JC, Keller JF, Carrouel F, et al: *Odontoblasts in the dental pulp immune response*, J Exp Zool B Mol Dev Evol 2009;312B: 425-436.
82. Farges JC, Keller JF, Carrouel F, et al: *O34-pathogen sensing by human odontoblasts*, Bull Group Int Rech Sci Stomatol Odontol 2010;49: 90.
83. Farges JC, Romeas A, Melin M, et al: *TGF-beta1 induces accumulation of dendritic cells in the odontoblast layer*, J Dent Res 2003;82: 652-656.
84. Farik B, Munksgaard EC, Andreasen JO, et al: *Drying and rewetting anterior crown fragments prior to bonding*,

Telegram: @dental_k

www.konkur.in

Endod Dent Traumatol 1999;15: 113-116.
85. Felton D. *Long-term effects of crown preparation on pulp vitality*, J Dent Res 1989;68: 1009- (abstract 139).
86. Ferracane JL, Condon JR. *Rate of elution of leachable components from composite*, Dent Mater 1990;6: 282-287.
87. Fitzgerald M, Hanks CT. *In vivo study of resin diffusion through intact vital human dentin (abstract)*, J Dent Res 1997;76: 305.
88. Flecha OD, Azevedo CG, Matos FR, et al: *Cyanoacrylate versus laser in the treatment of dentin hypersensitivity: a controlled, randomized, double-masked and non-inferiority clinical trial*, J Periodontol 2013;84: 287-294.
89. Ford TR, Torabinejad M, Abedi HR, et al: *Using mineral trioxide aggregate as a pulp-capping material*, J Am Dent Assoc 1996;127: 1491-1494.
90. Fosse G, Saele PK, Eide R. *Numerical density and distributional pattern of dentin tubules*, Acta Odontol Scand 1992;50: 201-210.
91. Fried K. *Changes in pulpal nerves with aging*, Proc Finn Dent Soc 1992;88: 517-528.
92. Fugaro JO, Nordahl I, Fugaro OJ, et al: *Pulp reaction to vital bleaching*, Oper Dent 2004;29: 363-368.
93. Fujitani M, Inokoshi S, Hosoda H. *Effect of acid etching on the dental pulp in adhesive composite restorations*, Int Dent J 1992;42: 3-11.
94. Garberoglio R, Brannstrom M. *Scanning electron microscopic investigation of human dentinal tubules*, Arch Oral Biol 1976;21: 355-362.
95. Gibson M, Sharif MO, Smith A, et al: *A practice-based randomised controlled trial of the efficacy of three interventions to reduce dentinal hypersensitivity*, J Dent 2013.

Telegram: @dental_k

www.konkur.in

96. Goodis HE, Schein B, Stauffer P. *Temperature changes measured in vivo at the dentinoenamel junction and pulpodentin junction during cavity preparation in the Macaca fascicularis monkey*, J Endod 1988;14: 336-339.
97. Graham L, Cooper PR, Cassidy N, et al: *The effect of calcium hydroxide on solubilisation of bio-active dentine matrix components*, Biomaterials 2006;27: 2865-2873.
98. Gwinnett AJ, Tay F. *Early and intermediate time response of the dental pulp to an acid etch technique in vivo*, Am J Dent 1998;11: S35- S44.
99. Gwinnett AJ, Tay FR, Pang KM, et al: *Quantitative contribution of the collagen network in dentin hybridization*, Am J Dent 1996;9: 140-144.
100. Hamersky PA, Weimer AD, Taintor JF. *The effect of orthodontic force application on the pulpal tissue respiration rate in the human premolar*, Am J Orthod 1980;77: 368-378.
101. Harada K, Sato M, Omura K. *Blood-flow and neurosensory changes in the maxillary dental pulp after differing Le Fort I osteotomies*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 12-17.
102. Harrington GW, Steiner DR, Ammons WF. *The periodontal-endodontic controversy*, Periodontol 2000 2002;30: 123-130.
103. Harris DM, White JM, Goodis H, et al: *Selective ablation of surface enamel caries with a pulsed Nd:YAG dental laser*, Lasers Surg Med 2002;30: 342-350.
104. Hartmann A, Azerad J, Boucher Y. *Environmental effects on laser doppler pulpal blood-flow measurements in man*, Arch Oral Biol 1996;41: 333-339.
105. Hasler JE, Mitchell DF. *Painless pulpitis*, J Am Dent Assoc 1970;81: 671-677.

Telegram: @dental_k

www.konkur.in

106. Hayden MS, West AP, Ghosh S. *NF-kappaB and the immune response*, Oncogene 2006;25: 6758-6780.
107. Heithersay GS. *Invasive cervical resorption: an analysis of potential predisposing factors*, Quintessence Int 1999;30: 83-95.
108. Heys DR, Cox CF, Heys RJ, et al: *Histological considerations of direct pulp capping agents*, J Dent Res 1981;60: 1371-1379.
109. Hilton TJ, Ferracane JL, Mancl L, et al: *Comparison of CaOH with MTA for direct pulp capping: a PBRN randomized clinical trial*, J Dent Res 2013;92: 16S- 22S.
110. Hirata K, Nakashima M, Sekine I, et al: *Dentinal fluid movement associated with loading of restorations*, J Dent Res 1991;70: 975-978.
111. Holland GR, Narhi MN, Addy M, et al: *Guidelines for the design and conduct of clinical trials on dentine hypersensitivity*, J Clin Periodontol 1997;24: 808-813.
112. Holland R, de Souza V, Nery MJ, et al: *Reaction of dogs' teeth to root canal filling with mineral trioxide aggregate or a glass ionomer sealer*, J Endod 1999;25: 728-730.
113. Horst OV, Horst JA, Samudrala R, et al: *Caries induced cytokine network in the odontoblast layer of human teeth*, BMC Immunol 2011;12: 9.
114. Horst OV, Tompkins KA, Coats SR, et al: *TGF-beta1 Inhibits TLR-mediated odontoblast responses to oral bacteria*, J Dent Res 2009;88: 333-338.
115. Horsted P, Sandergaard B, Thylstrup A, et al: *A retrospective study of direct pulp capping with calcium hydroxide compounds*, Endod Dent Traumatol 1985;1: 29-34.
116. Huang B, Cvitkovitch DG, Santerre JP, et al: *Biodegradation of resin-dentin interfaces is dependent on*

Telegram: @dental_k

www.konkur.in

the restorative material, mode of adhesion, esterase or MMP inhibition, Dent Mater 2018;34: 1253-1262.

117. Hume WR. *An analysis of the release and the diffusion through dentin of eugenol from zinc oxide-eugenol mixtures*, J Dent Res 1984;63: 881-884.
118. Hume WR, Gerzia TM. *Bioavailability of components of resin-based materials which are applied to teeth*, Crit Rev Oral Biol Med 1996;7: 172-179.
119. xxx.
120. Hume WR, Kenney AE. *Release of 3H-triamcinolone from Ledermix*, J Endod 1981;7: 509-514.
121. Ikawa M, Komatsu H, Ikawa K, et al: *Age-related changes in the human pulpal blood flow measured by laser doppler flowmetry*, Dent Traumatol 2003;19: 36-40.
122. Inoue H, Izumi T, Ishikawa H, et al: *Short-term histomorphological effects of Er:YAG laser irradiation to rat coronal dentin-pulp complex*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 246-250.
123. Inoue T, Miyakoshi S, Shimono M. *The in vitro and in vivo influence of 4-META/MMA-TBB resin components on dental pulp tissues*, Adv Dent Res 2001;15: 101-104.
124. Inoue T, Miyakoshi S, Shimono M. *Dentin/pulp adhesive resin interface. Biological view from basic science to clinic*, Shimono M MT Suda H Takahashi K Proceedings of the international conference on dentin/pulp complex 1995 1996; Quintessence Chiba, Japan. Chicago 217-220.
125. Izumi T, Inoue H, Matsuura H, et al: *Changes in the pattern of horseradish peroxidase diffusion into predentin and dentin after cavity preparation in rat molars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 675-681.
126. Izumi T, Kobayashi I, Okamura K, et al:

Telegram: @dental_k

www.konkur.in

Immunohistochemical study on the immunocompetent cells of the pulp in human non-carious and carious teeth, Arch Oral Biol 1995;40: 609-614.

127. Jaber L, Swaim WD, Dionne RA. *Immunohistochemical localization of mu-opioid receptors in human dental pulp*, J Endod 2003;29: 108-110.
128. Janeway CA Jr, Medzhitov R. *Innate immune recognition*, Annu Rev Immunol 2002;20: 197-216.
129. Jansson L, Ehnevid H, Lindskog S, et al: *Relationship between periapical and periodontal status, A clinical retrospective study*, J Clin Periodontol 1993;20: 117-123.
130. Jansson L, Sandstedt P, Laftman AC, et al: *Relationship between apical and marginal healing in periradicular surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 596-601.
131. Jiang HW, Zhang W, Ren BP, et al: *Expression of toll like receptor 4 in normal human odontoblasts and dental pulp tissue*, J Endod 2006;32: 747-751.
132. Jontell M, Hanks CT, Bratel J, et al: *Effects of unpolymerized resin components on the function of accessory cells derived from the rat incisor pulp*, J Dent Res 1995;74: 1162-1167.
133. Jukic S, Anic I, Koba K, et al: *The effect of pulpotomy using CO2 and Nd:YAG lasers on dental pulp tissue*, Int Endod J 1997;30: 175-180.
134. Kang CM, Sun Y, Song JS, et al: *A randomized controlled trial of various MTA materials for partial pulpotomy in permanent teeth*, J Dent 2017;60: 8-13.
135. Kanjevac T, Milovanovic M, Volarevic V, et al: *Cytotoxic effects of glass ionomer cements on human dental pulp stem cells correlate with fluoride release*, Med Chem 2012;8: 40-45.

Telegram: @dental_k

www.konkur.in

136. Katsuno K, Manabe A, Itoh K, et al: *A delayed hypersensitivity reaction to dentine primer in the guinea-pig*, J Dent 1995;23: 295-299.
137. Kawagishi E, Nakakura-Ohshima K, Nomura S, et al: *Pulpal responses to cavity preparation in aged rat molars*, Cell Tissue Res 2006;326: 111-122.
138. Keller JF, Carrouel F, Colomb E, et al: *Toll-like receptor 2 activation by lipoteichoic acid induces differential production of pro-inflammatory cytokines in human odontoblasts, dental pulp fibroblasts and immature dendritic cells*, Immunobiology 2010;215: 53-59.
139. Killough SA, Lundy FT, Irwin CR. *Substance P expression by human dental pulp fibroblasts: a potential role in neurogenic inflammation*, J Endod 2009;35: 73-77.
140. Kim D, Park SH. *Effects of age, sex, and blood pressure on the blood flow velocity in dental pulp measured by doppler ultrasound technique*, Microcirculation 2016;23: 523-529.
141. Kim S. *Ligamental injection: a physiological explanation of its efficacy*, J Endod 1986;12: 486-491.
142. Kim S, Edwall L, Trowbridge H, et al: *Effects of local anesthetics on pulpal blood flow in dogs*, J Dent Res 1984;63: 650-652.
143. Kim S, Trowbridge H, Suda H. *Pulpal reaction to caries and dental procedures*, Cohen S Burns RC Pathways of the pulp ed 8 2002; Mosby St. Louis 573-600.
144. Kimura Y, Wilder-Smith P, Yonaga K, et al: *Treatment of dentine hypersensitivity by lasers: a review*, J Clin Periodontol 2000;27: 715-721.
145. Kimura Y, Yonaga K, Yokoyama K, et al: *Histopathological changes in dental pulp irradiated by Er:YAG laser: a preliminary report on laser pulpotomy*, J

Telegram: @dental_k

www.konkur.in

Clin Laser Med Surg 2003;21: 345-350.
146. Kina JF, Huck C, Riehl H, et al: *Response of human pulps after professionally applied vital tooth bleaching*, Int Endod J 2010;43: 572-580.
147. Kivanc BH, Arisu HD, Ulusoy OI, et al: *Effect of light-activated bleaching on pulp chamber temperature rise: an in vitro study*, Aust Endod J 2012;38: 76-79.
148. Koh ET, Ford TR, Kariyawasam SP, et al: *Prophylactic treatment of dens evaginatus using mineral trioxide aggregate*, J Endod 2001;27: 540-542.
149. Kohli MR, Yamaguchi M, Setzer FC, et al: *Spectrophotometric analysis of coronal tooth discoloration induced by various bioceramic cements and other endodontic materials*, J Endod 2015;41: 1862-1866.
150. Kohn MW, White RP Jr. *Evaluation of sensation after segmental alveolar osteotomy in 22 patients*, J Am Dent Assoc 1974;89: 154-156.
151. Kokkas AB, Goulas A, Varsamidis K, et al: *Irreversible but not reversible pulpitis is associated with up-regulation of tumour necrosis factor-alpha gene expression in human pulp*, Int Endod J 2007;40: 198-203.
152. Konno Y, Daimaruya T, Iikubo M, et al: *Morphologic and hemodynamic analysis of dental pulp in dogs after molar intrusion with the skeletal anchorage system*, Am J Orthod Dentofacial Orthop 2007;132: 199-207.
153. Koshiro K, Inoue S, Tanaka T, et al: *In vivo degradation of resin-dentin bonds produced by a self-etch vs. a total-etch adhesive system*, Eur J Oral Sci 2004;112: 368-375.
154. Koubi G, Colon P, Franquin JC, et al: *Clinical evaluation of the performance and safety of a new dentine substitute, Biodentine, in the restoration of posterior teeth - a prospective study*, Clin Oral Investig 2013;17: 243-249.

Telegram: @dental_k

www.konkur.in

155. Koulaouzidou EA, Papazisis KT, Economides NA, et al: *Antiproliferative effect of mineral trioxide aggregate, zinc oxide-eugenol cement, and glass-ionomer cement against three fibroblastic cell lines*, J Endod 2005;31: 44-46.
156. Kuhnisch J, Bucher K, Henschel V, et al: *Reproducibility of DIAGNOdent 2095 and DIAGNOdent Pen measurements: results from an in vitro study on occlusal sites*, Eur J Oral Sci 2007;115: 206-211.
157. Kundzina R, Stangvaltaite L, Eriksen HM, et al: *Capping carious exposures in adults: a randomized controlled trial investigating mineral trioxide aggregate versus calcium hydroxide*, Int Endod J 2017;50: 924-932.
158. Kvinnsland S, Heyeraas K, Ofjord ES. *Effect of experimental tooth movement on periodontal and pulpal blood flow*, Eur J Orthod 1989;11: 200-205.
159. Langeland K. *Tissue response to dental caries*, Endod Dent Traumatol 1987;3: 149-171.
160. Langeland K, Dowden WE, Tronstad L, et al: *Human pulp changes of iatrogenic origin*, Oral Surg Oral Med Oral Pathol 1971;32: 943-980.
161. Langeland K, Rodrigues H, Dowden W. *Periodontal disease, bacteria, and pulpal histopathology*, Oral Surg Oral Med Oral Pathol 1974;37: 257-270.
162. Lefebvre CA, Wataha JC, Bouillaguet S, et al: *Effects of long-term sub-lethal concentrations of dental monomers on THP-1 human monocytes*, J Biomater Sci Polym Ed 1999;10: 1265-1274.
163. Lepinski AM, Haegreaves KM, Goodis HE, et al: *Bradykinin levels in dental pulp by microdialysis*, J Endod 2000;26: 744-747.
164. Lesot H B-KC, Kubler MD. *Experimental induction of odontoblast differentiation and stimulation during*

Telegram: @dental_k

www.konkur.in

reparative processes, Cells Materials 1993;3: 201-217.
165. Levin LG, Rudd A, Bletsa A, et al: *Expression of IL-8 by cells of the odontoblast layer in vitro*, Eur J Oral Sci 1999;107: 131-137.
166. Li Y, Sui B, Dahl C, et al: *Pulpotomy for carious pulp exposures in permanent teeth: A systematic review and meta-analysis*, J Dent 2019;84: 1-8.
167. Lier BB, Rosing CK, Aass AM, et al: *Treatment of dentin hypersensitivity by Nd:YAG laser*, J Clin Periodontol 2002;29: 501-506.
168. Lilja J, Nordenvall KJ, Branstrom M. *Dentin sensitivity, odontoblasts and nerves under desiccated or infected experimental cavities. A clinical, light microscopic and ultrastructural investigation*, Swed Dent J 1982;6: 93-103.
169. Lima DA, Aguiar FH, Liporoni PC, et al: *In vitro evaluation of the effectiveness of bleaching agents activated by different light sources*, J Prosthodont 2009;18: 249-254.
170. Lin S, Mayer Y. *Treatment of a large periradicular lesion of endodontic origin around a dental implant with enamel matrix protein derivative*, J Periodontol 2007;78: 2385-2388.
171. Linde A. *Dentin and Dentinogenesis*, Linde A Noncollagenous proteins and proteoglycans in dentinogenesis 1984; CRC Press Boca Raton 55-92.
172. Litkowski LJ, Hack GD, Sheaffer HB, et al: *Occlusion of Dentin Tubules by 45S5 Bioglass*, Paper presented at: Bioceramics, Proceedings of the 10th international symposium on ceramics in medicine October 1997; France Paris.
173. Liu HC, Lin CP, Lan WH. *Sealing depth of Nd:YAG laser on human dentinal tubules*, J Endod 1997;23: 691-693.

Telegram: @dental_k

www.konkur.in

174. Lockard MW. *A retrospective study of pulpal response in vital adult teeth prepared for complete coverage restorations at ultrahigh speed using only air coolant*, J Prosthet Dent 2002;88: 473-478.
175. Loyd DR, Sun XX, Locke EE, et al: *Sex differences in serotonin enhancement of capsaicin-evoked calcitonin gene-related peptide release from human dental pulp*, Pain 2012;153: 2061-2067.
176. Luk K, Tam L, Hubert M. *Effect of light energy on peroxide tooth bleaching*, J Am Dent Assoc 2004;135: 194-201; quiz 28-9.
177. Lupi-Pegurier L, Bertrand MF, Muller-Bolla M, et al: *Comparative study of microleakage of a pit and fissure sealant placed after preparation by Er:YAG laser in permanent molars*, J Dent Child (Chic) 2003;70: 134-138.
178. Lussi A, Hibst R, Paulus R. *DIAGNOdent: an optical method for caries detection*, J Dent Res Spec No C, 2004;83: C80- C83.
179. Mainkar A, Kim SG. *Diagnostic accuracy of 5 dental pulp tests: a systematic review and meta-analysis*, J Endod 2018;44: 694-702.
180. Margelos JT, Verdelis KG. *Irreversible pulpal damage of teeth adjacent to recently placed osseointegrated implants*, J Endod 1995;21: 479-482.
181. Mavridou AM, Bergmans L, Barendregt D, et al: *Descriptive analysis of factors associated with external cervical resorption*, J Endod 2017;43: 1602-1610.
182. Mazzoni A, Tjaderhane L, Checchi V, et al: *Role of dentin MMPs in caries progression and bond stability*, J Dent Res 2015;94: 241-251.
183. McCormack K, Davies R. *The enigma of potassium ion in the management of dentine hypersensitivity: is nitric oxide*

Telegram: @dental_k

www.konkur.in

the elusive second messenger?, Pain 1996;68: 5-11.
184. McLachlan JL, Sloan AJ, Smith AJ, et al: *S100 and cytokine expression in caries*, Infect Immun 2004;72: 4102-4108.
185. McLachlan JL, Smith AJ, Bujalska IJ, et al: *Gene expression profiling of pulpal tissue reveals the molecular complexity of dental caries*, Biochim Biophys Acta 2005.
186. Mente J, Geletneky B, Ohle M, et al: *Mineral trioxide aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcome*, J Endod 2010;36: 806-813.
187. Messer HH. *Permanent restorations and the dental pulp*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Carol Steam, IL.
188. Michaelson PL, Holland GR. *Is pulpitis painful?*, Int Endod J 2002;35: 829-832.
189. Mitchell DF, Tarplee RE. *Painful pulpitis; a clinical and microscopic study*, Oral Surg Oral Med Oral Pathol 1960;13: 1360-1370.
190. Mjor IA. *Bacteria in experimentally infected cavity preparations*, Scand J Dent Res 1977;85: 599-605.
191. Mjor IA, Ferrari M. *Pulp-dentin biology in restorative dentistry, Part 6: Reactions to restorative materials, tooth-restoration interfaces, and adhesive techniques*, Quintessence Int 2002;33: 35-63.
192. Mjor IA, Finn SB, Quigley MB. *The effect of calcium hydroxide and amalgam on non-carious, vital dentine*, Arch Oral Biol 1961;3: 283-291.
193. Moraschini V, da Costa LS, Dos Santos GO. *Effectiveness for dentin hypersensitivity treatment of non-carious cervical lesions: a meta-analysis*, Clin Oral Investig 2018;22: 617-631.

Telegram: @dental_k

www.konkur.in

194. Murray PE, About I, Lumley PJ, et al: *Postoperative pulpal and repair responses*, J Am Dent Assoc 2000;131: 321-329.
195. Murray PE, Smith AJ, Garcia-Godoy F, et al: *Comparison of operative procedure variables on pulpal viability in an ex vivo model*, Int Endod J 2008;41: 389-400.
196. Murray PE, Smith AJ, Windsor LJ, et al: *Remaining dentine thickness and human pulp responses*, Int Endod J 2003;36: 33-43.
197. Murray PE, Smyth TW, About I, et al: *The effect of etching on bacterial microleakage of an adhesive composite restoration*, J Dent 2002;30: 29-36.
198. Musselwhite JM, Klitzman B, Maixner W, et al: *Laser doppler flowmetry: a clinical test of pulpal vitality*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84: 411-419.
199. Nakabayashi N, Kojima K, Masuhara E. *The promotion of adhesion by the infiltration of monomers into tooth substrates*, J Biomed Mater Res 1982;16: 265-273.
200. Nanda R, Legan HL, Langeland K. *Pulpal and radicular response to maxillary osteotomy in monkeys*, Oral Surg Oral Med Oral Pathol 1982;53: 624-636.
201. Nathanson D. *Vital tooth bleaching: sensitivity and pulpal considerations*, J Am Dent Assoc 1997;128: 41S-44S.
202. Nayyar S, Tewari S, Arora B. *Comparison of human pulp response to total-etch and self-etch bonding agents*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: e45- e52.
203. Nixon CE, Saviano JA, King GJ, et al: *Histomorphometric study of dental pulp during*

Telegram: @dental_k

www.konkur.in

orthodontic tooth movement, J Endod 1993;19: 13-16.
204. Nomiyama K, Kitamura C, Tsujisawa T, et al: *Effects of lipopolysaccharide on newly established rat dental pulp-derived cell line with odontoblastic properties*, J Endod 2007;33: 1187-1191.
205. Nowicka A, Lipski M, Parafiniuk M, et al: *Response of human dental pulp capped with biodentine and mineral trioxide aggregate*, J Endod 2013;39: 743-747.
206. Nowicka A, Wilk G, Lipski M, et al: *Tomographic evaluation of reparative dentin formation after direct pulp capping with Ca(OH)2, MTA, biodentine, and dentin bonding system in human teeth*, J Endod 2015;41: 1234-1240.
207. Nup C, Rosenberg P, Linke H, et al: *Quantitation of catecholamines in inflamed human dental pulp by high-performance liquid chromatography*, J Endod 2001;27: 73-75.
208. Nyborg H, Brannstrom M. *Pulp reaction to heat*, J Prosthet Dent 1968;19: 605-612.
209. Odor TM, Pitt Ford TR, McDonald F. *Adrenaline in local anaesthesia: the effect of concentration on dental pulpal circulation and anaesthesia*, Endod Dent Traumatol 1994;10: 167-173.
210. Okamura K. *Histological study on the origin of dentinal immunoglobulins and the change in their localization during caries*, J Oral Pathol 1985;14: 680-689.
211. Okamura K, Maeda M, Nishikawa T, et al: *Dentinal response against carious invasion: localization of antibodies in odontoblastic body and process*, J Dent Res 1980;59: 1368-1373.
212. Olgart L, Gazelius B, Sundstrom F. *Intradental nerve activity and jaw-opening reflex in response to mechanical*

Telegram: @dental_k

www.konkur.in

deformation of cat teeth, Acta Physiol Scand 1988;133: 399-406.

213. Orchardson R, Peacock JM, Whitters CJ. *Effects of pulsed Nd:YAG laser radiation on action potential conduction in nerve fibres inside teeth in vitro*, J Dent 1998;26: 421-426.
214. Orchardson R, Whitters CJ. *Effect of HeNe and pulsed Nd:YAG laser irradiation on intradental nerve responses to mechanical stimulation of dentine*, Lasers Surg Med 2000;26: 241-249.
215. Ozturk M, Doruk C, Ozec I, et al: *Pulpal blood flow: effects of corticotomy and midline osteotomy in surgically assisted rapid palatal expansion*, J Craniomaxillofac Surg 2003;31: 97-100.
216. Paakkonen V, Ohlmeier S, Bergmann U, et al: *Analysis of gene and protein expression in healthy and carious tooth pulp with cDNA microarray and two-dimensional gel electrophoresis,* Eur J Oral Sci 2005;113: 369-379.
217. Paakkonen V, Vuoristo JT, Salo T, et al: *Comparative gene expression profile analysis between native human odontoblasts and pulp tissue*, Int Endod J 2008;41: 117-127.
218. Palosaari H, Wahlgren J, Larmas M, et al: *The expression of MMP-8 in human odontoblasts and dental pulp cells is down-regulated by TGF-beta1*, J Dent Res 2000;79: 77-84.
219. Pameijer CH, Stanley HR. *The disastrous effects of the "total etch" technique in vital pulp capping in primates [published erratum appears in Am J Dent 1998 Jun;11(3):148]*, Am J Dent 1998;11: S45- S54.
220. Pamir T, Dalgar H, Onal B. *Clinical evaluation of three desensitizing agents in relieving dentin hypersensitivity*, Oper Dent 2007;32: 544-548.

Telegram: @dental_k

www.konkur.in

221. Panitvisai P, Messer HH. *Cuspal deflection in molars in relation to endodontic and restorative procedures*, J Endod 1995;21: 57-61.
222. Pashley D. *Pulpodentin complex*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Carol Steam, IL 66.
223. Pashley DH. *The influence of dentin permeability and pulpal blood flow on pulpal solute concentrations*, J Endod 1979;5: 355-361.
224. Pashley DH. *In vitro simulations of in vivo bonding conditions*, Am J Dent 1991;4: 237-240.
225. Pashley DH. *Dynamics of the pulpo-dentin complex*, Crit Rev Oral Biol Med 1996;7: 104-133.
226. Pashley DH, Galloway SE. *The effects of oxalate treatment on the smear layer of ground surfaces of human dentine*, Arch Oral Biol 1985;30: 731-737.
227. Pashley DH, Kepler EE, Williams EC, et al: *Progressive decrease in dentine permeability following cavity preparation*, Arch Oral Biol 1983;28: 853-858.
228. Pashley DH, Tay FR, Yiu C, et al: *Collagen degradation by host-derived enzymes during aging*, J Dent Res 2004;83: 216-221.
229. Pashley EL, Talman R, Horner JA, et al: *Permeability of normal versus carious dentin*, Endod Dent Traumatol 1991;7: 207-211.
230. Paula AB, Laranjo M, Marto CM, et al: *Direct pulp capping: what is the most effective therapy? Systematic review and meta-analysis*, J Evid Based Dent Pract 2018;18: 298-314.
231. Pepersack WJ. *Tooth vitality after alveolar segmental osteotomy*, J Oral Maxillofac Surg 1973;1: 85-91.
232. Perdigao J, Geraldeli S, Hodges JS. *Total-etch versus*

Telegram: @dental_k

www.konkur.in

self-etch adhesive: effect on postoperative sensitivity, J Am Dent Assoc 2003;134: 1621-1629.

233. Perinetti G, Varvara G, Festa F, et al: *Aspartate aminotransferase activity in pulp of orthodontically treated teeth*, Am J Orthod Dentofacial Orthop 2004;125: 88-92.
234. Peters DD, Baumgartner JC, Lorton L. *Adult pulpal diagnosis. I. Evaluation of the positive and negative responses to cold and electrical pulp tests*, J Endod 1994;20: 506-511.
235. Petersson K, Soderstrom C, Kiani-Anaraki M, et al: *Evaluation of the ability of thermal and electrical tests to register pulp vitality*, Endod Dent Traumatol 1999;15: 127-131.
236. Polat S, Er K, Akpinar KE, et al: *The sources of laser doppler blood-flow signals recorded from vital and root canal treated teeth*, Arch Oral Biol 2004;49: 53-57.
237. Premdas CE, Pitt Ford TR. *Effect of palatal injections on pulpal blood flow in premolars*, Endod Dent Traumatol 1995;11: 274-278.
238. Quirynen M, Gijbels F, Jacobs R. *An infected jawbone site compromising successful osseointegration*, Periodontol 2000 2003;33: 129-144.
239. Qvist V, Stoltze K, Qvist J. *Human pulp reactions to resin restorations performed with different acid-etch restorative procedures*, Acta Odontol Scand 1989;47: 253-263.
240. Rakich DR, Wataha JC, Lefebvre CA, et al: *Effects of dentin bonding agents on macrophage mitochondrial activity*, J Endod 1998;24: 528-533.
241. Rakich DR, Wataha JC, Lefebvre CA, et al: *Effect of dentin bonding agents on the secretion of inflammatory*

Telegram: @dental_k

www.konkur.in

mediators from macrophages, J Endod 1999;25: 114-117.
242. Ramsay DS, Artun J, Bloomquist D. *Orthognathic surgery and pulpal blood flow: a pilot study using laser doppler flowmetry*, J Oral Maxillofac Surg 1991;49: 564-570.
243. Ranna V, Kellesarian SV, Feng C, et al: *Influence of the orthognathic surgical procedure Le Fort I osteotomy on the vascularity and neurosensory response of the dental pulp: A systematic review*, Quintessence Int 2016;47: 677-686.
244. Reeh ES, Messer HH, Douglas WH. *Reduction in tooth stiffness as a result of endodontic and restorative procedures*, J Endod 1989;15: 512-516.
245. Reeves R, Stanley HR. *The relationship of bacterial penetration and pulpal pathosis in carious teeth*, Oral Surg Oral Med Oral Pathol 1966;22: 59-65.
246. Reis A, Dalanhol AP, Cunha TS, et al: *Assessment of tooth sensitivity using a desensitizer before light-activated bleaching*, Oper Dent 2011;36: 12-17.
247. Reynolds RL. *The determination of pulp vitality by means of thermal and electrical stimuli*, Oral Surg Oral Med Oral Pathol 1966;22: 231-240.
248. Ricucci D, Loghin S, Siqueira JF Jr. *Correlation between clinical and histologic pulp diagnoses*, J Endod 2014;40: 1932-1939.
249. Ricucci D, Siqueira JF Jr, Loghin S, et al: *The cracked tooth: histopathologic and histobacteriologic aspects*, J Endod 2015;41: 343-352.
250. Ritter AV, Leonard RH Jr, St Georges AJ, et al: *Safety and stability of nightguard vital bleaching: 9 to 12 years post-treatment*, J Esthet Restor Dent 2002;14: 275-285.
251. Rodd HD, Boissonade FM. *Substance P expression in*

Telegram: @dental_k

www.konkur.in

human tooth pulp in relation to caries and pain experience [in process citation], Eur J Oral Sci 2000;108: 467-474.

252. Rodd HD, Boissonade FM. *Innervation of human tooth pulp in relation to caries and dentition type*, J Dent Res 2001;80: 389-393.
253. Rodd HD, Boissonade FM. *Vascular status in human primary and permanent teeth in health and disease*, Eur J Oral Sci 2005;113: 128-134.
254. Rotstein I, Simon JH. *Diagnosis, prognosis and decision-making in the treatment of combined periodontal-endodontic lesions*, Periodontol 2000 2004;34: 165-203.
255. Saito K, Nakatomi M, Ida-Yonemochi H, et al: *The expression of GM-CSF and osteopontin in immunocompetent cells precedes the odontoblast differentiation following allogenic tooth transplantation in mice*, J Histochem Cytochem 2011;59: 518-529.
256. Sarmast ND, Wang HH, Sajadi AS, et al: *Nonsurgical endodontic treatment of necrotic teeth resolved apical lesions on adjacent implants with retrograde/apical peri-implantitis: a case series with 2-year follow-up*, J Endod 2019;45: 645-650.
257. Sato M, Harada K, Okada Y, et al: *Blood-flow change and recovery of sensibility in the maxillary dental pulp after a single-segment Le Fort I osteotomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95: 660-664.
258. Schmalz G, Krifka S, Schweikl H. *Toll-like receptors, LPS, and dental monomers*, Adv Dent Res 2011;23: 302-306.
259. Schmalz G, Widbiller M, Galler KM. *Signaling molecules and pulp regeneration*, J Endod 2017;43: S7-S11.
260. Schroder U. *Effects of calcium hydroxide-containing*

Telegram: @dental_k

www.konkur.in

pulp-capping agents on pulp cell migration, proliferation, and differentiation, J Dent Res Spec No, 1985;64: 541-548.

261. Schuster GS, Caughman GB, Rueggeberg FA, et al: *Alterations in cell lipid metabolism by glycol methacrylate (HEMA)*, J Biomater Sci Polym Ed 1999;10: 1121-1133.
262. Seltzer S, Bender IB, Ziontz M. *The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp*, Oral Surg Oral Med Oral Pathol 1963;16: 846-871 contd.
263. Shabahang S, Bohsali K, Boyne PJ, et al: *Effect of teeth with periradicular lesions on adjacent dental implants*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 321-326.
264. Silva AF, Tarquinio SB, Demarco FF, et al: *The influence of haemostatic agents on healing of healthy human dental pulp tissue capped with calcium hydroxide*, Int Endod J 2006;39: 309-316.
265. Simon JH, Lies J. *Silent trauma*, Endod Dent Traumatol 1999;15: 145-148.
266. Sloan AJ, Smith AJ. *Stimulation of the dentine-pulp complex of rat incisor teeth by transforming growth factor-beta isoforms 1-3 in vitro*, Arch Oral Biol 1999;44: 149-156.
267. Smith AJ. *Pulpal responses to caries and dental repair*, Caries Res 2002;36: 223-232.
268. Smith AJ, Lesot H. *Induction and regulation of crown dentinogenesis: embryonic events as a template for dental tissue repair?*, Crit Rev Oral Biol Med 2001;12: 425-437.
269. Smith AJ, Tobias RS, Plant CG, et al: *Preliminary studies on the in vivo morphogenetic properties of dentine matrix proteins*, Biomaterials 1990;11: 22-24.

Telegram: @dental_k

www.konkur.in

270. Smith JG, Smith AJ, Shelton RM, et al: *Recruitment of dental pulp cells by dentine and pulp extracellular matrix components*, Exp Cell Res 2012;318: 2397-2406.
271. Soo-ampon S, Vongsavan N, Soo-ampon M, et al: *The sources of laser doppler blood-flow signals recorded from human teeth*, Arch Oral Biol 2003;48: 353-360.
272. Stabholz A, Rotstein L, Neev J, et al: *Efficacy of XeCl 308-nm excimer laser in reducing dye penetration through coronal dentinal tubules*, J Endod 1995;21: 266-268.
273. Stanley HR. *Pulp capping: conserving the dental pulp—can it be done? Is it worth it?*, Oral Surg Oral Med Oral Pathol 1989;68: 628-639.
274. Stanley HR, Swerdlow H. *An approach to biologic variation in human pulpal studies*, J Prosthet Dent 1964;14: 365.
275. Steiner DR. *The resolution of a periradicular lesion involving an implant*, J Endod 2008;34: 330-335.
276. Stenvik A. *Pulp and dentine reactions to experimental tooth intrusion (A histologic study–long-term effects)*, Rep Congr Eur Orthod Soc 1969; 449-464.
277. Stenvik A, Mjor IA. *Pulp and dentine reactions to experimental tooth intrusion. A histologic study of the initial changes*, Am J Orthod 1970;57: 370-385.
278. Stenvik A, Mjor IA. *The effect of experimental tooth intrusion on pulp and dentine*, Oral Surg Oral Med Oral Pathol 1971;32: 639-648.
279. Suhag K, Duhan J, Tewari S, et al: *Success of direct pulp capping using mineral trioxide aggregate and calcium hydroxide in mature permanent molars with pulps exposed during carious tissue removal: 1-year follow-up*, J Endod 2019.
280. Sussman HI. *Endodontic pathology leading to implant*

Telegram: @dental_k

www.konkur.in

failure—a case report, J Oral Implantol 1997;23: 112-115 discussion 115–116.

281. Sussman HI. *Tooth devitalization via implant placement: a case report*, Periodontal Clin Investig 1998;20: 22-24.
282. Swerdlow H, Stanley HR Jr. *Reaction of the human dental pulp to cavity preparation. I. Effect of water spray at 20,000 rpm*, J Am Dent Assoc 1958;56: 317-329.
283. Swift EJ Jr, Trope M. *Treatment options for the exposed vital pulp*, Pract Periodontics Aesthet Dent 1999;11: 735-739; quiz 40.
284. Tagami J, Hosoda H, Burrow MF, et al: *Effect of aging and caries on dentin permeability*, Proc Finn Dent Soc Suppl 1, 1992;88: 149-154.
285. Takamori K. *A histopathological and immunohistochemical study of dental pulp and pulpal nerve fibers in rats after the cavity preparation using Er:YAG laser*, J Endod 2000;26: 95-99.
286. Tanabe K, Yoshiba K, Yoshiba N, et al: *Immunohistochemical study on pulpal response in rat molars after cavity preparation by Er:YAG laser*, Eur J Oral Sci 2002;110: 237-245.
287. Teixeira LS, Demarco FF, Coppola MC, et al: *Clinical and radiographic evaluation of pulpotomies performed under intrapulpal injection of anaesthetic solution*, Int Endod J 2001;34: 440-446.
288. Tjan AH, Grant BE, Godfrey MF, et al: *Temperature rise in the pulp chamber during fabrication of provisional crowns*, J Prosthet Dent 1989;62: 622-626.
289. Tokita Y, Sunakawa M, Suda H. *Pulsed Nd:YAG laser irradiation of the tooth pulp in the cat: I. Effect of spot lasing*, Lasers Surg Med 2000;26: 398-404.
290. Tomas-Catala CJ, Collado-Gonzalez M, Garcia-Bernal D,

Telegram: @dental_k

www.konkur.in

et al: *Biocompatibility of New pulp-capping materials NeoMTA Plus, MTA Repair HP, and Biodentine on human dental pulp stem cells*, J Endod 2018;44: 126-132.

291. Tomson PL, Grover LM, Lumley PJ, et al: *Dissolution of bio-active dentine matrix components by mineral trioxide aggregate*, J Dent 2007;35: 636-642.
292. Torabinejad M, Kiger RD. *A histologic evaluation of dental pulp tissue of a patient with periodontal disease*, Oral Surg Oral Med Oral Pathol 1985;59: 198-200.
293. Tranasi M, Sberna MT, Zizzari V, et al: *Microarray evaluation of age-related changes in human dental pulp*, J Endod 2009;35: 1211-1217.
294. Trantor IR, Messer HH, Birner R. *The effects of neuropeptides (calcitonin gene-related peptide and substance P) on cultured human pulp cells*, J Dent Res 1995;74: 1066-1071.
295. Trowbridge H, Edwall L, Panopoulos P. *Effect of zinc oxide-eugenol and calcium hydroxide on intradental nerve activity*, J Endod 1982;8: 403-406.
296. Tsatsas BG, Frank RM. *Ultrastructure of the dentimal tubular substances near the dentino-enamel junction*, Calcif Tissue Res 1972;9: 238-242.
297. Turner DF, Marfurt CF, Sattelberg C. *Demonstration of physiological barrier between pulpal odontoblasts and its perturbation following routine restorative procedures: a horseradish peroxidase tracing study in the rat*, J Dent Res 1989;68: 1262-1268.
298. Tyndall AA, Brooks SL. *Selection criteria for dental implant site imaging: a position paper of the American Academy of Oral and Maxillofacial radiology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 630-637.

Telegram: @dental_k

www.konkur.in

299. Tziafas D. *Basic mechanisms of cytodifferentiation and dentinogenesis during dental pulp repair*, Int J Dev Biol 1995;39: 281-290.
300. Tziafas D, Kolokuris I. *Inductive influences of demineralized dentin and bone matrix on pulp cells: an approach of secondary dentinogenesis*, J Dent Res 1990;69: 75-81.
301. Umberto R, Claudia R, Gaspare P, et al: *Treatment of dentine hypersensitivity by diode laser: a clinical study*, Int J Dent 2012;2012: 858950.
302. Unemori M, Matsuya Y, Akashi A, et al: *Composite resin restoration and postoperative sensitivity: clinical follow-up in an undergraduate program*, J Dent 2001;29: 7-13.
303. Valderhaug J, Jokstad A, Ambjornsen E, et al: *Assessment of the periapical and clinical status of crowned teeth over 25 years*, J Dent 1997;25: 97-105.
304. Vale WA. *Cavity preparation*, Ir Dent Rev 1956;2: 33-41.
305. Vedtofte P. *Pulp canal obliteration after Le Fort I osteotomy*, Endod Dent Traumatol 1989;5: 274-278.
306. Versluis A, Douglas WH, Cross M, et al: *Does an incremental filling technique reduce polymerization shrinkage stresses?*, J Dent Res 1996;75: 871-878.
307. Versluis A, Tantbirojn D, Douglas WH. *Distribution of transient properties during polymerization of a light-initiated restorative composite*, Dent Mater 2004;20: 543-553.
308. von Troil B, Needleman I, Sanz M. *A systematic review of the prevalence of root sensitivity following periodontal therapy*, J Clin Periodontol Suppl 3, 2002;29: 173-177 discussion 195–196.
309. Vongsavan K, Samdrup T, Kijsamanmith K, et al: *The*

Telegram: @dental_k

www.konkur.in

effect of intraosseous local anesthesia of 4% articaine with 1:100,000 epinephrine on pulpal blood flow and pulpal anesthesia of mandibular molars and canines, Clin Oral Investig 2019;23: 673-680.

310. Vongsavan N, Matthews RW, Matthews B. *The permeability of human dentine in vitro and in vivo*, Arch Oral Biol 2000;45: 931-935.
311. Wakabayashi H, Hamba M, Matsumoto K, et al: *Effect of irradiation by semiconductor laser on responses evoked in trigeminal caudal neurons by tooth pulp stimulation*, Lasers Surg Med 1993;13: 605-610.
312. Wennberg A, Mjor IA, Heide S. *Rate of formation of regular and irregular secondary dentin in monkey teeth*, Oral Surg Oral Med Oral Pathol 1982;54: 232-237.
313. Widbiller M, Austah O, Lindner SR, et al: *Neurotrophic proteins in dentin and their effect on trigeminal sensory neurons*, J Endod 2019.
314. Wigdor H, Abt E, Ashrafi S, et al: *The effect of lasers on dental hard tissues*, J Am Dent Assoc 1993;124: 65-70.
315. Yiu CK, King NM, Suh BI, et al: *Incompatibility of oxalate desensitizers with acidic, fluoride-containing total-etch adhesives*, J Dent Res 2005;84: 730-735.
316. Yoshiba K, Yoshiba N, Iwaku M. *Class II antigen-presenting dendritic cell and nerve fiber responses to cavities, caries, or caries treatment in human teeth*, J Dent Res 2003;82: 422-427.
317. Yoshiba N, Yoshiba K, Iwaku M, et al: *Immunohistochemical localizations of class II antigens and nerve fibers in human carious teeth: HLA-DR immunoreactivity in Schwann cells*, Arch Histol Cytol 1998;61: 343-352.
318. Yoshiba N, Yoshiba K, Nakamura H, et al:

Telegram: @dental_k

www.konkur.in

Immunohistochemical localization of HLA-DR-positive cells in unerupted and erupted normal and carious human teeth, J Dent Res 1996;75: 1585-1589.

319. Yoshida S, Oshima K, Tanne K. *Biologic responses of the pulp to single-tooth dento-osseous osteotomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 152-160.
320. Yoshiyama M, Masada J, Uchida A, et al: *Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin*, J Dent Res 1989;68: 1498-1502.
321. Yoshiyama M, Tay FR, Doi J, et al: *Bonding of self-etch and total-etch adhesives to carious dentin*, J Dent Res 2002;81: 556-560.
322. Zach L, Cohen G. *Pulp response to externaly applied heat*, Oral Surg Oral Med Oral Pathol 1965;19: 515-530.
323. Zaia AA, Nakagawa R, De Quadros I, et al: *An in vitro evaluation of four materials as barriers to coronal microleakage in root-filled teeth*, Int Endod J 2002;35: 729-734.
324. Zhang J, Kawashima N, Suda H, et al: *The existence of CD11c1 sentinel and F4/801 interstitial dendritic cells in dental pulp and their dynamics and functional properties*, Int Immunol 2006;18: 1375-1384.
325. Zhong S, Naqvi A, Bair E, et al: *Viral microRNAs identified in human dental pulp*, J Endod 2017;43: 84-89.
326. Zhong S, Zhang S, Bair E, et al: *Differential expression of microRNAs in normal and inflamed human pulps*, J Endod 2012;38: 746-752.

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15: Microbiology of endodontic infections

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CHAPTER OUTLINE

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Apical periodontitis is essentially an inflammatory disease of microbial etiology primarily caused by infection of the root canal system.[217] Although chemical and physical factors can induce periradicular inflammation, a large body of scientific evidence indicates that endodontic infection is essential to the progression and perpetuation of the different forms of apical periodontitis.[18,91,123,273] Endodontic infection develops in root canals devoid of host defenses, as a consequence of either pulp necrosis (as a sequel to caries, trauma, periodontal disease, or invasive operative procedures) or pulp removal for treatment.

Microorganisms other than bacteria have been detected in association with endodontic infections, including fungi, archaea, and viruses.[54,194,254,261,291] However, given their high prevalence, dominance, organization, and pathogenicity, bacteria can be regarded as the major microorganisms implicated in the pathogenesis of apical periodontitis. In advanced stages of the endodontic infectious process, bacterial structures resembling biofilms are

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commonly observed adhering to the canal walls.[124,164,245] Consequently, apical periodontitis has been included in the roll of biofilm-related oral diseases.[256] Bacteria colonizing the root canal system enter in contact with the periradicular tissues via apical/lateral foramina or root perforations. As a consequence of the encounter between bacteria and host defenses, inflammatory changes take place in the periradicular tissues and give rise to the development of apical periodontitis. Depending on several bacterial and host-related factors, apical periodontitis can be symptomatic (acute) or asymptomatic (chronic).

The ultimate goal of endodontic treatment is either to prevent the development of apical periodontitis or, in cases where the disease is already present, to create appropriate conditions for periradicular tissue healing. The intent is to preserve the tooth and associated periradicular tissues in healthy conditions. Because apical periodontitis is an infectious disease, the rationale for endodontic treatment is to eradicate the occurring infection or prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues. The cardinal principle of any health care profession is the thorough understanding of disease etiology and pathogenesis, which provides a framework for effective treatment. In this context, understanding the microbiologic aspects of apical periodontitis is the basis for sound endodontic practice and sets the stage for an evidence-based therapeutic approach. This chapter focuses on diverse aspects of endodontic microbiology, including pathogenetic, taxonomic, morphologic, and ecologic issues.

Apical periodontitis as an infectious disease

The first recorded observation of bacteria in the root canal dates back to the 17th century and the Dutch amateur microscope builder Antony van Leeuwenhoek (1632–1723). He reported that the root canals of a decayed tooth “were stuffed with a soft matter” and that “the whole stuff” seemed to be alive.[44] At that time, the role of Leeuwenhoek’s “animalcules” in disease causation was unsuspected. It took almost 200 years until his observation was confirmed and a cause-and-effect relationship between bacteria and apical periodontitis was suggested. This occurred specifically in 1894, when

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Willoughby Dayton Miller, an American dentist working at the laboratory of Robert Koch in Berlin, Germany, published a milestone study reporting on the association between bacteria and apical periodontitis after an analysis of samples collected from root canals.[119] By means of bacterioscopy of the canal samples, he found bacterial cells in the three basic morphologies known at the time: cocci, bacilli, and spirilla (or spirochetes) (Fig. 15.1). Morphologically, the endodontic microbiota was clearly different in the coronal, middle, and apical parts of the root canal. Spirochetes were found in high frequencies in abscessed cases, and a pathogenic role was suspected for these bacteria. Most of the bacteria Miller observed under light microscopy could not be cultivated using the technology available at that time. Those bacteria were conceivably anaerobic bacteria, which were only successfully cultivated about 50 to 100 years later with the advent of anaerobic culture techniques. However, it is now widely recognized that a large number of bacterial species living in diverse environments still remain to be cultivated by current technology,[6,154,155] and the root canal is no exception (discussed later in this chapter). Based on his findings, Miller raised the hypothesis that bacteria were the causative agents of apical periodontitis.

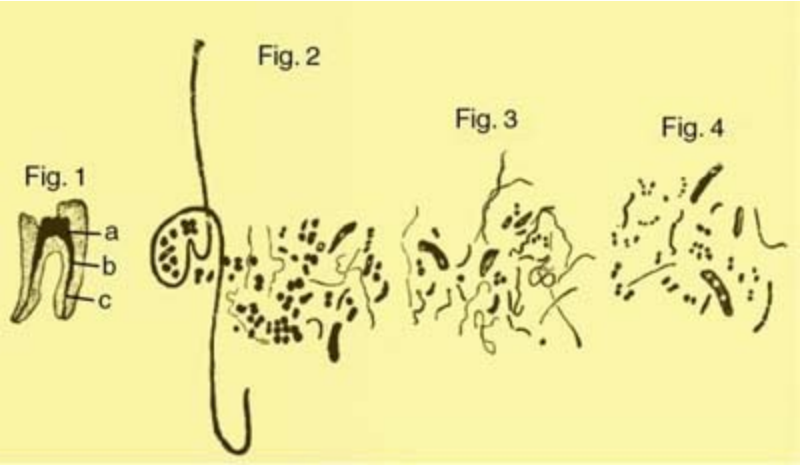

FIG. 15.1 Drawings from Miller's classic paper showing different bacterial forms in a root canal sample observed by microscopy.

Set of four hand drawings are marked figure 1 to 4 as follows:

Figure 1: Diagram of tooth shows three labels marked a to c.

Figure 2: Irregularly spherical shape of bacteria.

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Figure 3: Rod-shaped bacteria.

Figure 4: Spirally twisted bacteria.

Approximately 70 years after Miller's classic study, his assumptions were confirmed by an elegant study from Kakehashi et al.[91] These authors investigated the response of exposed dental pulps to the oral cavity in conventional and germ-free rats. Histologic evaluation was performed and revealed that pulp necrosis and apical periodontitis lesions developed in all conventional rats; however, the exposed pulps of germ-free rats not only remained vital but also repaired themselves with hard-tissue formation. Dentin-like tissue sealed the exposure area and isolated the pulps again from the oral cavity.

The important role of bacteria in the etiology of apical periodontitis was further confirmed by Sundqvist's study.[273] This author applied advanced anaerobic culturing techniques to the evaluation of bacteria occurring in the root canals of teeth whose pulps became necrotic after trauma. Bacteria were found only in the root canals of teeth exhibiting radiographic evidence of apical periodontitis, confirming the infectious cause of this disease. Anaerobic bacteria accounted for more than 90% of the isolates. Findings from the Sundqvist's study also demonstrated that in the absence of infection, the necrotic pulp tissue itself and stagnant tissue fluid in the root canal do not induce or perpetuate apical periodontitis lesions.

Möller et al.[123] also provided strong evidence about the microbial causation of apical periodontitis. Their study on monkeys demonstrated that only devitalized pulps that were infected induced apical periodontitis lesions, whereas devitalized and noninfected pulps showed an absence of significant pathologic changes in the periradicular tissues. In addition to corroborating the importance of microorganisms for the development of apical periodontitis, this study also confirmed that necrotic pulp tissue per se is unable to induce and maintain an apical periodontitis lesion.

Bacteria causing apical periodontitis are primarily organized in biofilms colonizing the root canal system. Bacterial agglomerations resembling what is known nowadays as biofilms had been observed by several morphologic studies,[124,210,245] but it was not until the study by Ricucci and Siqueira[164] that the high prevalence of bacterial biofilms was consistently disclosed and a

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strong association with primary and posttreatment apical periodontitis was reported (see the section Spatial Distribution of the Endodontic Microbiota for a further discussion of biofilms in endodontic infections).

Routes of root canal infection

Under normal conditions, the pulpodentin complex is sterile and isolated from the oral microbiota by overlying enamel and cementum. In the event that the integrity of these natural layers is breached (e.g., as a result of caries, trauma-induced fractures and cracks, restorative procedures, scaling and root planning, attrition, abrasion) or naturally absent (e.g., because of gaps in the cementum coating at the cervical root surface), the pulpodentin complex is exposed to the oral environment. The pulpodentin complex is then challenged by bacteria present in caries lesions, saliva bathing the exposed area, or dental plaque formed on the exposed area. Bacteria from subgingival biofilms associated with periodontal disease may also have access to the pulp via dentinal tubules at the cervical region of the tooth and lateral or apical foramina. Bacteria and other microorganisms may also have access to the root canal any time during or after endodontic intervention, emphasizing the need for maintaining strict asepsis during treatment and promoting an adequate coronal seal.

Whenever dentin is exposed, the pulp is put at risk of infection as a consequence of the permeability of normal dentin dictated by its tubular structure (Fig. 15.2).[140] Dentinal tubules traverse the entire width of the dentin and have a conical conformation, with the largest diameter located near the pulp (mean, 2.5 μm) and the smallest diameter near the enamel or cementum (mean, 0.9 μm).[58] The smallest tubule diameter is entirely compatible with the cell diameter of most oral bacterial species, which usually ranges from 0.2 to 0.7 μm. One might assume that once exposed, dentin offers an unimpeded pathway for bacteria to reach the pulp via tubules. However, it has been demonstrated that bacterial invasion of dentinal tubules occurs more rapidly with a nonvital pulp than with a vital pulp.[128] With a vital pulp, outward movement of dentinal fluid and the tubular contents (including odontoblast processes, collagen fibrils, and the sheathlike lamina limitans that lines the tubules) influence dentinal permeability and can

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conceivably delay intratubular invasion by bacteria. Because of the presence of tubular contents, the functional or physiologic diameter of the tubules is only 5% to 10% of the anatomic diameter seen by microscopy.[117] Other factors such as dentinal sclerosis, tertiary dentin, smear layer, and intratubular deposition of fibrinogen also reduce dentin permeability and thereby limit or even impede bacterial progression to the pulp via dentinal tubules.[142] Host defense molecules, such as antibodies and components of the complement system, may also be present in the dentinal fluid of vital teeth and can assist in the protection against deep bacterial invasion of dentin.[3,133,134] As long as the pulp is vital, dentinal exposure does not represent a significant route of pulpal infection, except when dentin thickness is considerably reduced or when the dentin permeability is significantly increased.

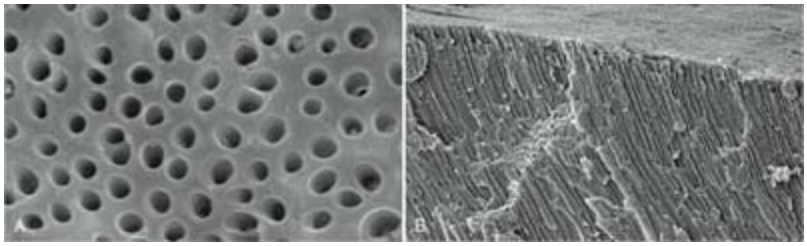

FIG. 15.2 **A,** Scanning electron micrographs of dentin showing tubules in cross-sectional view (magnification ×850). **B,** Longitudinal view (magnification ×130).

A) Scanning electron micrograph shows round hollow spaces on the surface.

B) Scanning electron micrograph shows slanting, fine, longitudinal lines.

Most of the bacteria in the carious process are nonmotile; they invade dentin by repeated cell division, which pushes cells into tubules. Bacterial cells may also be forced into tubules by hydrostatic pressures developed on dentin during mastication.[118] Bacteria inside tubules under a deep carious lesion can reach the pulp even before frank pulpal exposure.[85] As previously mentioned, it has been assumed that the pulp will not be easily infected if it is still vital. The few bacteria that reach the pulp may not be significant, because the vital pulp can eliminate such a transient infection and rapidly clear or remove bacterial products. This efficient clearance mechanism tends to prevent injurious agents from reaching a high enough concentration to

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induce significant inflammatory reactions.[141] On the other hand, if the vitality of the pulp is compromised and the defense mechanisms are impaired, even a small amount of bacteria may initiate pulpal infection.

Direct exposure of the dental pulp to the oral cavity is the most obvious route of endodontic infection. Caries is the most common cause of pulp exposure, but bacteria may also reach the pulp via direct pulp exposure as a result of iatrogenic restorative procedures or trauma. The exposed pulp tissue comes in direct contact with oral bacteria from carious lesions, saliva, or plaque accumulated onto the exposed surface. Almost invariably, exposed pulps will undergo inflammation and necrosis and become infected. The time elapsed between pulp exposure and infection of the entire canal is unpredictable, but it is usually a slow process.[35]

The egress of bacteria and their products from infected root canals through apical, lateral, or furcation foramina, dentinal tubules, and iatrogenic root perforations can directly affect the surrounding periodontal tissues and give rise to pathologic changes in these tissues. Conceptually, bacteria in subgingival plaque biofilms associated with periodontal disease could reach the pulp by the same pathways intracanal bacteria reach the periodontium and could thereby exert harmful effects on the pulp. It has been demonstrated that although degenerative and inflammatory changes of different degrees may occur in the pulp of teeth with associated periodontal disease, significant pulpal necrosis only develops if the periodontal biofilm reaches the apical foramen, leading to irreversible damage to the main blood vessels that penetrate through this foramen (Fig. 15.3).[102] After the pulp becomes necrotic, periodontal bacteria can invade the root canal system via apical foramina, lateral canals, and exposed dentinal tubules.

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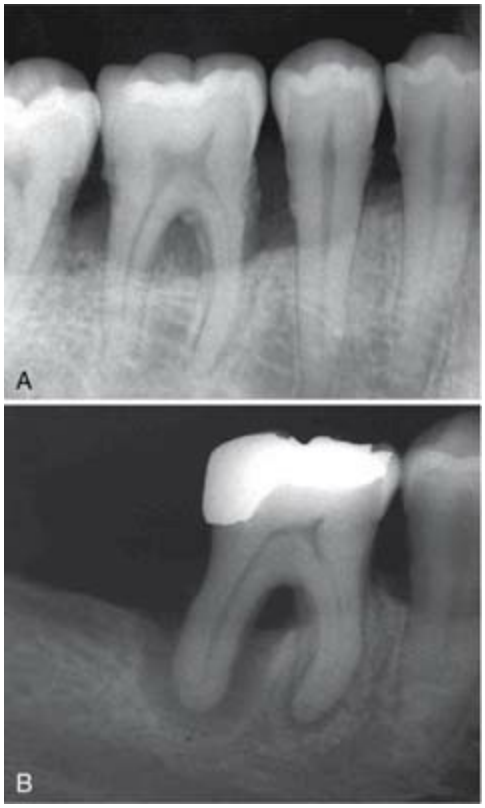

FIG. 15.3 Periodontal disease **(A)** mostly affects the pulp vitality when the subgingival biofilm reaches the apical foramen **(B)**.

A) Radiograph of four teeth shows blur radiolupency in the pulp horn.

B) Radiograph of a teeth shows gray patch surrounding the apical foramen with dentine depicted as radiopaque.

In the past, it was claimed that bacteria could reach the pulp by anachoresis.[72] Theoretically, bacteria can be transported in the blood or lymph to an area of tissue damage, where they leave the vessel, enter the damaged tissue, and establish an infection.[61,170] However, there is no clear evidence showing that this process represents a route for root canal infection. Bacteria could not be recovered from unfilled root canals of cats when the bloodstream was experimentally infected, unless the root canals were overinstrumented during the period of bacteremia, with resulting injury to periodontal blood vessels and blood seepage into the canal.[40] Another argument against anachoresis as a route for pulpal infection comes from the study by Möller et al.,[123] who induced pulpal necrosis in monkeys' teeth and reported that all cases of aseptic necrosis remained bacteria-free after 6 to 7

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months of observation.

Bacteria have been isolated from traumatized teeth with necrotic pulps with apparently intact crowns.[273,306] Although anachoresis has been suggested to be the mechanism through which these traumatized teeth become infected,[72] current evidence indicates that the main pathway of root canal infection in these cases is dentinal exposure due to enamel cracks.[111,112] Macro- and microcracks in enamel can occur in most teeth (not only traumatized teeth) and do not necessarily end at the enamel-dentin junction; they can extend deep into the dentin.[112] A large number of dentinal tubules are exposed to the oral environment by a single crack. Cracks can be clogged with plaque biofilm and provide portals of entry for bacteria. If the pulp remains vital after trauma, bacterial penetration into tubules is counteracted by the dentinal fluid and tubular contents, as discussed earlier, and pulpal health is not usually jeopardized. But if the pulp becomes necrotic as a consequence of trauma, it loses the ability to protect itself against bacterial invasion, and regardless of dentin thickness, dentinal tubules may become true avenues through which bacteria reach and colonize the necrotic pulp.

Whatever the route of bacterial access to the root canal, necrosis of pulp tissue is a prerequisite for the establishment of primary endodontic infections. To reiterate: If the pulp is vital, it can protect itself against bacterial invasion and colonization. If the pulp becomes necrotic as a sequel to caries, trauma, operative procedures, or periodontal disease, then it can be easily infected.

Another situation in which the root canal system is devoid of host defenses relates to cases in which the pulp was removed for treatment. Microbial penetration in the canal can occur during treatment, between appointments, or even after root canal obturation. The main causes of microbial introduction into the canal *during treatment* include residual dental plaque biofilm, calculus, or caries on the tooth crown; leaking rubber dam; contaminated endodontic instruments (e.g., after touching with the fingers); and contaminated irrigant solutions or other solutions of intracanal use (e.g., saline solution, distilled water, citric acid). Bacteria can also enter the root canal system *between appointments* by leakage through the temporary restorative material; breakdown, fracture, or loss of the temporary restoration; fracture of the tooth structure; and in teeth left open for drainage. Bacteria can penetrate the root canal system even *after completion of the root canal*

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obturation by leakage through the temporary or permanent restorative material; breakdown, fracture, or loss of the temporary/permanent restoration; fracture of the tooth structure; recurrent decay; or delay in the placement of the permanent restoration.[227]

Mechanisms of microbial pathogenicity and virulence factors

Pathogenicity is a term that refers to the ability of a microorganism to cause disease. The degree of pathogenicity of a microorganism is referred to as *virulence*. Microbial secreted products, structural cellular components, and strategies that contribute to pathogenicity are regarded as *virulence factors*. One example of bacterial strategy that contributes to pathogenicity is the ability to form biofilms, which confers protection against host defenses, microbial competitors, and antimicrobial agents. Some microorganisms routinely cause disease in a given host and are called *primary pathogens*. Other microorganisms cause disease only when host defenses are impaired and are called *opportunistic pathogens*. Bacteria that make up the normal resident microbiota are usually present as harmless commensals and live in balance with the host. One of the greatest beneficial effects of human microbiota is probably the tendency to protect the host from exogenous infections by excluding other microorganisms. Nevertheless, in certain situations, the balance may be disturbed by a decrease in the normal level of resistance, and then the commensal bacteria are usually the first to take advantage. Most bacteria involved with endodontic infections are normal inhabitants of the oral microbiota that exploit changes in the balance of the host–bacteria relationship, becoming opportunistic pathogens.

The bacterial species involved with the pathogenesis of primary apical periodontitis may have participated in the early stages of pulp inflammation and necrosis, or they may have gained entry into the root canal space any time after pulpal necrosis. In the former situation, involved bacteria are usually those present in the advanced front of caries lesions and from saliva bathing the affected area. Bacteria in caries lesions form authentic biofilms adhered to dentin (Fig. 15.4). Diffusion of bacterial products through dentinal tubules induces pulp inflammation long before the tissue is exposed. After

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frank exposure, the surface of the pulp tissue can be colonized and covered by bacteria present in the caries biofilm. The exposed pulp tissue enters in direct contact with bacteria and their products and responds with severe inflammation. Some tissue invasion by the involved bacteria may occur. Bacteria in the forefront of infection have to survive the attack from the host defenses and at the same time acquire nutrients to keep themselves alive. In this bacteria–pulp clash, the pulp invariably is "defeated" and becomes necrotic, so bacteria move forward and "occupy the territory"—that is, they colonize the necrotic tissue. These events advance through tissue compartments, coalesce, and move toward to the apical part of the canal until virtually the entire root canal is necrotic and infected (Fig. 15.5). At this stage, involved bacteria can be regarded as the early root canal colonizers or pioneer species.

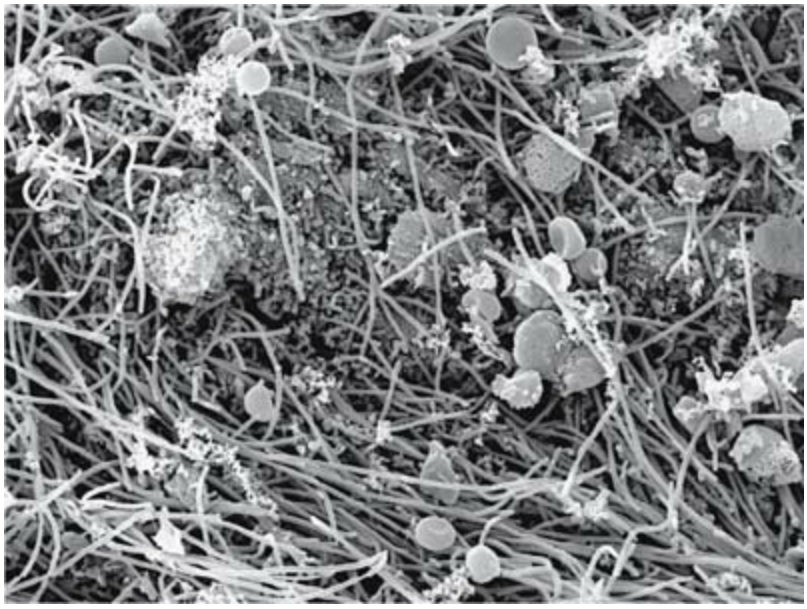

FIG. 15.4 Scanning electron micrograph showing a bacterial biofilm covering dentin in a deep carious lesion. Note the presence of different bacterial morphotypes (magnification ×3500).

Scanning electron micrograph shows a layer of bacteria covering the dentin with bacteria depicted as round structures with long tails.

Source: (From Torabinejad M, Walton RE: *Endodontics: principles and practice,* ed 5, St. Louis, 2015, Saunders/Elsevier.)

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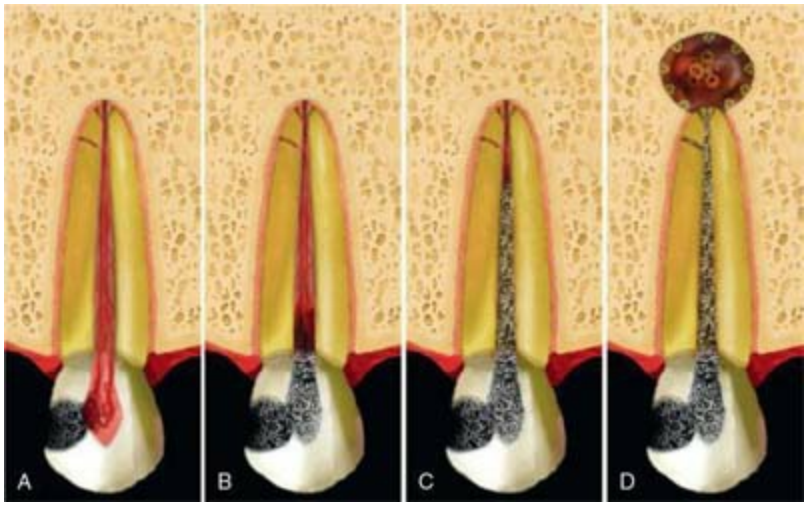

FIG. 15.5 Dynamics of pulp response from caries exposure **(A)** to pulp inflammation **(B)** to pulp necrosis **(C)** to apical periodontitis formation **(D)**.

Set of four illustrations marked A through D shows changes in pulp brought about on caries exposure depicted as necrosis of pulp moving from crown to root.

Early colonizers play an important role in the initiation of the apical periodontitis disease process. Moreover, they may significantly modify the environment, making it conducive to the establishment of other bacterial groups. These new species may have access to the canal via coronal exposure or exposed dentinal tubules, establish themselves, and contribute to a shift in the microbiota. Rearrangement in the proportions of the pioneer species and latecomers occurs, and, as the environment changes, some early colonizers may even disappear. With the passage of time, the endodontic microbiota becomes more and more structurally and spatially organized.

Some virulence attributes required for pathogens to thrive in other sites may be of no value for bacteria that reach the root canal after necrosis—for instance, the ability to evade host defenses. This is because latecomers face no significant opposition from host defenses, which are no longer active in the canal after necrosis. Although colonization may appear an easy task for late colonizers, other environmental factors (e.g., interaction with pioneer species, oxygen tension, nutrient availability) will determine whether new species entering the canal will succeed in establishing themselves and join the early colonizers to make up a dynamic mixed community in the root canal. Ultimately, the root canals of teeth with radiographically detectable apical

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periodontitis lesions harbor both early colonizers that managed to stay in the canals and late colonizers that managed to adapt to the new environmental conditions.[234]

Periradicular inflammation can be observed, even before the frontline of intracanal bacterial infection reaches the apical foramen.[8,124,265,307] Bacteria exert their pathogenicity by wreaking havoc on the host tissues through direct or indirect mechanisms. Bacterial virulence factors that cause direct tissue harm include those that damage host cells or the intercellular matrix of the connective tissue. Secreted bacterial products, such as enzymes, exotoxins, heat-shock proteins, and metabolic end products, are the main examples.[234] Furthermore, bacterial structural components, including lipopolysaccharide (LPS), peptidoglycan, lipoteichoic acid (LTA), fimbriae, flagella, outer membrane proteins and vesicles, lipoproteins, DNA, and exopolysaccharides, can act as modulins by stimulating the development of host immune reactions capable not only of defending the host against infection but also of causing severe tissue destruction (Fig. 15.6).[80,234,287] For instance, inflammatory and noninflammatory host cells are stimulated by bacterial components to release proinflammatory cytokines that are involved in bone resorption characteristically observed in apical periodontitis.[201] Another example of indirect damage is pus formation in acute apical abscesses, which results from the destruction of the connective extracellular matrix by oxygen-derived free radicals and lysosomal enzymes released by polymorphonuclear leukocytes in response to bacteria.[285] Although direct damage caused by bacterial products may certainly be involved in the pathogenesis of apical periodontitis, bacterial indirect destructive effects seem to be more significant in this regard.[201]

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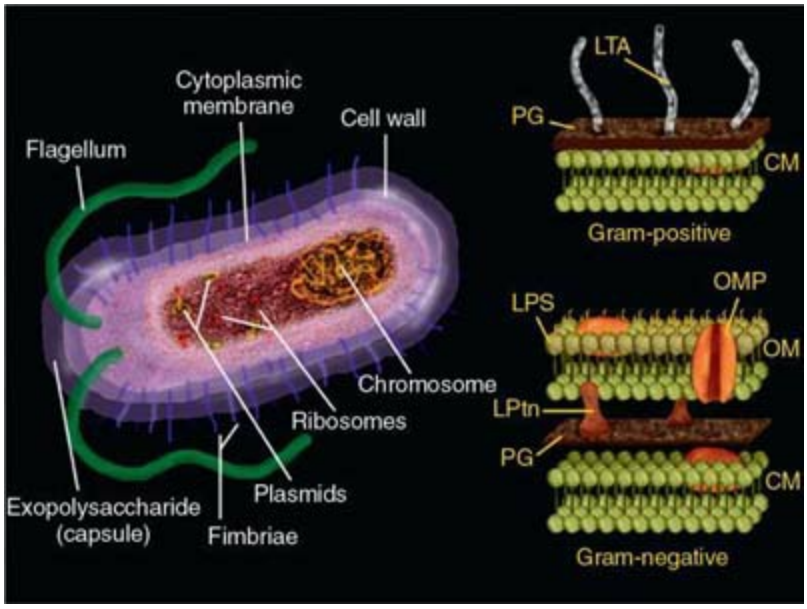

FIG. 15.6 Bacterial cell and its structural components that can act as virulence factors. *Right:* A detailed scheme of the bacterial cell walls from gram-positive and gram-negative bacteria. *CM,* Cytoplasmic membrane; *LPS,* lipopolysaccharide (endotoxin); *LPtn,* lipoproteins; *LTA,* lipoteichoic acid; *OM,* outer membrane; *OMP,* outer membrane protein; *PG,* peptidoglycan.

On the left, cross-sectional diagram of bacterial cell shows labels for cell wall, cytoplasmic membrane, exopolysaccharide (capsule), chromosome, ribosomes, plasmids, flagellum, and fimbriae. On the right, gram-positive cells are shown to have a thick peptidoglycan layer between cytoplasmic membrane, and lipoteichoic acid present on peptidoglycan layer while gram negative cells are shows to have a outer membrane and peptidoglycan layer connected by lipoproteins. The outer membrane lipopolysaccharide and outer membrane proteins while peptidoglycan layer is followed by cytoplasmic membrane.

Apical periodontitis is a multifactorial disease that is resultant of the interplay of many host and bacterial factors. Few if any of the putative endodontic pathogens are individually capable of inducing all of the events involved in the pathogenesis of the different forms of apical periodontitis. Therefore, the process requires an integrated and orchestrated interaction of the selected members of the mixed endodontic microbiota and their respective virulence attributes.[295] LPS, which is exclusive of gram-negative bacteria, has been detected in high concentrations in canals of teeth with large and/or symptomatic apical periodontitis, and in conditions of persistent exudation.[38,64,84,88,205,206] Although LPS has been widely studied and quoted

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as an important virulence factor with regard to the pathogenesis of apical periodontitis,[39,127] many other bacterial mediators are also expected to contribute to disease causation. For instance, some cases of primary infections and many cases of secondary/persistent infections may harbor exclusively gram-positive bacteria, which indicates that the involvement of other factors must not be overlooked. Indeed, LTA, which is exclusive of gram-positive bacteria, has been detected in all teeth with posttreatment apical periodontitis.[10] Many bacterial secreted products have been detected in endodontic infections.[79,152,153] Therefore, the pathogenesis of different forms of apical periodontitis and even the same form in different individuals is unlikely to follow a stereotyped course with regard to the bacterial mediators involved.[218]

Spatial distribution of the endodontic microbiota

Mounting evidence indicates that apical periodontitis, like caries and periodontal diseases, is also a biofilm-related disease. Morphologic studies have shown that the root canal microbiota in primary infections is dominated by bacterial morphotypes that include cocci, rods, filaments, and spirilla (spirochetes) (Fig. 15.7). Fungal cells are sporadically found (Fig. 15.8).[210,245] Although planktonic bacterial cells suspended in a fluid phase and enmeshed in necrotic pulp tissue can be observed in the main root canal, most bacteria colonizing the root canal system usually grow in sessile multispecies biofilms adhered to the dentinal walls (Fig. 15.9).[124,165,245] Lateral canals, apical ramifications, and isthmuses connecting main canals may also be clogged with bacterial biofilms (Figs. 15.10 and 15.11).[160,163,290]

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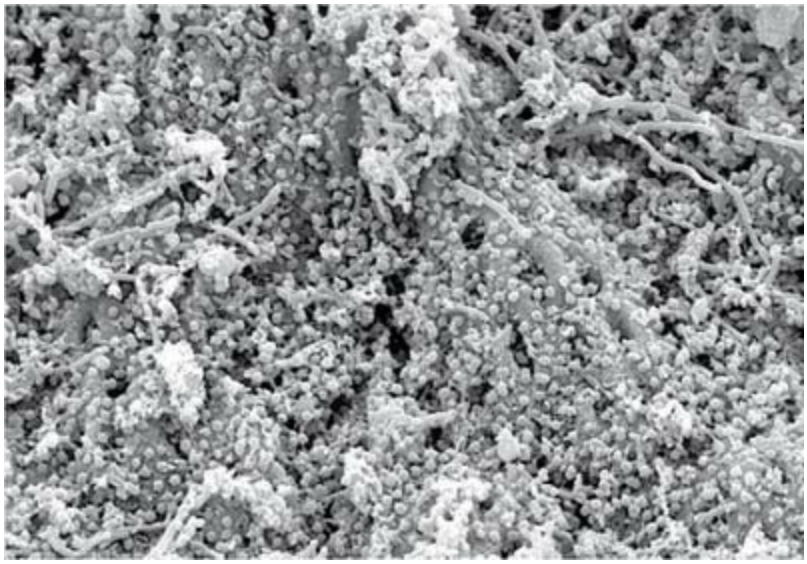

FIG. 15.7 Mixed bacterial population colonizing the root canal wall. Cocci are the predominant forms, but rods, filaments, and spirochetes are also observed. In some areas, coccoid cells are relatively apart from each other (magnification ×2200).

Electron micrograph of microbial population consists of clusters of spherical cells surrounded by rod-shaped and spiral cells.

Source: (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

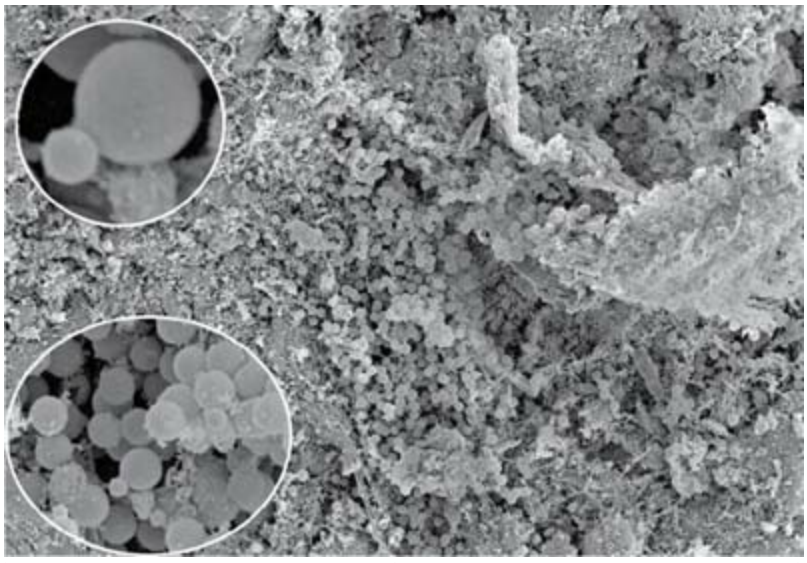

FIG. 15.8 Heavy colonization of yeast cells in the root canal of an extracted tooth with primary infection associated with apical

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periodontitis (magnification ×300). Note that some cells are in the stage of budding. A daughter cell is growing on the surface of the mother cell (insets: magnification ×2700 [*bottom*], magnification ×3500[*top*]).

Electron micrograph of microbial population taken from an extracted tooth shows an inset on top-left highlighting a spherical cell adjoint to a small bud on the top. Another inset on the bottom-left shows a cluster of spherical cells with few of them showing budding of cells depicted as dots on the surface.

Source: (From Siqueira JF Jr, Sen BH: Fungi in endodontic infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:632, 2004.)

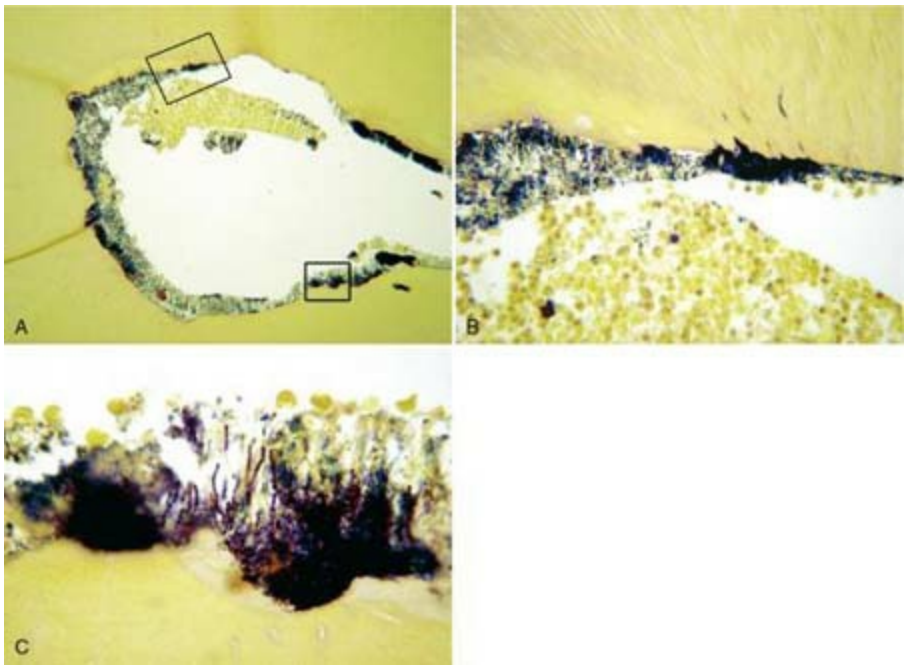

FIG. 15.9 **A,** Biofilm on the walls of a mesial root canal from a mandibular first molar (magnification ×100). The tooth was symptomatic, and an apical periodontitis lesion was present. Sections in **B** and **C** correspond to higher magnifications of the larger and smaller insets, respectively. Note the accumulation of polymorphonuclear neutrophils in the canal near the biofilm. (**B,** magnification ×400; **C,** magnification ×1000). Sections stained with a Taylor-modified Brown and Brenn technique.

Set of three stained micrographs are marked A through C as follows:

A) Mesial root canal shows two rectangular boxes marked on the border near mandibular first molar.

B) Magnified view shows tiny blue dots surrounding the biofilm.

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C) Higher magnified view of B shows two dark blue patches in the walls of mesial root.

Source: (Courtesy Dr. Domenico Ricucci.)

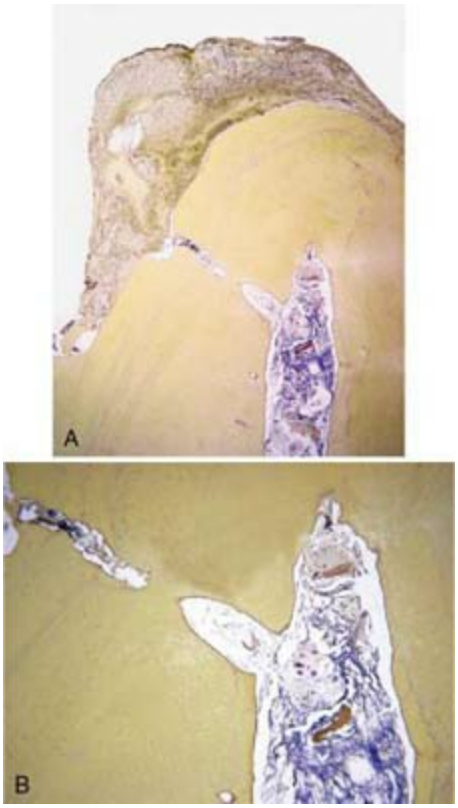

FIG. 15.10 **A,** Bacterial biofilm in the necrotic canal and in an apical ramification contiguous to inflamed periradicular tissues (magnification ×25). **B,** Higher magnification of **A** (magnification ×100). Sections stained with a Taylor-modified Brown and Brenn technique.

Set of two stained micrographs are marked A and B as follows:

A) Micrograph of tooth shows a brown layer of film formed over the dentinal wall, with canal at the center stained blue.

B) Magnified view of necrotic canal in part A shows a budding patch from dentinal wall approaching canal at the center.

Source: (Courtesy Dr. Domenico Ricucci.)

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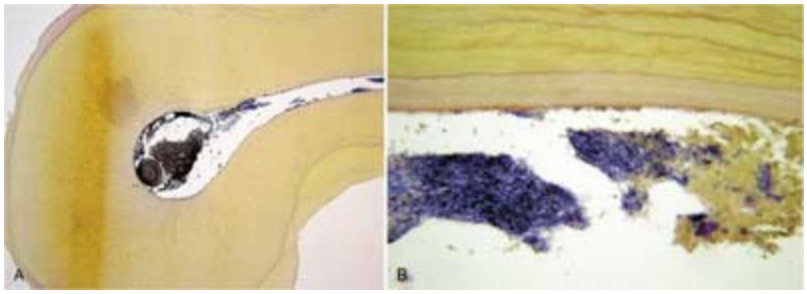

FIG. 15.11 Transverse sections of a maxillary second molar with fused mesial and palatal roots. **A,** Heavy bacterial infection of the canal, spreading to an isthmus (magnification ×25). **B,** Higher magnification of the isthmus clogged with bacteria (magnification ×400). Sections stained with a Taylor-modified Brown and Brenn technique.

Set of two stained micrographs are marked A and B as follows:

A) Micrograph of tooth shows a thick brown layer of film with a blue stain present in isthmus at the center.

B) Magnified view of micrograph A shows isthmus with patches of blue and brown stains depicting blockage caused by bacterial growth.

Source: (Courtesy Dr. Domenico Ricucci.)

Bacterial cells at the bottom of the endodontic biofilm structure are often seen penetrating the dentinal tubules (Fig. 15.12). Dentinal tubule infection occurs in 70% to 80% of the teeth evincing apical periodontitis lesions.[116,148] A shallow penetration is more common, but in some teeth bacterial cells are observed reaching approximately 300 μm deep (Fig. 15.13).[245] Dividing bacterial cells are frequently seen within tubules (see Fig. 15.13),[245] indicating that they are alive and probably derive nutrients from degrading odontoblastic processes, denatured collagen, bacterial cells that die during the course of infection, and intracanal fluids that enter the tubules by capillarity.

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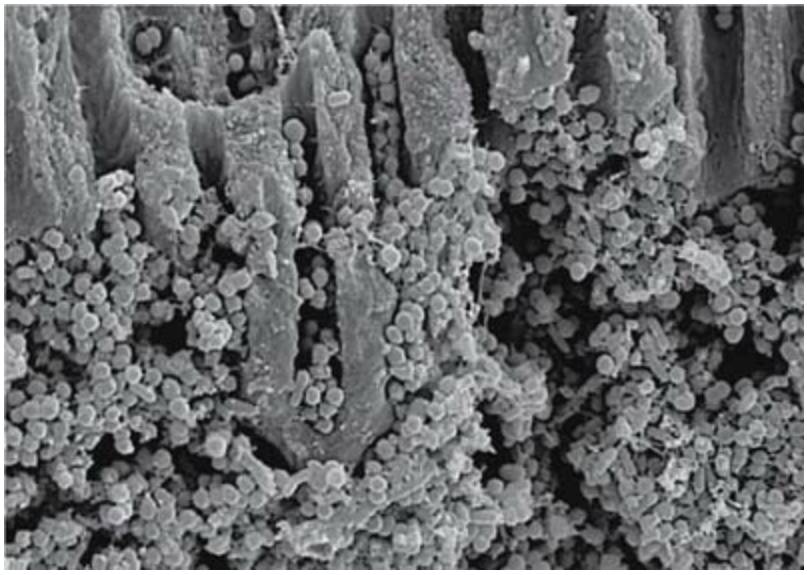

FIG. 15.12 Heavy infection of the root canal walls, mainly by cocci, but some small rods are also seen. Cocci are penetrating into dentinal tubules (magnification ×3500).

Electron micrograph shows clusters of spherical shaped bacteria surrounding the dentinal tubule.

Source: (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

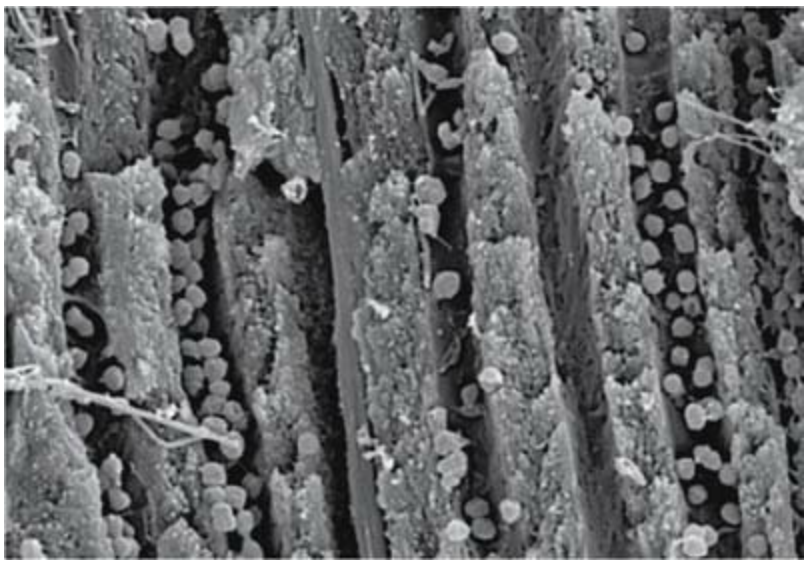

FIG. 15.13 Cocci in dentinal tubules approximately 300 μm from the main root canal (magnification ×5000).

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Magnified electron micrograph of bacterial population in tooth shows multiple chains of spherical shaped cells present on either side of the vertical tubules.

Source: (From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:174, 2002.)

Several putative endodontic pathogens have been shown to be capable of penetrating dentinal tubules in vitro, including *Porphyromonas endodontalis, Porphyromonas gingivalis, Fusobacterium nucleatum, Actinomyces israelii, Propionibacterium acnes, Enterococcus faecalis, Candida albicans*, and streptococci.[112,146,223,246,299] A clinical study isolated and identified bacteria present in root dentin at different depths, and the most common isolates belonged to the genera *Prevotella, Porphyromonas, Fusobacterium, Veillonella, Peptostreptococcus, Eubacterium, Actinomyces,* lactobacilli, and streptococci.[148] Using immunohistologic analysis, Matsuo et al.[116] observed the occurrence of *F. nucleatum, Pseudoramibacter alactolyticus, Eubacterium nodatum, Lactobacillus casei*, and *Parvimonas micra* inside dentinal tubules from the canal walls of extracted teeth with apical periodontitis.

Whereas bacteria present as planktonic cells in the main root canal may be easily accessed and eliminated by instruments and antimicrobial substances used during endodontic treatment, those organized in biofilms attached to the canal walls or located into isthmuses, recesses, lateral canals, and dentinal tubules are definitely more difficult to reach,[290] and may require special therapeutic strategies to be eradicated.

Biofilm and community-based microbial pathogenesis

Individual microorganisms proliferating in a habitat give rise to *populations*. Such populations often occur as microcolonies in the environment. Populations interact with one another to form a *community*. The community and habitat are part of a larger system called an *ecosystem*, which is defined as a functional self-supporting system that includes the microbial community and its environment.

It has been recognized that the dental plaque biofilms associated with

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caries and periodontal diseases represent sophisticated communities that exert functions essential for the biofilm architecture and physiology, with consequent pathogenetic implications. Evidence indicates that apical periodontitis can also develop as a result of collaborative activities of a biofilm community established in the root canal system.

For many endogenous infections in the human body, the community, instead of a single species, has been regarded as the unit of pathogenicity. This community-as-pathogen concept is based on a holistic approach to understand the group behavior of bacteria in mixed infections, and assumes that the whole is more than the simple sum of its parts. Factors such as the composition (types of species), abundance (proportion of each species), interactions (synergism), and virulence factors will act in concert to define the collective pathogenicity of a certain community and determine the disease outcome.

Community profiling studies revealed that bacterial composition of the endodontic microbiota differs consistently between individuals suffering from the same disease (Fig. 15.14).[30,113,184,251] This indicates that apical periodontitis has a heterogeneous etiology, where multiple bacterial combinations can play a role in disease causation. Interindividual variability is even more pronounced when different geographic locations are studied.[60,113,184,244] Moreover, community structure differs significantly between different disease forms (e.g., asymptomatic apical periodontitis vs. acute apical abscess),[196,200,251] suggesting existence of a pattern associated with each form.

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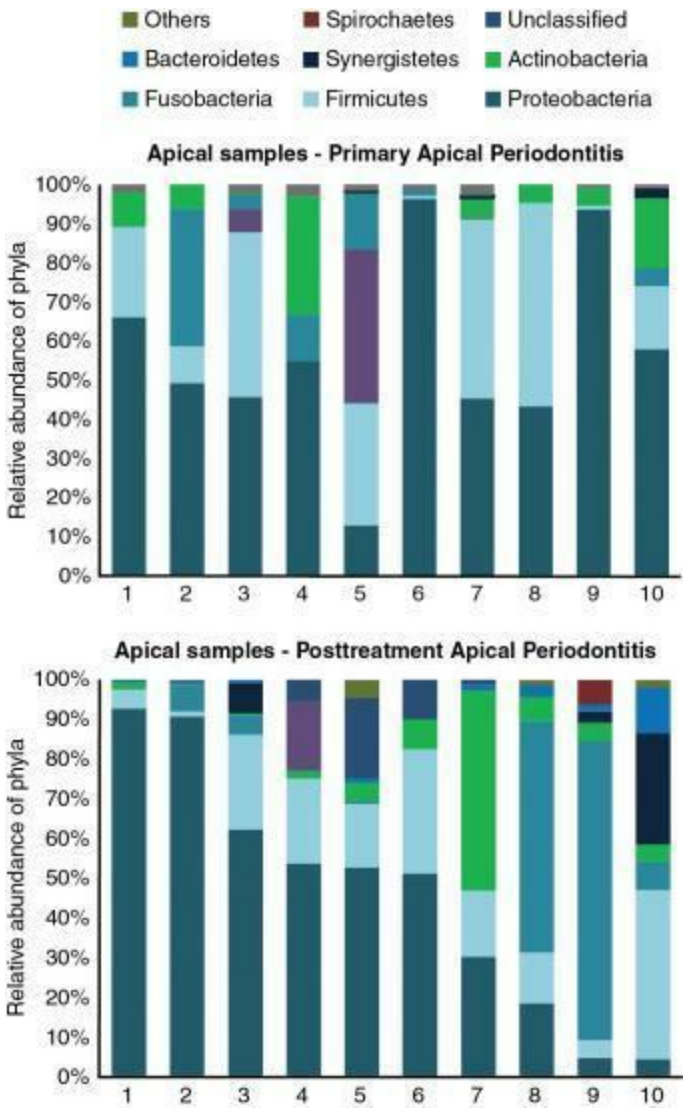

FIG. 15.14 High interindividual variability in bacterial diversity (richness and abundance) at the phylum level in samples taken from cryopulverized apices of teeth with primary and posttreatment apical periodontitis. Variability is much more pronounced at the genus and species (data not shown).

Set of two stacked bar charts compares apical samples pre- and post-treatment of apical periodontitis depicting relative abundance of phyla (in percentage).

The data for primary apical periodontitis is given as follows:

The richness of different bacterial species from highest to lowest is marked as follows: proteobacteria; firmicutes; actinobacteria, fusobacteria; and synergistetes.

The data for post-treatment apical periodontitis shows decreases in percentage of proteobacteria from 90 percent yo 5 percent from 1 to 10 on the horizontal

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axis. Highest percentage of fusobacteria is shown at 8 and 9 while that of actinobacteria is shown at 7. The mixed population of bacteria increases from three species at 1 to six species at 10. Note: All data is approximate.

Source: (Based on data from Siqueira JF Jr, Alves FR, Rôças IN. Pyrosequencing analysis of the apical root canal microbiota. *J Endod* 37:1499, 2011, and Siqueira JF Jr, Antunes HS, Rôças IN, Rachid CT, Alves FR. Microbiome in the apical root canal system of teeth with post-treatment apical periodontitis. *PLoS One* 11:e0162887, 2016).

Biofilm and bacterial interactions

The community-forming ability can be regarded as essential for microbial survival in virtually all environments. Indeed, the majority of microorganisms in nature invariably grow and function as members of metabolically integrated communities, or biofilms.[33,115] *Biofilm* can be defined as a sessile multicellular microbial community characterized by cells that are firmly attached to a surface and enmeshed in a self-produced matrix of extracellular polymeric substance (EPS), mostly composed of polysaccharide but also proteins and nucleic acids (Fig. 15.15).[33,45,75] The ability to form biofilms has been regarded as a virulence factor,[75] and biofilm infections account for an estimated 65% to 80% of bacterial infections that affect humans in the developed world.[32] Given its importance in varied aspects, biofilm properties have been extensively studied not only in medical microbiology but also in different sectors of industrial and environmental microbiology.

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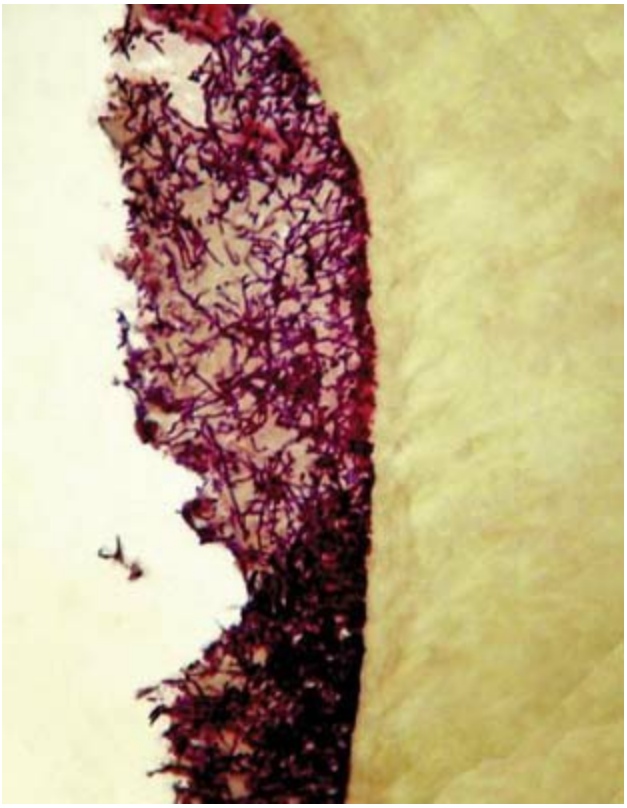

FIG. 15.15 Mixed bacterial biofilm adhered to the tooth surface (Brown and Brenn staining, magnification ×1000).

A micrograph shows a purple blue layer of stain present to the left of wall of tooth.

As a community, biofilms are composed of populations or microcolonies (±15% by volume) that are embedded and nonrandomly distributed in the EPS matrix (±85% by volume) and separated by water channels.[34,45,263,267] Dental biofilms can reach up to 300 or more cell layers in thickness.[263] Individual microcolonies may consist of a single bacterial species, but more frequently are composed of several different species in a mixed community.

The EPS matrix is not only important physically as part of the scaffold that determines the biofilm structure, but it is also biologically active and can retain nutrients, water, and essential enzymes within the biofilm.[5] The matrix also protects the biofilm community from exogenous threats and may participate in adherence to the surfaces.

In most biofilms in nature, bacterial populations arise from surface colonization by planktonic (unattached) bacterial cells. Floating bacteria approach to the surface to be colonized and attach to host proteins and/or

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coaggregate with previously bound bacteria. This process may occur in canals that are frankly exposed to the oral cavity and filled with saliva. However, biofilm formation in the root canal system in most circumstances, especially as a sequel to caries, is expected to occur in a different way. As the caries biofilm reaches the pulp, the latter becomes inflamed and necrotic, and the biofilm advances in apical direction. Therefore, biofilm formation on the root canal walls in most cases occurs gradually as the pulp becomes necrotic and the infectious process advances towards the apical canal.[256]

Bacterial interactions in biofilms lead to changes in growth rate, gene expression, and protein production. Genes expressed by cells in biofilms can differ by 20% to 70% from those expressed by the same cells growing in planktonic culture.[17,135,203] Thus, bacteria in biofilms adopt a radically different phenotype compared with their planktonic counterparts. Within biofilms, some bacteria also use sophisticated systems of cell-cell communication (quorum sensing) to coordinate gene expression. Phenotypic heterogeneity in biofilms is also observed as a result of exposure of microcolonies to a variety of gradients (e.g., oxygen tension, pH, type and amount of nutrients, cell density), which contribute to form diverse microenvironments throughout the biofilm structure.

Biofilm resistance to antimicrobial agents

From a clinical standpoint, the biofilm increased resistance to antimicrobial agents is of special concern. Bacterial cells arranged in biofilms are considered more resistant to antibiotics than the same cells grown in planktonic state. The antibiotic concentration required to kill bacteria in the biofilm is about 100 to 1000 times higher than that needed to kill the same species in planktonic state.[114]

Several possible mechanisms help explain the biofilm resistance to antimicrobials: (a) the biofilm structure may restrict penetration of antimicrobial agents[266]; (b) starved bacteria entering the stationary phase in biofilms are much less susceptible to antimicrobials than rapidly-growing cells[75]; and (c) presence of a subpopulation of specialized survivor cells known as persisters.[94]

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Apical periodontitis as a biofilm-related disease

Numerous morphologic studies have reported the occurrence of bacterial agglomerations on the root canal walls resembling structures that later were defined as biofilm.[23,124,163,166,167,204,245] However, the strongest evidence about the association of bacterial biofilms with apical periodontitis came from a histobacteriologic study by Ricucci and Siqueira.[164] These authors evaluated the prevalence of biofilms in the apical root canal system of untreated teeth with primary apical periodontitis and treated teeth with posttreatment disease and looked for associations between biofilms and clinical/histopathologic conditions. Some of the most important findings of their study are as follows:

1. Intraradicular biofilms were generally observed in the apical segment of approximately 80% of the root canals of teeth with primary or posttreatment apical periodontitis.
2. Morphology of endodontic biofilms differed consistently from individual to individual (e.g., thickness, morphotypes, bacterial cells/extracellular matrix ratio).
3. Dentinal tubules underneath biofilms were often invaded by bacterial cells from the bottom of the biofilm community.
4. Biofilms were also commonly seen covering the walls of apical ramifications, lateral canals, and isthmuses.
5. Bacterial biofilms were more frequent in root canals of teeth with large apical periodontitis lesions. Because it takes time for apical periodontitis to develop and become radiographically visible, one can surmise that large lesions represent a longstanding pathologic process caused by an even “older” intraradicular infection. In longstanding infectious processes, involved bacteria may have had enough time and conditions to adapt themselves to the environment and set a mature and organized biofilm community. The fact that the apical root canal of teeth with large lesions harbors a large number of bacterial cells and species almost always organized in biofilms may help explain the concept that treatment outcome is influenced by

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lesion size.[31,131]

6. The prevalences of intraradicular biofilms in teeth associated with apical cysts, abscesses, and granulomas were 95%, 83%, and 69.5%, respectively. Biofilms were significantly associated with epithelialized lesions. Because apical cysts develop as a result of epithelial proliferation in some granulomas,[107] it may be anticipated that the older the apical periodontitis lesion, the greater the probability of it becoming a cyst. Similar to teeth with large lesions, the age of the pathologic process may also help explain the high prevalence of biofilms in association with cysts.
7. Extraradicular biofilms were infrequent; they occurred in only 6% of the cases. Except for one case, they were always associated with intraradicular biofilms. All cases showing an extraradicular biofilm exhibited clinical symptoms.
8. Bacteria were also seen in the lumen of the main canal, ramifications, and isthmuses as flocs and planktonic cells, either intermixed with necrotic pulp tissue or possibly suspended in a fluid phase. Bacterial flocs are sometimes regarded as “planktonic biofilms” and may originate from growth of cell aggregates/coaggregates in a fluid or they may have detached from biofilms.[76]

Ricucci and Siqueira[164] also used the following criteria available in the literature to establish a causal link between biofilms and a given infectious disease[76,139,164]:

1. Infecting bacteria are adhered to or associated with a surface.
2. Direct examination of the infected tissue shows bacteria forming clusters or microcolonies encased in an extracellular matrix.
3. The infection is generally confined to a particular site and although dissemination may occur, it is a secondary event.
4. The infection is difficult or impossible to eradicate with antibiotics in spite of the responsible microorganisms being susceptible to killing in the planktonic cell state.
5. Ineffective host clearance is evident, as suggested by the location of bacterial colonies in areas of the host tissue associated with host

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inflammatory cells. Accumulation of polymorphonuclear neutrophils and macrophages surrounding bacterial aggregates/coaggregates in situ considerably increases the suspicion of biofilm involvement with disease causation.

6. Elimination or significant disruption of the biofilm structure and ecology leads to remission of the disease process.

Based on findings from the Ricucci and Siqueira's study,[164] apical periodontitis can be considered to fulfill four of the six criteria. Bacterial agreggates/coaggregates are observed adhered to or at least associated with the dentinal root canal walls (criterion 1). Bacterial colonies are often seen encased in an amorphous extracellular matrix (criterion 2). Endodontic biofilms are frequently confined to the root canal system, in only a few cases extending to the external root surface, but dissemination through the lesion never occurred (criterion 3). In the great majority of cases, biofilms are directly faced by an accumulation of inflammatory cells, especially polymorphonuclear neutrophils (criterion 5).

As for criterion 4, it is widely known that intraradicular endodontic infections cannot be effectively treated by systemic antibiotic therapy, even though most endodontic bacteria in the planktonic cell state are susceptible to currently used antibiotics.[15,65,96] The lack of efficacy of systemic antibiotics against intraradicular infections is mainly because the drug does not reach endodontic bacteria that are located in the avascular pulp necrotic space. The recognition of biofilms as the main mode of bacterial establishment in the root canal system further strengthens the explanations for the lack of antibiotic effectiveness against endodontic infections. Finally, there is a clear potential for fulfillment of criterion 6, because biofilms are frequently observed in canals of treated teeth with posttreatment apical periodontitis,[167] whereas teeth with a successful outcome show no biofilm infection of the root canal.[158] In addition, canals that exhibit negative cultures at the time of filling have a better treatment outcome.[52,296]

Methods for microbial identification

The endodontic microbiota has been traditionally investigated by culture

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methods. Culture is the process of propagating microorganisms in the laboratory by providing them with the required nutrients and proper physicochemical conditions, including temperature, moisture, atmosphere, salt concentration, and pH.[260] Essentially, culture analyses involve the following steps: sample collection and transport, dispersion, dilution, cultivation, isolation, and identification.[274] Endodontic samples are collected and transported to the laboratory in a viability-preserving, nonsupportive, anaerobic medium. They are then dispersed by sonication or vortex mixing, diluted, distributed onto various types of agar media, and cultivated under aerobic or anaerobic conditions. After a suitable period of incubation, individual colonies are subcultivated and identified on the basis of multiple phenotype-based aspects, including colony and cellular morphology, gram-staining pattern, oxygen tolerance, comprehensive biochemical characterization, and metabolic end-product analysis by gas-liquid chromatography. The outer cellular membrane protein profile is examined by gel electrophoresis, fluorescence under ultraviolet light, and susceptibility tests to selected antibiotics may be needed for identification of some species.[50] Marketed packaged kits that test for preformed enzymes have also been used for rapid identification of several species. Isolated colonies can also be identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS)[303] or 16S rRNA sequencing.[250]

Culture analyses of endodontic infections have provided a substantial body of information about the etiology of apical periodontitis, composition of the endodontic microbiota in different clinical conditions, effects of treatment procedures in bacterial elimination, and susceptibilities of endodontic bacteria to antibiotics. Advantages and limitations of culture methods are listed in Box 15.1; some important limitations of culture methods make a comprehensive analysis of the endodontic microbiota difficult to achieve.

Box 15.1

Advantages and Limitations of Culture Methods

Advantages	Limitations
Broad-range nature, identification of unexpected species	Impossibility of culturing a large number of extant bacterial species

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Allow quantification of all major viable cultivable microorganisms in samples Allow determination of antimicrobial susceptibilities of isolates Physiologic studies are possible Pathogenicity studies are possible Widely available	Not all viable bacteria can be recovered Once isolated, bacteria require identification using a number of techniques Misidentification of strains with ambiguous or aberrant phenotypic behavior Low sensitivity Strict dependence on the mode of sample transport Samples require immediate processing Costly, time consuming, and laborious, as for cultivation of anaerobes Specificity is dependent on experience of microbiologist Extensive expertise and specialized equipment needed to isolate anaerobes Takes several days to weeks to identify most anaerobes

The difficulties in culturing or identifying many microbial species are of special concern. Unfortunately, not all microorganisms can be cultivated under artificial conditions, and this is simply because the nutritional and physiologic needs of most microorganisms are still unknown. Investigations of many environments using culture-independent methods have revealed that the large majority of microbial members of these systems still remain to be cultivated.[6,302] Furthermore, 40% to 80% of bacterial species composing the microbiota associated with diverse human sites, including the oral cavity, represent unknown and still uncultivated bacteria.[1,2,42,47,99,143,151,268]

That a given species has not been cultivated does not imply that this species will remain indefinitely impossible to cultivate. Myriad obligate anaerobic bacteria were uncultivable in the early 1900s, but further developments in anaerobic culturing techniques have to a large extent helped to solve this problem. It must be assumed that no single method or culture medium is suitable for isolating the vast diversity of microorganisms present in most environments.[69] Because we remain relatively unaware of the requirements for many bacteria to grow, identification methods not based on

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cultivability are required.

In some situations, even the successful cultivation of a given microorganism does not necessarily mean that this microorganism can be successfully identified. Culture-dependent identification is based on phenotypic traits observed in reference strains, with predictable biochemical and physical properties under optimal growth conditions. However, many phenotype-related factors can lead to difficulties in identification and even to misidentification.[16,20,240,282] As a consequence, phenotype-based identification does not always allow an unequivocal identification.

To sidestep the limitations of culturing, tools and procedures based on molecular biology have become available and have substantially improved the ability to achieve a more realistic description of the microbial world without the need for cultivation (Fig. 15.16). Molecular technology has also been applied to reliably identify cultivated bacteria, including strains with ambiguous or aberrant phenotypic behavior, rare isolates, poorly described or uncharacterized bacteria, and newly named species.[21,46,149,231,264,280] The 16S rRNA gene (or 16S rDNA) has been the most widely used gene for bacterial identification, while the 18S rRNA gene of fungi and other eukaryotes has also been used extensively to identify these organisms.

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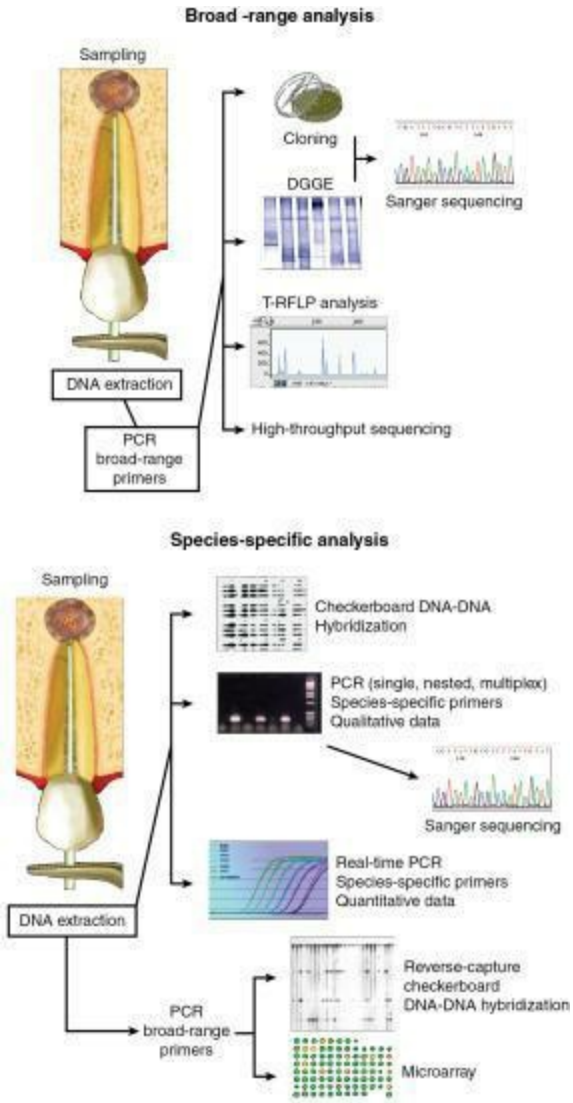

FIG. 15.16 Molecular biology methods used (or with potential to be used) in the study of endodontic infections. The choice for a particular technique will depend on the type of analysis to be performed. Some methods can be used for detection of target microbial species or groups and others for a broader analysis of the microbiota.

Set of two illustrations compares two types of molecular biology methods including broad-range and species-specific analysis. In broad range analysis, DNA is extracted from sample and PCR is conducted using broad-range primers for cloning, DGGE, T-RFLP analysis, and pyrosequencing. Cloning and DGGE is collectively used for sequencing.

In species-specific analysis, DNA is extracted from sample following two purposes:

1. In PCR, broad-range primers are used in generating reverse-capture checkerboard DNA-DNA hybridization and microarray; and

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2. Checkerboard DNA-DNA hybridization; PCR (single, nested, multiplex) utilize species-specific primers, used to deduce qualitative data for sequencing; and real-time PCR using species-specific primers to generate qualitative data.

Data from 16S rRNA gene sequences can be used for accurate and rapid identification of known and unknown bacterial species, using techniques that do not require cultivation. The 16S RNA gene of virtually all bacterial species in a given environment, including still uncultivated and uncharacterized bacteria, can be amplified by polymerase chain reaction (PCR) using broad-range (or universal) primers that are complementary to conserved regions of this gene. Sequencing of the variable regions flanked by the broad-range primers will provide information for accurate bacterial identification. Primers or probes that are complementary to variable regions can also be designed to detect specific target species directly in clinical samples.

Many molecular methods for the study of microorganisms exist; the choice of a particular approach depends on the questions being addressed.[240] Broad-range PCR followed by cloning and Sanger sequencing can be used to unravel the breadth of bacterial diversity in a given environment. High-throughput (or “next-generation”) sequencing (HTS) approaches permit DNA sequencing from samples to a far deeper coverage and higher throughput in comparison with the traditional Sanger sequencing approach[82,294]; even low-abundant components of the bacterial community can be disclosed by HTS approaches. In addition to HTS, bacterial community structures can be analyzed and compared by fingerprinting techniques such as denaturing gradient gel electrophoresis (DGGE) and terminal restriction fragment length polymorphism (T-RFLP). Fluorescence in situ hybridization (FISH) can measure the abundance of target species and provide information on their spatial distribution in tissues. Among other applications, DNA-DNA hybridization arrays (checkerboard techniques, DNA microarrays), species-specific single PCR, nested PCR, multiplex PCR, and quantitative real-time PCR can be used to survey large numbers of clinical samples for the presence of target species. Quantitative real-time PCR can also be applied to the study of the antibacterial effectiveness of treatment procedures by quantifying all bacteria or certain groups before and after treatment. Variations in PCR

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technology can also be used to type microbial strains. As with any other method, molecular methods have their own advantages and limitations (Box 15.2).

Box 15.2

Advantages and Limitations of Molecular Biology Methods

Advantages	Limitations
Detect both cultivable and as-yet-uncultivated species or strains	Most assays are qualitative or semiquantitative (exception: real-time PCR)
High specificity and accurate identification of strains with ambiguous or aberrant phenotypic behavior	Most assays only detect one species or a few different species at a time (exceptions: broad-range PCR, checkerboard, microarray)
Detect species directly in clinical samples	Most assays detect only the target species and fail to detect unexpected species (exception: broad-range PCR)
High sensitivity	Some assays can be laborious and costly (e.g., broad-range PCR)
Rapid; most assays take no more than minutes to a few hours to identify a microbial species	Biases in broad-range PCR introduced by homogenization procedures, preferential DNA amplification, and differential DNA extraction
Do not require carefully controlled anaerobic conditions during sampling and transportation	Hybridization assays using whole genome probes detect only cultivable species
Can be used during antimicrobial treatment	Can be very expensive
Anaerobic handling and expertise not required	
Samples can be stored frozen for later analysis	
DNA can be transported easily between laboratories	
Detect dead microorganisms	

PCR, Polymerase chain reaction.

The five generations of endodontic microbiology studies

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Microbiologic studies for identification of the species participating in endodontic infections can be chronologically divided into five generations on the basis of the different strategic approaches used.[255] These generations are detailed in Table 15.1.

Table 15.1

Generations of Studies for Microbiologic Identification in Endodontic Infections

Study Generation	Identification Method	Nature	Description and Findings
First	Culture	Open ended (broad range)	Revealed many cultivable species in association with apical periodontitis
Second	Molecular methods (e.g., PCR and its derivatives, original checkerboard assay)	Closed ended (species specific)	Target cultivable bacteria Confirmed and strengthened data from first generation Allowed inclusion of some culture-difficult species in the set of candidate endodontic pathogens
Third	Molecular methods (e.g., PCR-cloning-sequencing, T-RFLP)	Open ended (broad range)	Allowed a more comprehensive investigation of the bacterial diversity in endodontic infections Not only cultivable species but also as-yet-uncultivated and uncharacterized bacteria were identified
Fourth	Molecular methods (e.g., PCR, microarrays, reverse-capture checkerboard)	Closed ended (species specific)	Target cultivable and as-yet-uncultivated bacteria Large-scale clinical studies to investigate prevalence and association of species/phylotypes with endodontic infections
Fifth	Molecular methods (high-throughput sequencing platforms, such as Roche 454 GS-FLX pyrosequencing, Illumina MiSeq and HiSeq)	Open ended (broad range)	Permit a deep-coverage and more comprehensive analysis of the diversity of endodontic infections

PCR, Polymerase chain reaction.

Impact of molecular methods in endodontic microbiology

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Culture studies (first generation) identified a set of species thought to play an important role in the pathogenesis of apical periodontitis.[11,209,276] Further, not only have findings from culture-based methods been confirmed, but they have also been significantly supplemented with those from culture-independent molecular biology techniques, which constitute the other four generations of endodontic microbiology studies.[236] Molecular methods have confirmed and strengthened the association of many cultivable bacterial species with apical periodontitis and have also revealed new suspected endodontic pathogens.[232] The list of candidate pathogens has expanded to include culture-difficult species or as-yet-uncultivated bacteria that had never been found in endodontic infections by culture. The results from molecular studies impact remarkably on the knowledge of bacterial diversity in endodontic infections. More than 400 different bacterial species have been detected in different types of endodontic infections.[236] Of these, about 45% were exclusively reported by molecular biology studies, compared with 32% detected by culture studies alone.[236] Twenty-three percent of the total bacterial species richness have been detected by application of both culture and molecular studies (Fig. 15.17). As a consequence, it becomes quite evident that the knowledge about the composition of the endodontic microbiota has been refined and redefined by molecular methods.[232]

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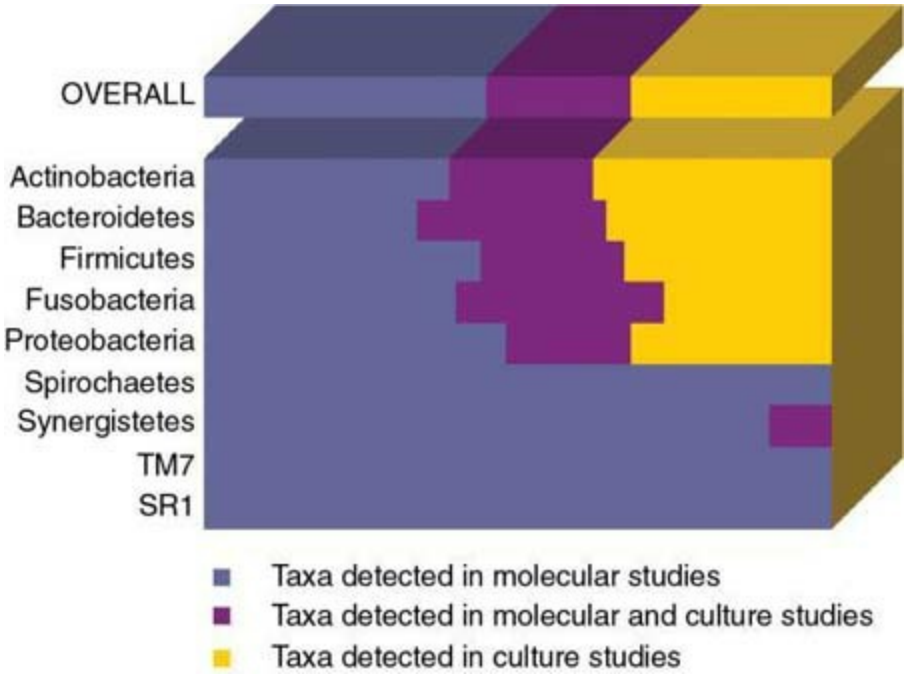

FIG. 15.17 Percentage distribution of bacterial species/phylotypes found in endodontic infections according to the detection method. Data refer to the percentage of species or phylotypes overall or in each of the nine phyla that have endodontic representatives.

Percentage distribution of bacterial species are given as follows in increasing percentage:

Overall, actinobacteria, bacteriodetes, firmicutes, fusobacteria, and proteobacteria: Taxa detected in molecular studies; taxa detected in culture studies; and taxa detected in molecular and cultural studies.

Taxa detected only in molecular studies: Spirochaetes, TM7, and SR 1.

Synergistetes are majorly detected by molecular studies with a minor portion detected by molecular and culture studies.

Note: All data is approximate.

Types of endodontic infections

Endodontic infections can be classified according to the anatomic location as intraradicular or extraradicular infection. *Intraradicular infection* is caused by microorganisms colonizing the root canal system and can be subdivided into three categories according to the time microorganisms entered the root canal system: *primary infection*, caused by microorganisms that initially

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invade and colonize the necrotic pulp tissue (initial or "virgin" infection); *secondary infection,* caused by microorganisms not present in the primary infection but introduced in the root canal at some time after professional intervention (i.e., *secondary* to intervention); and *persistent infection,* caused by microorganisms that were members of a primary or secondary infection and in some way resisted intracanal antimicrobial procedures and were able to endure periods of nutrient deprivation in treated canals. *Extraradicular infection* in turn is characterized by microbial invasion of the inflamed periradicular tissues and is a sequel to the intraradicular infection. Extraradicular infections may be dependent on or independent of the intraradicular infection.

Diversity of the endodontic microbiota

The terms *microbiota* and *microbiome* have been used interchangeably in many texts, while others have used some confounding definitions. Here, we refer to *microbiome* as a collection of genomes from all the microorganisms in a given site, and *microbiota* as all the microorganisms in a site. Microbiota should replace terms such as *flora* and *microflora,* which perpetuate an outdated classification of microorganisms as plants.[41] *Diversity* refers to the number of different species present (richness) and their relative abundance (evenness) in a given ecosystem.[87]

The oral cavity harbors one of the highest accumulations of microorganisms in the human body. Even though viruses, archaea, fungi, and protozoa can be found as constituents of the oral microbiota, bacteria are by far the most dominant inhabitants of the oral cavity. Culture-independent studies (microscopy and molecular biology technologies) have shown that over 40% to 60% of the oral microbiota still remains to be cultivated and fully characterized.[2,42,262] A high diversity of bacterial species has been revealed in the oral cavity by culturing approaches,[125] but application of molecular biology methods to the analysis of the bacterial diversity has revealed a still broader and more diverse spectrum of extant oral bacteria.[144] More than 1000 bacterial species/phylotypes have been found in studies of the human oral microbiome,[42] but advanced HTS studies have indicated that this number may still have been largely underestimated.[4,71,93] Anaerobic

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culture and molecular biology techniques have demonstrated that, at a broader taxonomic level, endodontic bacteria fall into nine phyla—namely, Firmicutes, Bacteroidetes, Spirochaetes, Fusobacteria, Actinobacteria, Proteobacteria, Synergistetes, TM7, and SR1 (Fig. 15.18).[126,156,177,195–197,233,236] However, data from studies using HTS technology reveal that several other bacterial phyla may have been overlooked by previous identification techniques, including Tenericutes, Acidobacteria, Deinococcus-Thermus, Chloroflexi, and Cyanobacteria.[63,83,86,95,105,200,213,219,220,224,286,289,309] Members of these less common phyla are usually low-abundant components of the endodontic bacterial communities, only detected because of deep sequencing coverage. Fungi and archaea are types of microorganisms that have been only occasionally found in endodontic infections.

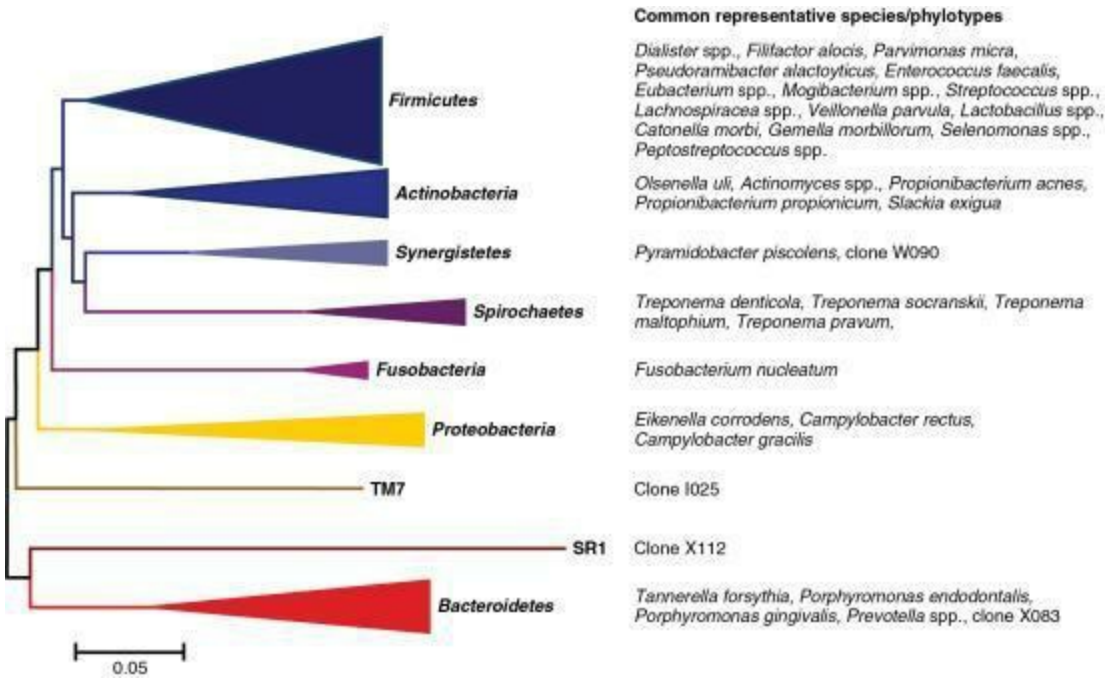

FIG. 15.18 Bacterial phyla with their respective endodontic representatives. *Right:* Example species or phylotypes for each phylum.

Phylogenetic tree shows bacteriodetes and SR1 leading to a node connecting TM 7, which further connects to node linked to proteobacteria. Together theory are linked to fusobacteria, which leads to three sub-classes: Firmicutes linked to actinobacteria and a combination of synergistetes and spirochaetes.

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Endodontic infections develop in a previously sterile place that does not contain a normal microbiota. Any species found has the potential to be an endodontic pathogen or at least play a role in the ecology of the endodontic microbial community. Culture and molecular studies of endodontic infections are cross-sectional in nature and consequently reveal only prevalence of species; thus, only association can be inferred. A role in causation of apical periodontitis is usually surmised on the basis of both frequency of detection and potential pathogenicity (in animal models or association with other human diseases), and several species have emerged as candidate endodontic pathogens. The following sections discuss specific aspects of each type of endodontic infection.

Primary intraradicular infection

Microbial diversity

Primary intraradicular infection develops in the necrotic pulp tissue of untreated teeth and is the cause of primary apical periodontitis (Fig. 15.19). Participating bacteria may be involved in earlier stages of pulp invasion (usually via caries) that culminated in inflammation and further necrosis, or they can be latecomers that took advantage of the environmental conditions in the root canal after pulp necrosis.

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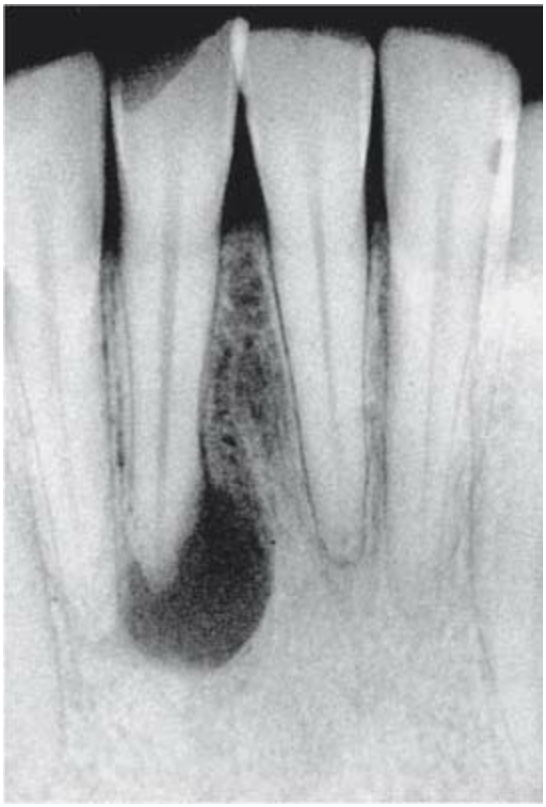

FIG. 15.19 Apical periodontitis due to primary intraradicular infection. The dental pulp is necrotic and the lesion size is usually directly proportional to the complexity of the microbiota involved.

Radiograph shows a gray patch present at the apical foramen in the root.

Primary infections are characterized by a mixed (multispecies) community conspicuously dominated by anaerobic bacteria. The number of bacterial cells may vary from 10^3 to 10^8 per root canal.[19,130,197,249,273,292] Second- to fourth-generation molecular studies have disclosed a mean of 10 to 20 species/phylotypes per infected canal.[126,156,177,232,251] However, these numbers may be even higher based on fifth-generation HTS studies.[83,105,200,219] Canals of teeth with associated sinus tracts may exhibit a number of species close to the top of this range.[177] The size of apical periodontitis lesion has been shown to be proportional to the number of bacterial species (richness) and cells (density or load) in the root canal.[177,250,273] A molecular study[177] demonstrated that the number of taxa per canal was clearly in direct proportion to the lesion size: small lesions (<5 mm) harbored about 12 taxa, lesions from 5 to less than 10 mm harbored 16 taxa, and lesions over 10 mm harbored about 20 species. Some canals

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associated with large lesions may harbor even more than 40 taxa.[177] Therefore, the larger the lesion, the higher the bacterial diversity and density in the canal.

The most prevalent named bacterial species detected in primary infections, including acute abscess cases, belong to diverse genera of gram-negative (i.e., *Fusobacterium*, *Dialister*, *Porphyromonas*, *Prevotella*, *Tannerella*, *Treponema*, *Pyramidobacter*, *Campylobacter*, and *Veillonella*) and gram-positive (*Parvimonas*, *Filifactor*, *Pseudoramibacter*, *Streptococcus*, *Propionibacterium*, *Olsenella*, and *Actinomyces*) bacteria.[14,55,56,66,74,96,126,156,177,179,183,195,196,232,250,252,253,273,275,293] Bacterial prevalence in primary infections may vary from study to study as a function of several factors, such as sensitivity and specificity of the detection and identification methods, sampling technique, geographic location, and accuracy or divergence in clinical diagnosis and disease classification. Even so, the species most frequently detected are expected to be the same in most well-conducted studies. Figs. 15.20–15.22 display the most frequently detected species associated with asymptomatic apical periodontitis, symptomatic apical periodontitis, and acute apical abscesses, as revealed by studies from the authors' group using a highly sensitive molecular biology technique (nested PCR).

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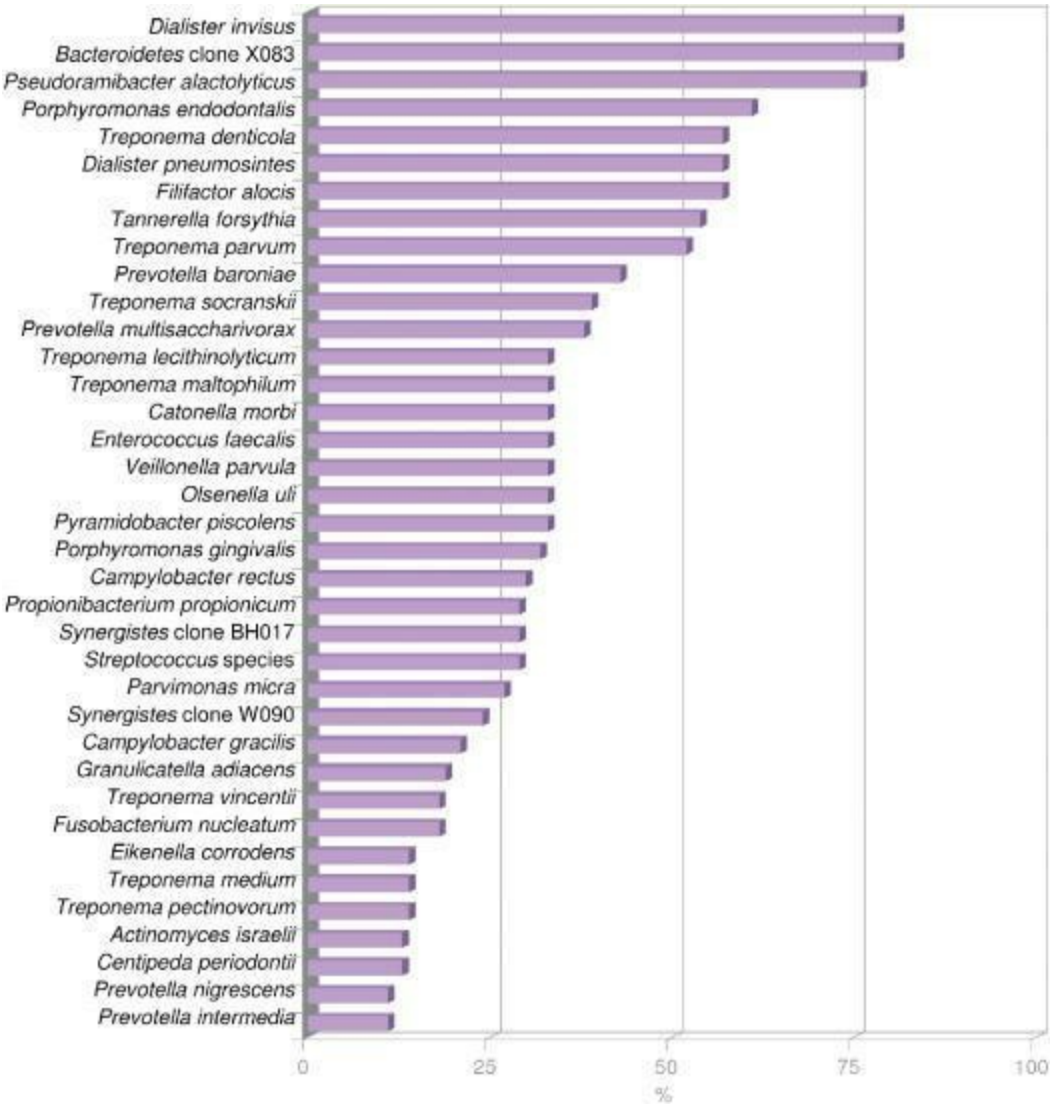

FIG. 15.20 Prevalence of bacteria detected in primary infections of teeth with asymptomatic (chronic) apical periodontitis.

Horizontal bar chart shows percentage distribution of bacterial species marked in decreasing order from top to bottom as follows:

More than 75 percent: Dialister invisus; Bacteriodetes clone X 083.

50 to 75 percent: Pseudoarmibacter alactolyticys; Porphyromonas endodontalis; Treponema denticola; Diaslister pneumosintes; Filifactor alocis; Tannerelia forsythia; and Treponema parvum.

25 to 50 percent: Prevotela baroniae; Treponema socranskii; Prevotella multisaccharivorax; Treponema maltophilum; Catonella morbi;Enterococcus fecalis; Veilonella parvula; Olsenella uli; Pyramidobacter piscolens; Porphyomonas gingivalis; Camphylobacter rectus; Propioibacterium proponicum; Synergistes clone BH 017; Streptococcis species; and

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Parvimonas micra.

0 to 25 percent: Synergistes clone W 090; Campylobacter gracilis; Granulicatella adiacens; Treponema vincentii; Fusobacterium nucleatum; Eikenella corrodens; Treponema medium; Treponema pectinovorum; Actinomyces israelil; Centipeda periodontil; Prevotella nigrescens; and Prevotella inermedia.

Source: Data from the authors' findings using a taxon-specific nested-polymerase chain reaction protocol.

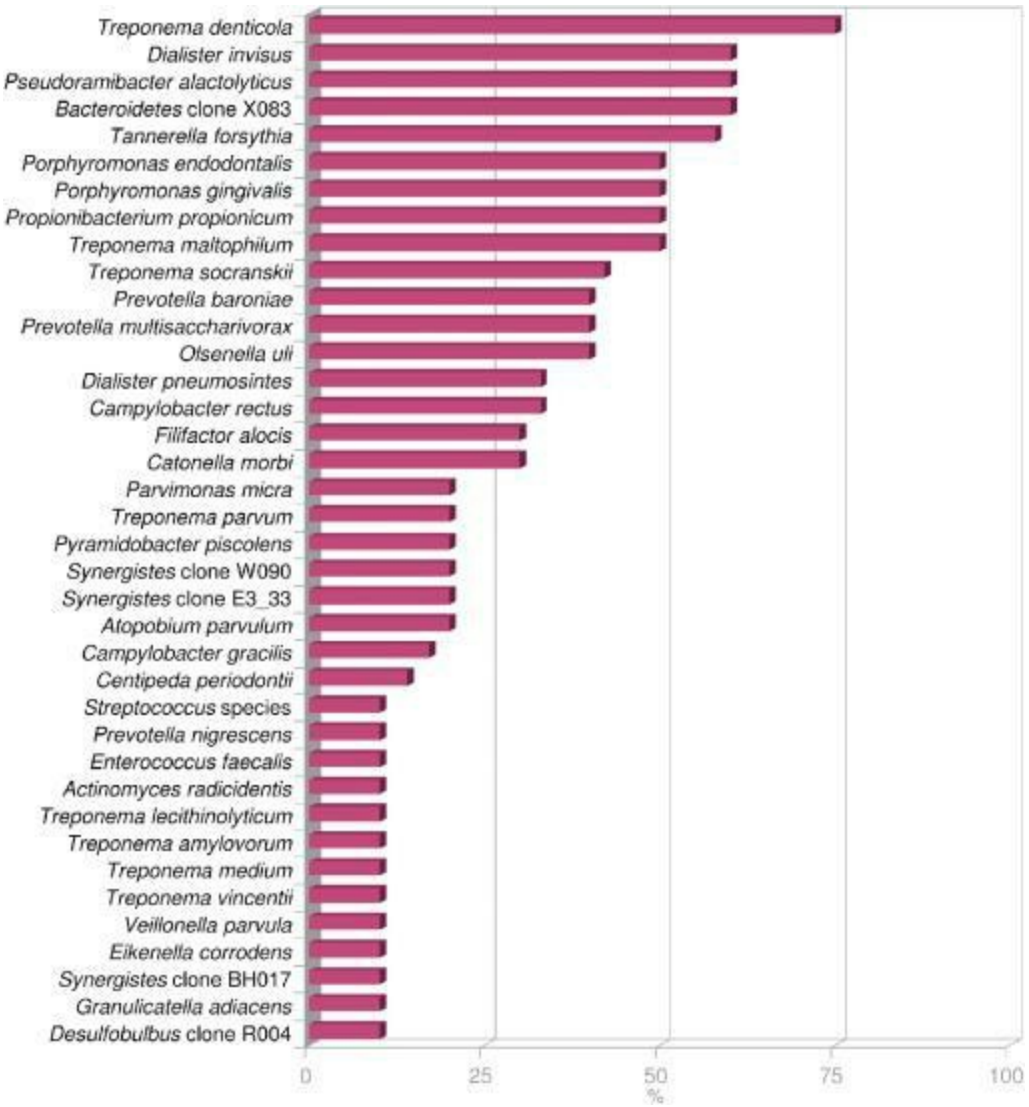

FIG. 15.21 Prevalence of bacteria detected in primary infections of teeth with symptomatic (acute) apical periodontitis.

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Horizontal bar chart shows percentage distribution of bacterial species marked in decreasing order from top to bottom as follows:

50 to 75 percent; Treponema denticola; Dialster invisus; Pseudoramibacter alactolyticus; Bacteriodetes clone X 083; Tannerella forsythia.

25 to 50 percent: Porphyromonas endodontalis; Porphyromonas gingivalis; Propionibacterium propionicum; Treponema maltphilum; Treponema socranskii; Prevotella baroniae; Prevotella multisaccharivorax; Olsenellauli; Dialister pneumosintes; Campylobatcer rectus; Filifactor alocis; Catonella morbi.

0 to 25 percent: Parvimonas micra; Treponema parvum; Pyramidobacter piscolens; Synergistes clone W090; Synergistes clone E3_33; Atopobium parvulum; Campylobacter gracilis; Cenipeda peiodontii; Streptococcus species; Prevotella nigrescens; Enterococcus faecalis; Actinomyces radicidentis; Treponema lecithinolyticum; Treponema amylovorum; Treponema medium; Treponema vincentii; Veilonella parvula; Eikenella corrodens; Synegistes clone BH 107; Granulicatella adiacens; and Desulfobulbus clone R004.

Source: Data from the authors' findings using a taxon-specific nested-polymerase chain reaction protocol.

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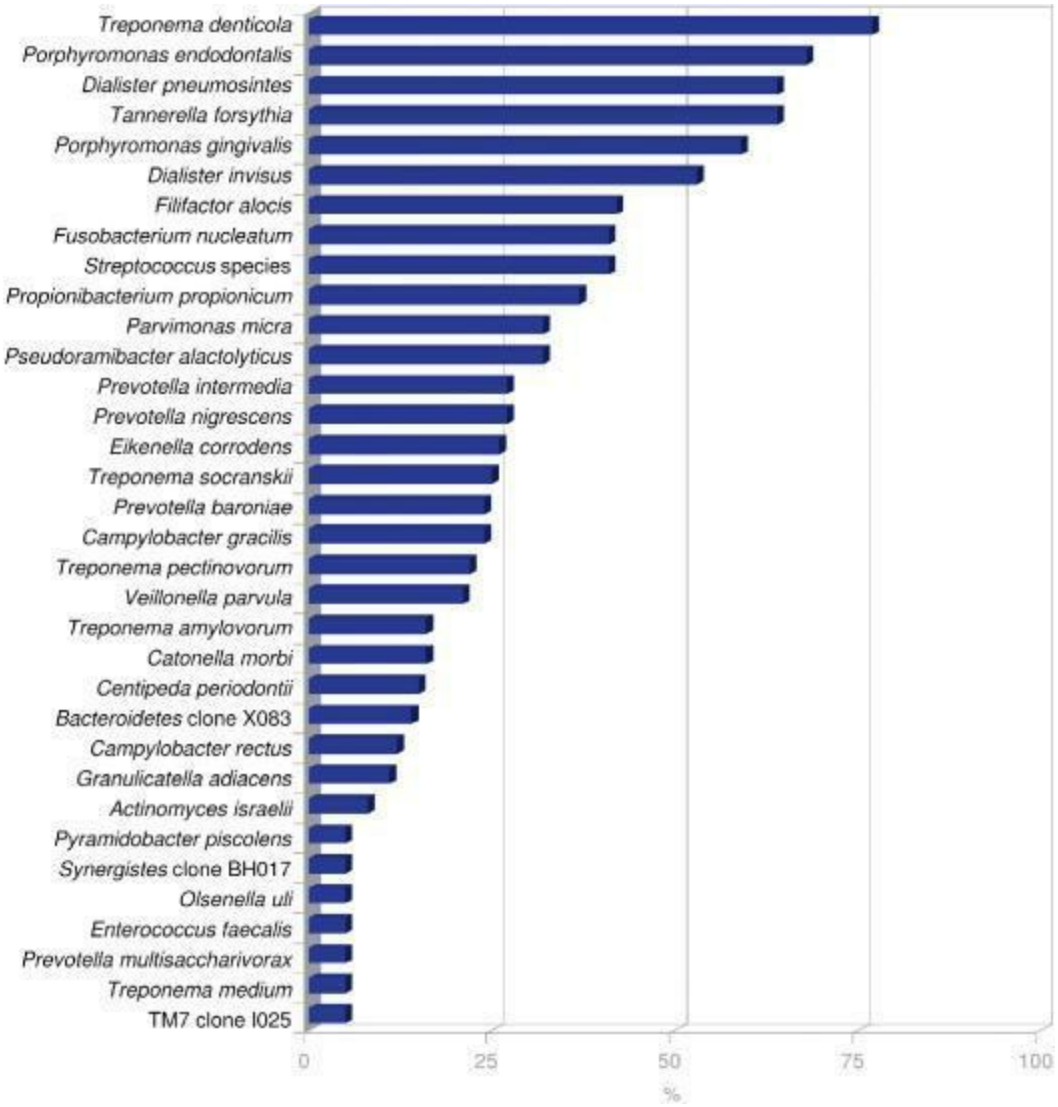

FIG. 15.22 Prevalence of bacteria detected in primary infections of teeth with acute apical abscesses.

Horizontal bar chart shows percentage distribution of bacterial species responsible for acute apical abscesses are marked in decreasing order from top to bottom as follows:

More than 75 percent: Treponema denticola.

50 to 75 percent: Porphyromonas endodontalis; Dialister pneumosintes; Tannerella forsynthia; Porphyromonas gingivalis; and Dialister invisus.

25 to 50 percent: Filifactor alocis; Fusobacterium propionicum; Parvimonas micra; Pseudoramibacer alacolyticus; Prevotella intermedia; and Prevotella nigrescens.

0 to 25 percent: Eikenella corrodens; Treponema socranskii; Prevotella baroniae; Campylobacter gracilis; Treponema pectinovorum; Veilonellla

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parvula; Treponema amylovorum; Caonella morbia; Centipeda periodontii; Bateriodees clone X 083; Campylobacter rectus; Granulicatella adiacens; Actinomyces israelli; Pyramidobacter piscolens; Synergistes clone BH 107; Olsenella uli; Enterococcus faecalis;Prevotella multisacharivorax; Treponema medium; and TM 7 clone I 025.

Source: Data from the authors' findings using a taxon-specific nested-polymerase chain reaction protocol.

Approximately 40% to 66% of the endodontic microbiota in primary infections is composed of species still uncultivated.[126,156,196] As for their abundance in these infections, as-yet-uncultivated phylotypes corresponded to about 40% of the clones sequenced.[196] Molecular studies investigating the breadth of bacterial diversity in infected root canals have disclosed the occurrence of uncultivated phylotypes belonging to several genera, including *Dialister*, *Prevotella*, *Solobacterium*, *Olsenella*, *Fusobacterium*, *Treponema*, *Eubacterium*, *Megasphaera*, *Veillonella*, and *Selenomonas*, as well as phylotypes related to the family *Lachnospiraceae* or the TM7 and Synergistetes phyla.[126,156,175,176,188,195,196,199,233,243] Some uncultivated phylotypes can even be among the most prevalent bacteria in primary intraradicular infections, and others may be associated with pain.[196] *Bacteroidaceae* sp. HOT-272 (synonym, Bacteroidetes oral clone X083) is one of the most prevalent phylotypes found in endodontic infections.[177–179,241] Detection of as-yet-uncultivated phylotypes in samples from endodontic infections suggests that they can be previously unrecognized bacteria that play a role in the pathogenesis of different forms of apical periodontitis. The fact that they have not yet been cultivated and phenotypically characterized does not mean that they are not important.[238]

Symptomatic infections

Symptomatic apical periodontitis and acute apical abscesses are typical examples of endodontic infections causing severe symptoms. In these cases, the infection is located in the canal, but it has also reached the periradicular tissues and, in abscessed cases, can spread to other anatomic spaces. Acute apical abscesses are caused by bacteria that egress from the infected root canal and invade the periradicular tissues to establish an extraradicular infection and evoke purulent inflammation. Clinically, the disease leads to

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pain or swelling and has the potential to diffuse to sinuses and fascial spaces of the head and neck to form a cellulitis or other complications (Fig. 15.23). The microbiota involved in endodontic abscesses is mixed and dominated by anaerobic bacteria (see Fig. 15.22).[37,96,100,169,196,237,239,251] Direct comparisons using molecular technology revealed an average of 12 to 18 taxa per abscess case, compared with 7 to 12 taxa present in root canals of teeth with asymptomatic lesions.[196,251] Uncultivated phylotypes constitute approximately 40% of the taxa found in abscesses (richness) and collectively represent more than 30% of the 16S rRNA gene sequences retrieved in clone libraries (abundance).[196] The total number of bacterial cells in acute apical abscesses has been shown to vary from 10^4 to 10^9.[96,103,305]

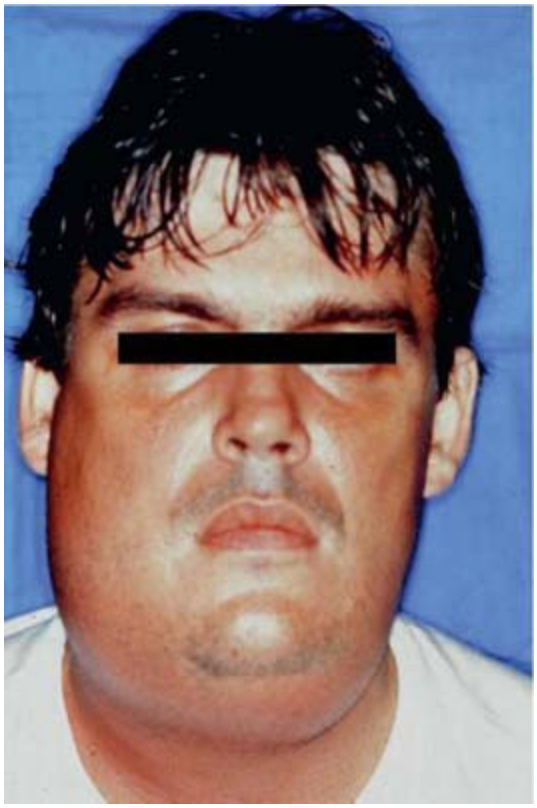

FIG. 15.23 Acute apical abscess. The infection has spread to other anatomic spaces to form cellulitis.

Close-up of face of a patient shows swelling on the left side of the face with submandibular region also shown swollen.

Source: (Courtesy Dr. Henrique Martins.)

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Whereas microbial causation of apical periodontitis is well established, there is no strong evidence disclosing specific involvement of a single species with any particular sign or symptom of apical periodontitis. Some gram-negative anaerobic bacteria have been suggested to be involved with symptomatic lesions,[66,70,185,196,273,288,308] but the same species may also be present in somewhat similar frequencies in asymptomatic cases.[14,56,74,90,183,252,253] Thus, factors other than the mere presence of a given putative pathogenic species may play a role in the etiology of symptomatic endodontic infections.[214,221,239] These factors include: differences in virulence ability among strains of the same species, bacterial interactions resulting in synergistic effects among species in mixed infections, overall number of bacterial cells (overall infectious load), counts and relative abundance of specific pathogenic species (specific infectious load), environmental cues regulating expression of virulence factors, host resistance, and concomitant herpesvirus infection. Association of some or all of these factors is likely to determine the occurrence and intensity of symptoms.[214,221]

Molecular studies using DGGE, T-RFLP, or HTS analysis have revealed that the structure of the endodontic bacterial communities in symptomatic teeth, including abscess cases, is significantly different from that of asymptomatic teeth.[196,200,251] Differences are represented by different dominant species in the communities and larger numbers of species in symptomatic cases. Differences in the type and load of dominant species and the resulting bacterial interactions may affect virulence of the whole bacterial community. The counts of some specific pathogenic species have been shown to be important for the community pathogenicity in abscesses. Molecular studies have shown that the levels of *Porphyromonas endodontalis, Prevotella baroniae, Tannerella forsythia, Treponema denticola,* and *Streptococcus* species were significantly higher in samples from abscesses than in asymptomatic teeth.[183,202] In addition, different species combinations may result in different outcomes because of the network of interactions.[186] The possibility exists that bacterial interactions result in communities that are more or less aggressive and consequently can cause host responses of corresponding intensity. This is consistent with the concept that the entire bacterial community is the unit of pathogenicity.

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Geographic influence

Findings from laboratories in different countries are often different regarding the prevalence of the species involved in endodontic infections. Although these differences may be attributed to technical issues, a geographic influence in the composition of the root canal microbiota has been suspected. Studies using molecular biology techniques directly compared the prevalence of candidate endodontic pathogens in endodontic samples from patients residing in distinct geographic locations and observed significant differences.[13,171,226] Analysis of the bacterial community profiles of the microbiota associated with endodontic infections from different countries also revealed a geography-related pattern, with several species being exclusive for each location and others shared by the locations showing great differences in prevalence.[113,244] The factors that can lead to differences in the composition of the endodontic microbiota and the impact of these differences on therapy, particularly in abscess cases requiring systemic antibiotic therapy, remain elusive.

Microbial ecology and the root canal ecosystem

A root canal with necrotic pulp provides a space for bacterial colonization and affords bacteria a moist, warm, nutritious, and anaerobic environment, which is by and large protected from the host defenses because of lack of an active blood circulation in the necrotic pulp tissue. Also, the root canal walls are nonshedding surfaces conducive to persistent colonization and formation of complex communities. The necrotic root canal might be considered a fertile environment for bacterial growth, with colonization not being a difficult task for most oral bacterial species. Although a large number of bacterial species (about 100 to 200) can be found in the oral cavity of a particular individual,[144] only a limited assortment of these species (about 10 to 20) is consistently selected out for growth and survival within a root canal containing necrotic pulp tissue from the same individual. This indicates that ecologic determinants operate in the necrotic canal and dictate which species will succeed in colonizing this previously sterile environment. The major ecologic factors that determine the composition of the root canal microbiota include oxygen tension, type and amount of available nutrients, and bacterial interactions. Other factors such as temperature, pH, and receptors for

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adhesins may also be involved.

The root canal infection is a dynamic process, and different bacterial species apparently dominate at different stages.[281] Shifts in the composition of the microbiota are largely due to changes in environmental conditions, particularly in regard to oxygen tension and nutrient availability. In the initial phases of the pulpal infectious process, facultative bacteria predominate.[51] After a few days or weeks, oxygen is depleted within the root canal as a result of pulp necrosis and consumption by facultative bacteria. Further oxygen supply is interrupted with loss of blood circulation in the necrotic pulp. An anaerobic milieu develops and is highly conducive to the survival and growth of obligate anaerobic bacteria. With the passage of time, anaerobic conditions become even more pronounced, particularly in the apical third of the root canal; as a consequence, anaerobes will dominate the microbiota, outnumbering facultative bacteria (Fig. 15.24).

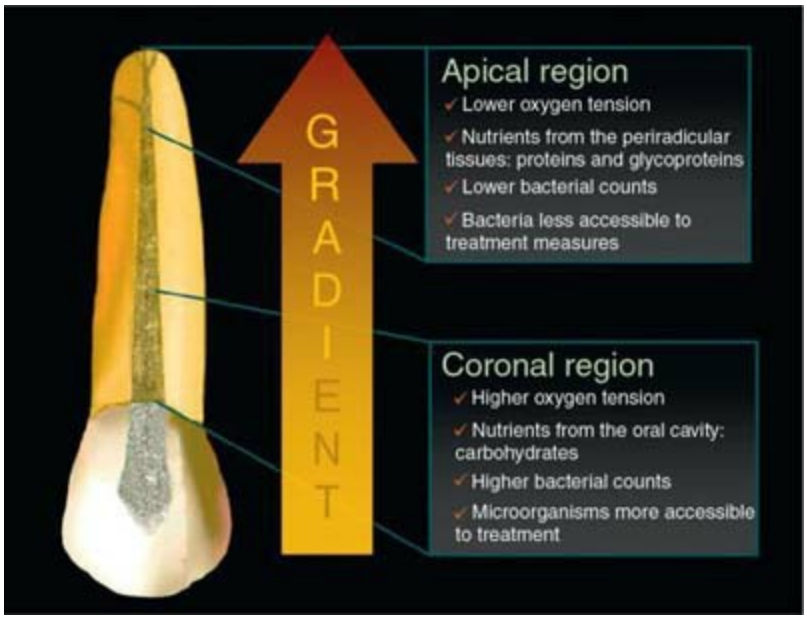

FIG. 15.24 Ecologic conditions in different areas of the root canal. A gradient of oxygen tension and nutrients (type and availability) is formed. Consequently, the microbiota residing in different parts can also differ in diversity, density, and accessibility to treatment procedures.

An upward arrow marked to the right of a tooth shows gradient divided into two regions as follows:

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Apical region:

• Lower oxygen tension.

• Nutrient from the periandicula tissues: proteins and glycoproteins.

• Lower bacterial counts.

• Bacteria less accessible to treatment measures.

Coronal region:

• Higher oxygen tension.

• Nutrients from the oral cavity: carbohydrates.

• Higher bacterial counts.

• Microorganisms more accessible to treatment.

The main sources of nutrients for bacteria colonizing the root canal system include (1) the necrotic pulp tissue, (2) proteins and glycoproteins from tissue fluids and exudate that seep into the root canal system via apical and lateral foramina, (3) components of saliva that may coronally penetrate into the root canal, and (4) products of the metabolism of other bacteria. Because the largest amount of nutrients is available in the main canal, which is the most voluminous part of the root canal system, the highest numbers of bacteria (particularly fastidious anaerobic species) are expected to be located in this region. Bacterial species that can best utilize and compete for nutrients in the root canal system will succeed in colonization.

In addition to being influenced by variations in oxygen levels, shifts in the composition of the microbiota colonizing the root canal system can also be dependent upon the dynamics of nutrient utilization. Saccharolytic species dominate the very early stages of the infectious process but are soon outnumbered by asaccharolytic species, which will dominate later stages. Even though the necrotic pulp tissue can be regarded as a finite source of nutrients to bacteria (given the small volume of tissue that is progressively degraded), induction of periradicular inflammation guarantees a sustainable source of nutrients, particularly in the form of proteins and glycoproteins present in the exudate that seep into the canal. At this stage of the infectious process, bacteria that have a proteolytic capacity or establish a cooperative

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interaction with those that can utilize this substrate in the metabolism, start to dominate.[278] Therefore, as the infectious process reaches the stage of induction of periradicular inflammation, proteins become the principal nutrient source, particularly in the apical part of the canal, favoring the establishment of anaerobic species that utilize peptides or amino acids in their metabolism (see Fig. 15.24).

Because primary endodontic infections are usually characterized by mixed communities, different bacterial species are in close proximity with one another, and interactions become inevitable. Thus, establishment of certain species in the root canal is also influenced by interactions with other species. In this regard, early colonizers play an important role in dictating which species will live along with them in the community. Bacterial interactions can be positive or negative. Positive interactions enhance the survival capacity of the interacting bacteria and enable different species to coexist in habitats where neither could exist alone. For instance, interbacterial nutritional interactions are important ecologic determinants that result in higher metabolic efficiency of the whole community. Nutritional interactions are mainly represented by food webs, including utilization of metabolic end products from one species by another and bacterial cooperation for the breakdown of complex host-derived substrates. Moreover, one species can provide growth conditions favorable to another—for example, by reducing oxygen tension in the environment and favoring the establishment of anaerobes, or by releasing proteinases that can provide protection from host defenses. Negative interactions in turn act as feedback mechanisms that limit population densities. Examples include competition (for nutrients and space) and amensalism (or antagonism, when one species produces a substance that inhibits another species, e.g., bacteriocins).

Many species adhere directly to host surfaces, whereas other species adhere to bacteria already attached to the surface. The latter is called *coaggregation* and is a highly specific phenomenon with regard to the partners involved.[98] A given pair of species can attach to each other by means of specific receptor-adhesin interactions, which are usually lectin-like interactions (attachment of a specific protein on the surface of one species to a specific carbohydrate on the surface of the other). Coaggregation can favor colonization of host surfaces and also facilitate metabolic interactions

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between the partners. Coaggregation has been demonstrated for several pairs of bacterial taxa found in endodontic infections.[97]

Other microorganisms in endodontic infections

Fungi

Fungi are eukaryotic microorganisms that may colonize the oral cavity, especially *Candida* species, but they have been only occasionally detected in primary intraradicular infections.[49,101,122,248] Fungi are more frequently detected in canals of teeth with posttreatment disease (discussed later).

Archaea

Archaea consist of a highly diverse group of prokaryotes distinct from bacteria. Members of this domain have been traditionally recognized as extremophiles, but some of these microorganisms have also been found to flourish in nonextreme environments, including the human body.[48] No member of the *Archaea* domain has been described as a human pathogen. Several studies have failed to detect archaea in samples from primary endodontic infections.[180,181,242] However, other studies have reported on the detection of these organisms, including a *Methanobrevibacter oralis*–like phylotype, in some cases.[136,137,291,293] Because archaea have not been consistently detected in infected root canals, their role, if any, in the pathogenesis of apical periodontitis is questionable.

Viruses

Viruses are inanimate particles composed of a nucleic acid molecule (DNA or RNA) and a protein coat. On their own, they have no metabolism. In order to replicate the viral genome, they need to infect living cells and use the cell's machinery; as a consequence, they cannot prosper in the necrotic root canal. Viruses have been detected in root canals only in teeth with vital pulps. A study found the human immunodeficiency virus (HIV) in vital pulps of HIV-seropositive individuals.[62] Another study reported the occurrence of some herpesviruses in normal and inflamed vital pulps.[104]

Apical periodontitis lesions, in turn, are characterized by dense accumulations of viable immune and connective tissue cells. Sabeti and Slots

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first reported on the occurrence of herpesviruses in these lesions.[190–193] It was hypothesized that herpesviruses might be implicated in the pathogenesis of apical periodontitis as a direct result of virus infection and replication or as a result of virally induced impairment of local host defenses, which would favor the overgrowth of pathogenic bacteria in the very apical part of the root canal.[261] Bacterial challenge emanating from the canals might cause an influx of virus-infected cells into the periradicular tissues. Reactivation of herpesviruses by tissue injury induced by bacteria would evoke impairment of host immune response in the periradicular microenvironment, changing the potential of local defense cells to mount an adequate response against infectious agents. In addition, herpesviruses may directly stimulate inflammatory cells to release proinflammatory cytokines.[120,301]

Herpesviruses have been found in samples from symptomatic apical periodontitis lesions,[191,193] abscesses,[27,54] large lesions,[192,193] and lesions from HIV-positive patients.[194] Most studies reported on the occurrence of herpesviruses in samples of apical periodontitis, but this cannot be necessarily interpreted as active infection. Herpesvirus-infected inflammatory cells may be attracted to the periradicular tissues in response to bacterial infection. Once these cells accumulated in the area, herpesviruses can be easily detected. While more consistent information is not available, the role of herpesviruses in the pathogenesis of apical periodontitis, if any, cannot be ascertained.

Persistent/secondary endodontic infections

As defined earlier in this chapter, persistent intraradicular infections are caused by microorganisms that resisted intracanal antimicrobial procedures and survived in the treated canal. Involved microorganisms are usually remnants of a primary infection. The secondary infection, in turn, is caused by microorganisms that at some time entered the root canal system secondary to clinical intervention. The moment can be during treatment, between appointments, or even after root canal filling. In any circumstance, if penetrating microorganisms manage to adapt themselves to the new environment, surviving and flourishing, a secondary infection is established. Species involved can be oral species or not, depending on the source of

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secondary infection.

Persistent and secondary infections are for the most part clinically indistinguishable. Exceptions include infectious complications (such as an apical abscess) arising after the treatment of noninfected vital pulps or cases in which apical periodontitis was absent at the time of treatment but present on the follow-up radiograph. Both situations are typical examples of secondary infections. Both persistent and secondary infections can be responsible for several clinical problems, including persistent exudation ("wet" canal), persistent symptoms, interappointment flare-ups, and failure of the endodontic treatment, characterized by a posttreatment apical periodontitis lesion.

Persistent/secondary infections and treatment failure

Intraradicular infections, either persistent or secondary, can be regarded as the major causes of posttreatment apical periodontitis (Fig. 15.25). Other causes include extraradicular infections (discussed later in this chapter) and non-microbial factors, for which there is only weak evidence coming from case reports and speculation.[129] The major role of persistent/secondary infections in treatment failure is supported by two strong evidence-based arguments. First, it has been demonstrated that there is an increased risk of adverse treatment outcome when bacteria are present in the canal at the time of obturation.[52,297] Second, virtually all root canal–treated teeth evincing persistent apical periodontitis lesions have been demonstrated to harbor an intraradicular infection.[106,108,150,167,173,198,230,277] Based on these arguments, studies investigating bacteria remaining in the root canals at the obturation stage disclose species that have the potential to influence the treatment outcome *(predicting the outcome).* On the other hand, studies dealing with the microbiota of root canal–treated teeth with apical periodontitis show the association of species with treatment failure, because the microorganisms detected are likely to be the cause of posttreatment disease *(outcome established).*

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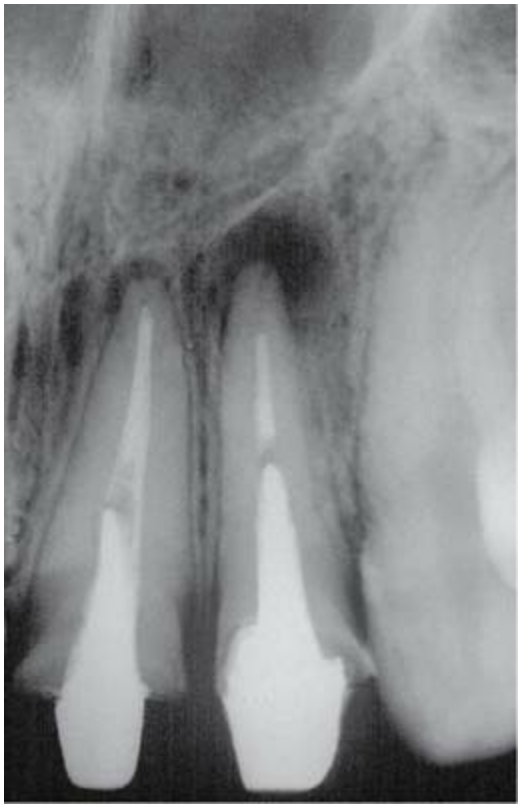

FIG. 15.25 Posttreatment apical periodontitis lesions in root canal–treated teeth. In inadequately treated canals, the microbiota is similar to primary infections. In cases apparently well treated, fewer species are found. Regardless of treatment quality, persistent or secondary intraradicular infections are the main causative agents of endodontic treatment failure.

Radiograph of two teeth shows a lesion present in between apical and coronal region depicted as a radiolucent patch.

Bacteria at the root canal obturation stage

Diligent antimicrobial treatment may still fail to completely eliminate bacteria from the infected root canal system.[235] This is mostly because persisting bacteria are located in areas inaccessible to treatment procedures,[247] although resistance to some substances have been suggested as cause of bacterial persistence.[57,300] Whatever the cause of persistence, bacterial diversity and density in infected canals are substantially reduced after treatment. Root canal samples positive for bacterial growth after chemomechanical procedures, followed or not by intracanal medication, have

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been shown to harbor one to five bacterial species per case, and the number of persistent bacterial cells usually varies from 10^2 to 10^5 per sample.[9,22,130,174,,228,229,292]

No single species has been significantly found to persist after treatment procedures. Gram-negative bacteria, which are common members of primary infections, are usually eliminated. Exceptions include some anaerobic rods, such as *F. nucleatum* and *Prevotella* species, which are among the species found in postinstrumentation or postmedication samples.[22,67,147,197,249,258,309] Most studies on this subject have clearly revealed that when bacteria resist treatment procedures, gram-positive bacteria are more frequently present. Gram-positive facultatives or anaerobes often detected in these samples include streptococci, *P. micra, Propionibacterium* species, *P. alactolyticus, Actinomyces* species, lactobacilli, *E. faecalis*, and *Olsenella uli*.[22,24–26,29,67,145,147,179–181,197,225,228,229,258,279] This supports the notion that gram-positive bacteria can be more resistant to antimicrobial treatment measures and have the ability to adapt to the harsh environmental conditions in instrumented and medicated root canals. As-yet-uncultivated bacteria have also been detected in posttreatment samples,[138,197] indicating that they may also resist antimicrobial treatment.

Bacteria persisting in the root canal after chemomechanical procedures or intracanal medication will not always maintain an infectious process. This statement is supported by evidence that some apical periodontitis lesions healed even after bacteria were isolated from the canal at the obturation stage.[52,258] There are some possible explanations[235]:

- Residual bacteria may die after obturation because of toxic effects of the filling material, access denied to nutrients, or disruption of bacterial ecology.
- Residual bacteria may be present in quantities and virulence subcritical to sustaining periradicular inflammation.
- Residual bacteria remain in locations of the root canal system where access to periradicular tissues is denied.

Bacteria that resist intracanal procedures and are present in the canal at the time of obturation can influence the outcome of the endodontic treatment

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provided they do the following[235]:

- Have the ability to withstand periods of starvation, scavenging for low traces of nutrients or assuming a dormant state or a state of low metabolic activity (viable but noncultivable -VBNC), to prosper again when the nutrient source is reestablished.
- Resist treatment-induced disturbances in the ecology of the bacterial community, including disruption of quorum-sensing systems, food webs, genetic exchanges, and disorganization of protective biofilm structures.
- Reach a critical population density (load) necessary to inflict damage to the host.
- Have unrestrained access to the periradicular tissues through apical/lateral foramina or iatrogenic root perforations.
- Possess virulence attributes that are expressed in the modified environment and reach concentrations adequate to directly or indirectly induce damage to the periradicular tissues.
- In this context, the host resistance to infection is also an important and decisive counteracting factor.

Microbiota in root canal–treated teeth

The microbiota in root canal–treated teeth with apical periodontitis exhibits decreased diversity in comparison to primary infections. In addition, canals apparently well treated harbor less species than canals with inadequate treatment; the latter can exhibit similar species richness to untreated canals.[150,184,198,230,] The bacterial density in treated teeth with posttreatment disease varies from 10^3 to 10^7 per canal.[19,145,182,207,] Likewise, inadequately treated canals are expected to have a higher bacterial density than well-treated canals.

Several culture and molecular biology studies have revealed that *E. faecalis* is the most frequent species in root canal–treated teeth, with prevalence values in some studies reaching more than 90% of cases (Fig. 15.26).[121,150,173,182,187,207,230,277,312] Rôças et al.[187] reported that root canal–treated teeth are about nine times more likely to harbor *E. faecalis* than cases of primary infections. This suggests that other members of a mixed bacterial

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consortium commonly present in primary infections can inhibit this species, and that the bleak environmental conditions within obturated root canals do not prevent its survival. The fact that *E. faecalis* has been commonly recovered from teeth treated at multiple visits or left open for drainage[257] suggests that this species is a secondary invader capable of colonizing the canal and resisting treatment. In other words, *E. faecalis* may cause secondary infections that later become persistent. *E. faecalis is* considered a transient species in the oral cavity; its source may be food.[311]

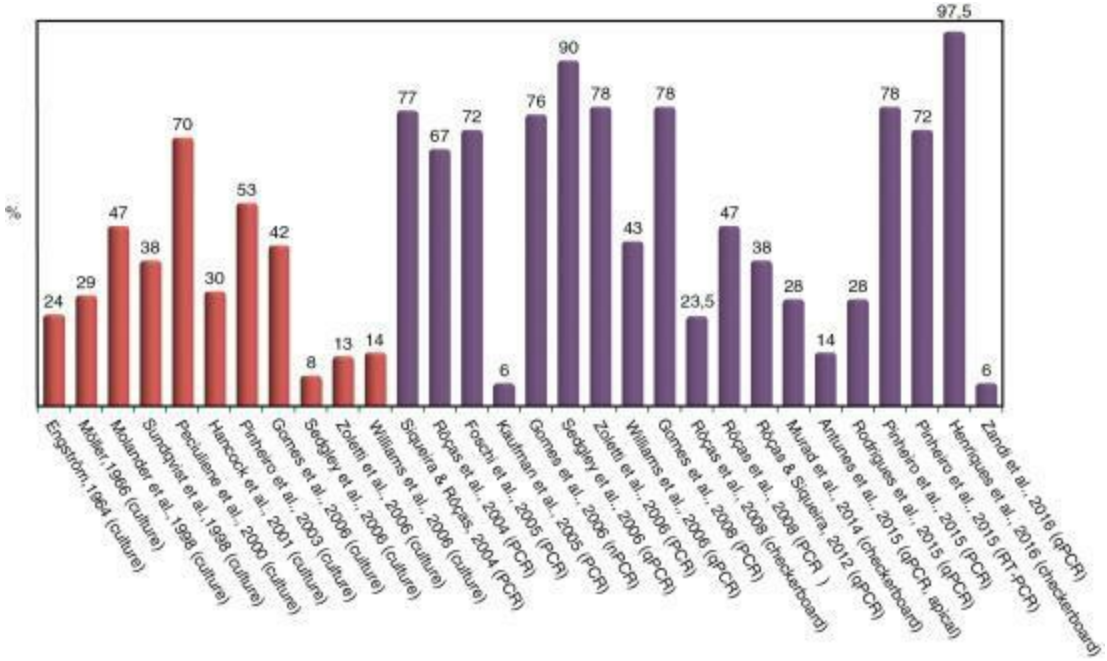

FIG. 15.26 Prevalence of *Enterococcus faecalis* in samples from root canal–treated teeth with apical periodontitis.

Bar chart shows percentage distribution of enterococcus faecalis divided into two categories as follows:

The data from culture is given as follows: Engström,1964 (culture): 24; Möller,1966 (culture):29; Molander et al.,1998 (culture): 47; Sundqvist et al.,1998 (culture):38; Peciuliene et al., 2000 (culture): 70; Hancock et al., 2001 (culture): 30; Pinheiro et al., 2003 (culture0: 53; Gomes et al., 2006 (culture): 42; Sedgley et al., 2006 (culture): 8; Zoletti et al., 2006 (culture):13; and Williams et al., 2006 (culture): 14.

The data from molecular studies is given as follows: Siqueira & Rôças, 2004 (PCR): 77; Rôças et al., 2004 (PCR): 67; Foschi et al., 2005 (PCR): 72; Kaufman et al., 2005 (PCR): 6; Gomes et al., 2006 (nPCR); 76; Sedgley et al.,

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2006 (qPCR): 90; Zoletti et al., 2006 (PCR): 78; Williams et al., 2006 (qPCR): 43; Gomes et al., 2008 (PCR): 78; Rôças et al., 2008 (checkerboard); 23,5; Rôças et al., 2008 (PCR):47; Rôças & Siqueira, 2012 (qPCR) 38; Murad et al., 2014 (checkerboard): 28; Antunes et al., 2015 (qPCR, apical): 14; Rodrigues et al., 2015 (qPCR); 28; Pinheiro et al., 2015 (PCR): 78; Pinheiro et al., 2015 (RT-PCR): 72; Henriques et al., 2016 (checkerboard): 92,5; and Zandi et al., 2016 (qPCR): 6.

Source: Data from culture *(redx bars)* and molecular *(purple bars)* studies follow.

Molecular Studies

1. Antunes HS, Rôças IN, Alves FR, Siqueira JF Jr: Total and specific bacterial levels in the apical root canal system of teeth with post-treatment apical periodontitis. *J Endod* 41: 1037, 2015.
2. Foschi F, Cavrini F, Montebugnoli L, et al: Detection of bacteria in endodontic samples by polymerase chain reaction assays and association with defined clinical signs in Italian patients. *Oral Microbiol Immunol* 20:289, 2005.
3. Gomes BP, Pinheiro ET, Jacinto RC, et al: Microbial analysis of canals of root-filled teeth with periapical lesions using polymerase chain reaction. *J Endod* 34:537, 2008.
4. Gomes BP, Pinheiro ET, Sousa EL, et al: *Enterococcus faecalis* in dental root canals detected by culture and by polymerase chain reaction analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:247, 2006.
5. Henriques LC, de Brito LC, Tavares WL, et al: Microbial ecosystem analysis in root canal infections refractory to endodontic treatment. *J Endod* 42: 1239, 2016.
6. Kaufman B, Spangberg L, Barry J, Fouad AF: *Enterococcus* spp. in endodontically treated teeth with and without periradicular lesions. *J Endod* 31:851, 2005.
7. Murad CF, Sassone LM, Faveri M, et al: Microbial diversity in persistent root canal infections investigated by checkerboard DNA-DNA hybridization. *J Endod* 40: 899, 2014.
8. Pinheiro ET, Candeiro GT, Teixeira SR, et al: RNA-based assay demonstrated *Enterococcus faecalis* metabolic activity after chemomechanical procedures. *J Endod* 41: 1441, 2015.
9. Rôças IN, Hulsmann M, Siqueira JF Jr: Microorganisms in root canal-treated teeth from a German population. *J Endod* 34:926, 2008.
10. Rôças IN, Siqueira JF Jr, Santos KR: Association of *Enterococcus faecalis* with different forms of periradicular diseases. *J Endod* 30:315, 2004.
11. Rôças IN, Siqueira JF Jr: Characterization of microbiota of root canal-treated teeth with posttreatment disease. *J Clin Microbiol* 50:1721, 2012.
12. Rodrigues RC, Antunes HS, Neves MA, et al: Infection control in retreatment cases: in vivo antibacterial effects of 2 instrumentation

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systems. *J Endod* 41: 1600, 2015.
13. Sedgley C, Nagel A, Dahlen G, et al: Real-time quantitative polymerase chain reaction and culture analyses of *Enterococcus faecalis* in root canals. *J Endod* 32:173, 2006.
14. Siqueira JF Jr, Rôças IN: Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:85, 2004.
15. Williams JM, Trope M, Caplan DJ, Shugars DC: Detection and quantitation of *Enterococcus faecalis* by real-time PCR (qPCR), reverse transcription-PCR (RT-PCR), and cultivation during endodontic treatment. *J Endod* 32:715, 2006.
16. Zandi H, Rodrigues RC, Kristoffersen AK, et al: Antibacterial effectiveness of 2 root canal irrigants in root-filled teeth with infection: a randomized clinical trial. *J Endod*. 42: 1307, 2016.
17. Zoletti GO, Siqueira JF Jr, Santos KR: Identification of *Enterococcus faecalis* in root-filled teeth with or without periradicular lesions by culture-dependent and-independent approaches. *J Endod* 32:722, 2006.

Culture Studies

1. Engström B: The significance of enterococci in root canal treatment. *Odontol Rev* 15:87, 1964.
2. Gomes BP, Pinheiro ET, Sousa EL, et al: *Enterococcus faecalis* in dental root canals detected by culture and by polymerase chain reaction analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:247, 2006.
3. Hancock HH 3rd, Sigurdsson A, Trope M, Moiseiwitsch J: Bacteria isolated after unsuccessful endodontic treatment in a North American population. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:579, 2001.
4. Molander A, Reit C, Dahlen G, Kvist T: Microbiological status of root-filled teeth with apical periodontitis. *Int Endod J* 31:1, 1998.
5. Möller AJR: Microbial examination of root canals and periapical tissues of human teeth. *Odontol Tidskr* 74(suppl):1, 1966.
6. Peciuliene V, Balciuniene I, Eriksen HM, Haapasalo M: Isolation of *Enterococcus faecalis* in previously root-filled canals in a Lithuanian population. *J Endod* 26:593, 2000.
7. Pinheiro ET, Gomes BP, Ferraz CC, et al: Microorganisms from canals of root-filled teeth with periapical lesions. *Int Endod J* 36:1, 2003.
8. Sedgley C, Nagel A, Dahlen G, et al: Real-time quantitative polymerase chain reaction and culture analyses of *Enterococcus faecalis* in root canals. *J Endod* 32:173, 2006.
9. Sundqvist G, Figdor D, Persson S, Sjogren U: Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 85:86, 1998.

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10. Williams JM, Trope M, Caplan DJ, Shugars DC: Detection and quantitation of *Enterococcus faecalis* by real-time PCR (qPCR), reverse transcription-PCR (RT-PCR), and cultivation during endodontic treatment. *J Endod* 32:715, 2006.
11. Zoletti GO, Siqueira JF Jr, Santos KR: Identification of *Enterococcus faecalis* in root-filled teeth with or without periradicular lesions by culture-dependent and -independent approaches. *J Endod* 32:722, 2006.

For a given microorganism to survive in root canal–treated teeth, it has to resist intracanal disinfection procedures and adapt to the harsh environmental conditions caused by treatment. The ability of *E. faecalis* to penetrate dentinal tubules, sometimes to a deep extent (Fig. 15.27),[73,223] can enable it to escape the action of endodontic instruments and irrigants used during chemomechanical preparation.[73,222] Moreover, its ability to form biofilms in root canals can be important for its resistance to and persistence after intracanal antimicrobial procedures.[43] *Enterococcus faecalis* is also resistant to calcium hydroxide.[73] Finally, environmental cues can regulate gene expression in *E. faecalis*, affording this bacterium the ability to adapt to varying (and adverse) conditions.[89] Indeed, *E. faecalis* can enter a VBNC state,[109] which is a survival mechanism adopted by several bacteria when exposed to unfavorable environmental conditions.[110] In the VBNC state, bacteria lose the ability to grow in culture media but maintain viability and pathogenicity and sometimes can resume division when optimal environmental conditions are restored. *E. faecalis* has the capacity to recover from a prolonged starvation state in obturated canals,[208] suggesting that viable cells of this species, entombed at the time of canal obturation, may provide a long-term nidus for subsequent infection.

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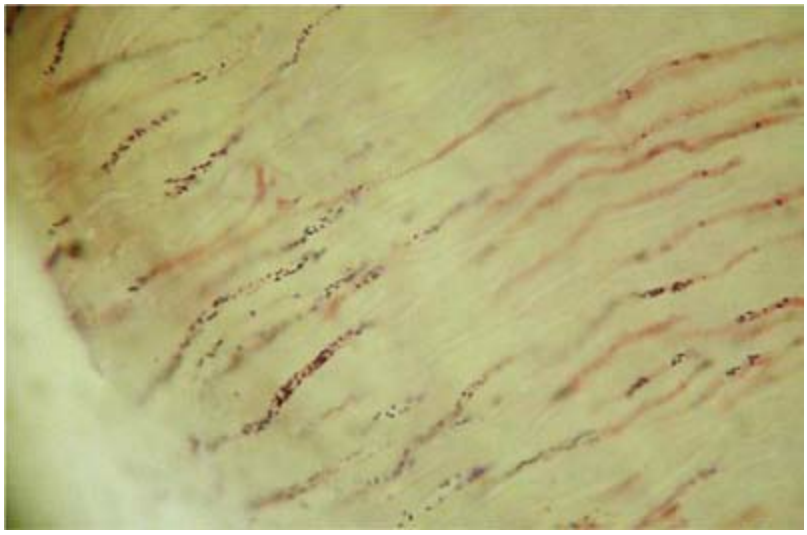

FIG. 15.27 Dentinal tubule infection by *Enterococcus faecalis* in a dog's tooth after experimental infection. Notice that cells invaded the entire extent of some tubules up to the cementum (Brown and Brenn staining, ×1000).

Radiograph shows curvy pink and blue lines on a dull background.

Taken together, all these properties help explain the significantly high prevalence of *E. faecalis* in root canal–treated teeth. Although association of this species with posttreatment disease is suggested by cross-sectional studies and supported by the species attributes that allow it to survive under unfavorable environmental conditions, causation is unproved. In fact, the following findings from studies carried out in independent laboratories have questioned the status of *E. faecalis* as the main causative agent of endodontic failures:

- Despite being easily cultivated, *E. faecalis* is not detected in all studies evaluating the microbiota of root canal–treated teeth with posttreatment disease.[28,188]
- In many studies that detected *E. faecalis*, it was rarely one of the most dominant species in the microbiota of retreatment cases.[81,172,184,198,]
- *E. faecalis* is found in similar prevalence in root canal–treated teeth with or without apical periodontitis.[92,312]

Other bacteria found in endodontically treated teeth with apical

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periodontitis include *Streptococcus* species and some fastidious anaerobic species, such as *P. alactolyticus*, *Propionibacterium* species, *Filifactor alocis*, *Dialister* species, *Tannerella forsythia*, *P. micra*, and *Prevotella* species (Fig. 15.28).[7,68,81,121,150,172,182,184,198,230,233,277,309,310] Uncultivated phylotypes correspond to 55% of the taxa detected in treated canals, and collectively they can also be in high proportions, corresponding to about half of the 16S rRNA gene sequences retrieved in clone libraries.[198] Detection of uncultivated phylotypes helps explain why culture studies fail to detect bacteria in some treated root canals.

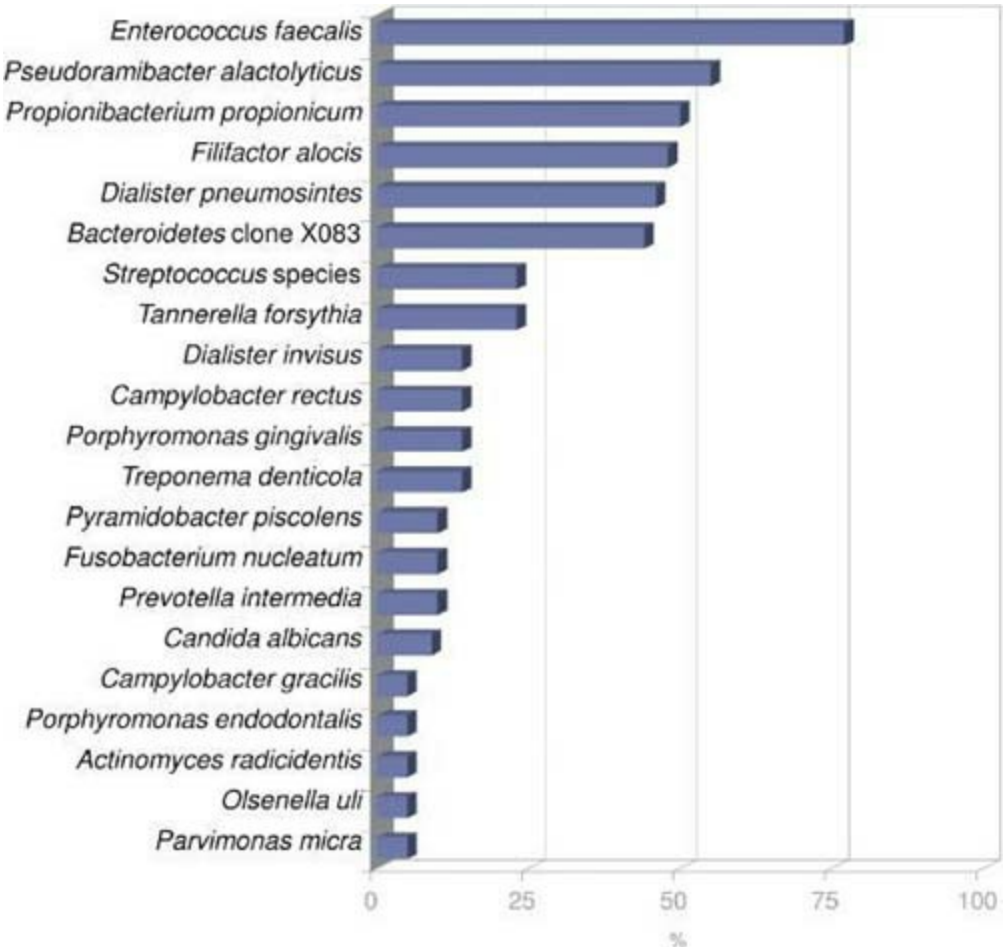

FIG. 15.28 Prevalence of microorganisms detected in root canal–treated teeth with posttreatment disease.

Horizontal bar chart shows percentage distribution of bacterial species found in root canal–treated teeth with posttreatment disease are marked in the decreasing order from top to bottom as follows:

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50 to 75 percent: Enterococcus faecalis and Psueodoramibacter alactolyticus.

25 to 50 percent: Propionibacterium propionicum; Folifactor alocis; Dialister pneumosintes; and Bacteriodetes clone X 083.

0 to 25 percent: Streptococcus species; Tannerella forsythia; Dialister invisus; Campylobacter rectus; Porphyromonas gingvalis; Treponema denticola; Pyramidobacte pisclens; Fusobacterium nucleatum; Prevotella intermedia; Candida albicans; Campylobacter gracilis; Porphromonas endodontalis; Actinomyces radicidentis; Olsenella uli; and Parvimonas micra.

Source: Data from the authors' findings using a taxon-specific polymerase chain reaction assay.

The bacterial community profiles in treated cases vary from individual to individual, indicating that distinct bacterial combinations can play a role in treatment failure (Fig. 15.29).[184,198,220,309] Molecular findings indicate that the microbiota of root canal–treated teeth with apical periodontitis is more complex than previously demonstrated by culture studies.

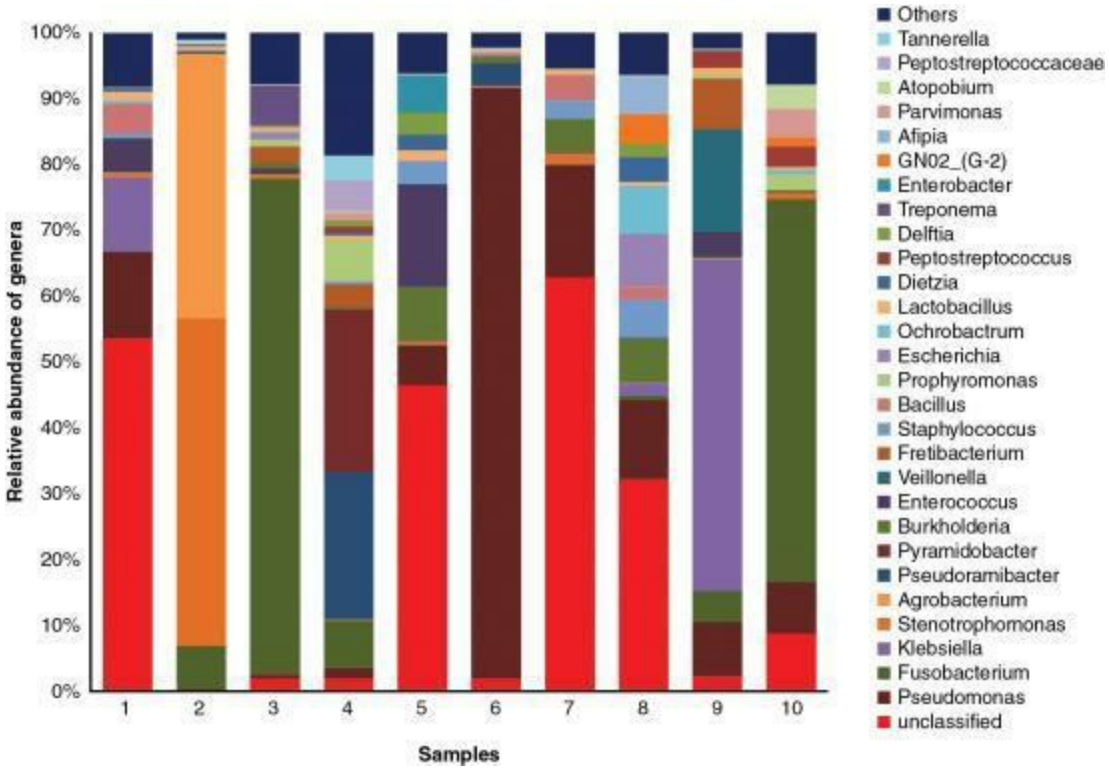

FIG. 15.29 Interindividual variability in bacterial diversity (richness and abundance) at the genus levels in cryopulverized apices from teeth with posttreatment apical periodontitis. Notice that no two samples are

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the same in terms of bacterial composition.

Stacked bar chart of samples against relative abundance of genera (in percentage) shows the sample-wise data as follows:

Sample 1: Majority of population is unclassified.

Sample 2: Majority population is of Agrobacterium and Stenotrophomonas.

Sample 3: Majority population is of Fusobacterium.

Sample 4: Mixed population of Fusobacterium, Pseudomonas; Peptostreptococcus.

Sample 5: Mixed population of Delftia; Fusobacterium; Treponema; with majority as unclassified.

Sample 6: 80 percent population is of pyramidobacter.

Sample 7: 60 percent of population is unclassified; remaining contains pyramidobacter, Afipia; Parvimonas; and others.

Sample 8: 30 percent is unclassified; remaining is a mixed population.

Sample 9: 40 percent is Klebsiella; remaining is a mixed population of Veillonella; fusobacterium; pseudomonas; Fretibacterium; Peptostreptococcus; and other.

Sample 10: 50 percent of fusobacterim; remaining is a mixed population.
Note: All data is approximate.

Source: (Based on data from Siqueira JF Jr, Antunes HS, Rôças IN, Rachid CT, Alves FR. Microbiome in the apical root canal system of teeth with post-treatment apical periodontitis. *PLoS One* 11:e0162887, 2016)

Fungi are only occasionally found in primary infections, but *Candida* species have been detected in root canal–treated teeth in up to 18% of the cases.[28,49,121,122,145,150,230,277] Fungi gain access to root canals via contamination during endodontic therapy (secondary infection) or they overgrow after inefficient intracanal antimicrobial procedures that cause an imbalance in the primary endodontic microbiota.[254] *Candida albicans* is by far the most commonly detected fungal species in root canal–treated teeth. This species has several properties that can be involved in persistence following treatment, including its ability to colonize and invade dentin[211,212,246] and resistance to calcium hydroxide.[298,300]

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Extraradicular infections

Apical periodontitis lesions are formed in response to intraradicular infection and by and large constitute an effective barrier against spread of the infection to the alveolar bone and other body sites. In most situations, apical periodontitis inflammatory lesions succeed in preventing bacteria from invading the periradicular tissues. Nevertheless, in some specific circumstances, bacteria can overcome this defense barrier and establish an extraradicular infection. The most common form of extraradicular infection is the acute apical abscess, characterized by purulent inflammation in the periradicular tissues in response to a massive egress of virulent bacteria from the root canal. Extraradicular infections in the form of biofilm covering the outer root surface, along with complex bacterial arrangements in the apical canal system, are commonly observed in cases of chronic abscesses (with sinus tract) and "wet canals"(persistent exudation).[157,159] Extraradicular biofilm, adhered to the external root surface around the apical foramina, and apical actinomycosis, characterized by cohesive actinomycotic colonies in the lesion body (Fig. 15.30),[78,132,161,168,] have been regarded as possible extraradicular causes of posttreatment apical periodontitis.

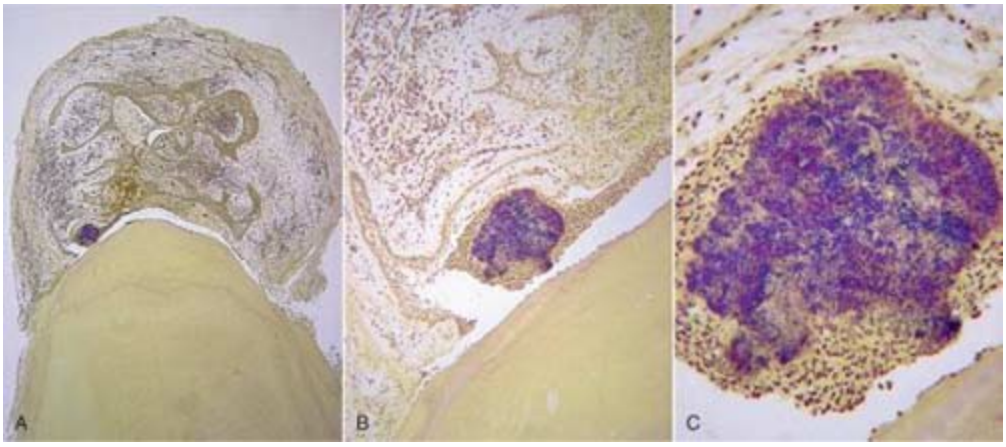

FIG. 15.30 Bacterial aggregate in an epithelialized apical periodontitis lesion, suggestive of actinomycosis. This case presented a severe flare-up in the past, but the tooth was asymptomatic at the time of extraction. **A,** Section not passing through the canal; note cyst cavity and bacterial aggregation on the left (magnification ×25). **B,** Detail of area with colony (magnification ×100). **C,** Higher magnification of colony, which is surrounded by neutrophils (magnification ×400).

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Sections stained with a Taylor-modified Brown and Brenn technique.

A) Micrograph shows a blue dot depicting bacterial aggregation in the cavity.

B) Magnified view of A shows blue stain surrounded by brown layer present in clear between root and lesion.

C) Magnified view of B shows multiple blue dots on a brown background.

Source: (Courtesy Dr. Domenico Ricucci.)

An extraradicular infection can develop as follows[216]:

- It can be a result of direct advance of some bacterial species that overcome host defenses concentrated near or beyond the apical foramen, an extension of the intraradicular infectious process, or bacterial penetration into the lumen of a cyst that is in direct communication with the apical foramen ("bay" cyst). The borderline between the infecting endodontic microbiota and the host defenses is often located intraradicularly, short of or at the apical foramen. In some cases, however, bacteria may reach the periradicular tissues, and the borderline is then situated extraradicularly, beyond the boundaries of the apical foramen (Fig. 15.31). In the latter situation, the whole infectious process is a continuum composed of an intraradicular and an extraradicular segment, in which the former usually fosters the latter (see Fig. 15.31). It has been suggested that in some cases, the extraradicular segment may become independent of the intraradicular component of the infectious process.[168]
- It can result from bacterial persistence in the apical periodontitis lesion after remission of an acute apical abscess. The acute apical abscess is for the most part clearly dependent on the intraradicular infection; once the intraradicular infection is properly controlled by root canal treatment or tooth extraction and drainage of pus is achieved, the extraradicular infection is handled by the host defenses and usually subsides. Nonetheless, it should be appreciated that in some rare cases, bacteria that have participated in acute apical abscesses may persist in the periradicular tissues following resolution of the acute response and establish a persistent extraradicular infection associated with chronic periradicular

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inflammation, sometimes resulting in an actively draining sinus tract.

- It can be a sequel to apical extrusion of debris during root canal instrumentation (particularly after overinstrumentation). Bacteria embedded in dentinal chips can be physically protected from the host defense cells and therefore can persist in the periradicular tissues and sustain periradicular inflammation. In addition, large bacterial flocs ("planktonic biofilms") may be located in the apical canal and be extruded during root canal procedures; once in the periapical tissues, they can induce or maintain inflammation (Fig. 15.32).[161] The virulence and quantity of the extruded bacteria, as well as the host's ability to deal with infection, will be decisive factors dictating whether an extraradicular infection will develop or not as a result of apical extrusion of debris.

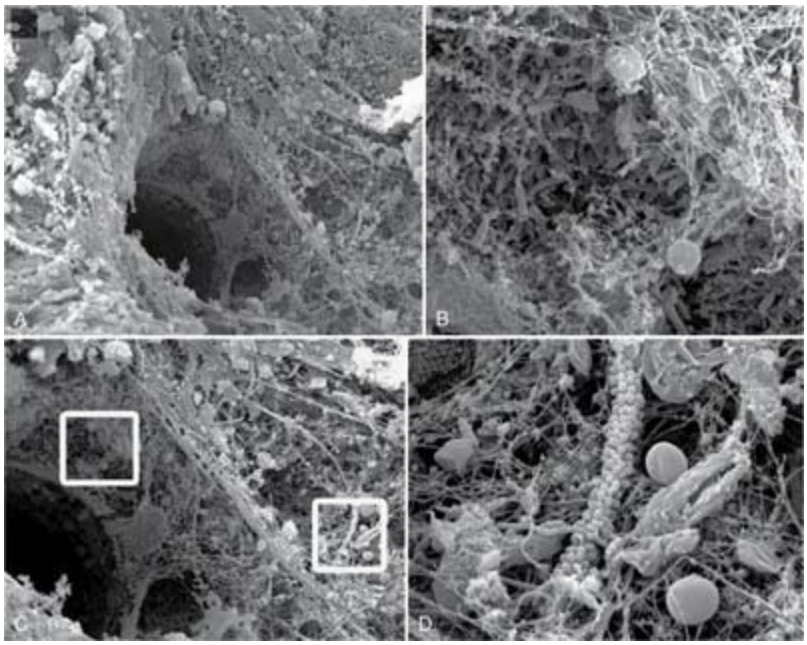

FIG. 15.31 Scanning electron micrograph showing extensive bacterial colonization in the very apical part of the canal, near and at the apical foramen (**A–B,** magnification ×550 and magnification ×850, respectively). **C,** Higher magnification of left inset in **B** showing a bacterial biofilm adhered to the very apical canal walls (magnification ×3700). **D,** Higher magnification of right inset in **B** showing a fully developed "corncob" in tissue meshwork adjacent to the apical foramen (magnification ×4000).

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Set of four scanning electron micrographs marked A through D depict formation of bacterial biofilm in and around wall of canal at the apical foramen.

Source: (Modified from Siqueira JF Jr, Lopes HP: Bacteria on the apical root surfaces of untreated teeth with periradicular lesions: a scanning electron microscopy study. *Int Endod J* 34:216, 2001.)

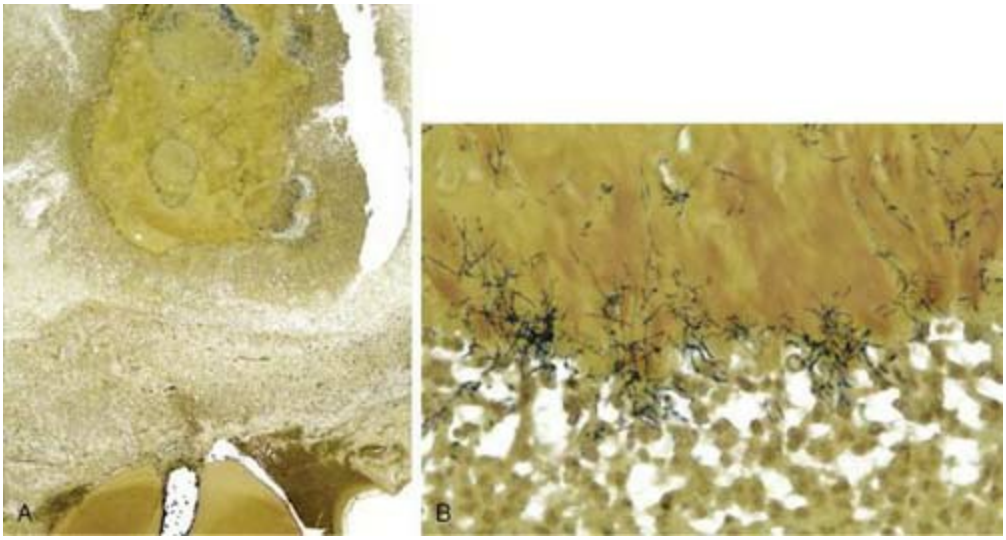

FIG. 15.32 **A,** Large bacterial floc located in the center of the apical periodontitis lesion, at some distance from the apical foramen. This represents an independent extraradicular infection that was the cause of persistent disease (magnification ×16). **B,** Higher magnification of the periphery of the large colony, showing intense accumulation of inflammatory cells (magnification ×400).

A) Micrograph shows cluster of tiny blue dots in the apical lesion.

B) Magnified view of A shows fine, blue dots and lines marked at the periphery.

Source: (From Ricucci D, Lopes WP, Loghin S, Rocas IN, Siqueira JF Jr. Large bacterial floc causing an independent extraradicular infection and posttreatment apical periodontitis: a case report. *J Endod* 44:1308, 2018.)

Conceivably, the extraradicular infection can be dependent on or independent of the intraradicular infection.[215] Independent extraradicular infections are those that are no longer fostered by the intraradicular infection and can persist even after successful eradication of the latter. For an

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extraradicular infection to be classified as independent of the intraradicular infection, a comprehensive analysis of the apical canal should demonstrate absence of infection. So far, only a few cases have been reported that fulfill this requirement.[161,168]

It is still controversial whether asymptomatic apical periodontitis lesions can harbor bacteria for very long beyond initial tissue invasion.[12] Studies using culture-dependent[270,284,304] or culture-independent molecular biology methods, such as checkerboard hybridization,[59,272] FISH,[271] clone library analysis,[77] and HTS,[189] have reported the extraradicular occurrence of several bacterial species, especially anaerobes, in teeth that did not respond favorably to the root canal treatment. Because of the cross-sectional nature of the studies, it is not possible to infer if the extraradicular bacteria were transient or established therein. Apart from a discussion about whether contamination can be effectively prevented during surgical sampling of apical periodontitis lesions, these studies did not evaluate the bacteriologic conditions of the apical part of the root canal either. This makes it difficult to ascertain whether those extraradicular infections were dependent on or independent of an intraradicular infection. Actually, findings from a study evaluating both the resected root ends and the apical periodontitis lesions from treated teeth suggested that the large majority of cases of extraradicular infections are maintained by a concomitant apical root canal infection.[269]

Extraradicular biofilms are infrequent in untreated teeth with apical periodontitis, and when present they are virtually always associated with intraradicular biofilms; the occurrence of a sinus tract is usually indicative of the presence of an extraradicular biofilm.[159,164] The high success rate of nonsurgical root canal treatment[36,162,259] suggests that, in the occasional cases it is present, the extraradicular infection component may not suffice to maintain apical periodontitis provided the intraradicular infection is properly managed. This is consistent with the concept of dependent infection. Even in root canal–treated teeth with recalcitrant lesions in which a higher incidence of extraradicular bacteria has been reported, a high rate of healing following retreatment[53,259] suggests that the major cause of posttreatment disease is located within the root canal system, characterizing a persistent or secondary intraradicular infection.[108,121,150,167,184,230,277,309,310]

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References

1. Aas JA, Griffen AL, Dardis SR, et al: *Bacteria of dental caries in primary and permanent teeth in children and young adults*, J Clin Microbiol 2008;46: 1407-1417.
2. Aas JA, Paster BJ, Stokes LN, et al: *Defining the normal bacterial flora of the oral cavity*, J Clin Microbiol 2005;43: 5721-5732.
3. Ackermans F, Klein JP, Frank RM. *Ultrastructural localization of immunoglobulins in carious human dentine*, Arch Oral Biol 1981;26: 879-886.
4. Ahn J, Yang L, Paster BJ, et al: *Oral microbiome profiles: 16S rRNA pyrosequencing and microarray assay comparison*, PLoS One 2011;6: e22788.
5. Allison DG. *The biofilm matrix*, Biofouling 2003;19: 139-150.
6. Amann RI, Ludwig W, Schleifer KH. *Phylogenetic identification and in situ detection of individual microbial cells without cultivation*, Microbiol Rev 1995;59: 143-169.
7. Anderson AC, Hellwig E, Vespermann R, et al: *Comprehensive analysis of secondary dental root canal infections: a combination of culture and cultureindependent approaches reveals new insights*, PLoS One 2012;7: e49576.
8. Armada-Dias L, Breda J, Provenzano JC, et al: *Development of periradicular lesions in normal and diabetic rats*, J Appl Oral Sci 2006;14: 371-375.
9. Arruda MEF, Neves MAS, Diogenes A, et al: *Infection control in teeth with apical periodontitis using a triple antibiotic solution or calcium hydroxide with chlorhexidine: a randomized clinical trial*, J Endod

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2018;44: 1474-1479.
10. Barbosa-Ribeiro M, De-Jesus-Soares A, Zaia AA, et al: *Quantification of lipoteichoic acid contents and cultivable bacteria at the different phases of the endodontic retreatment*, J Endod 2016;42: 552-556.
11. Baumgartner JC. *Microbiologic and pathologic aspects of endodontics*, Curr Opin Dent 1991;1: 737-743.
12. Baumgartner JC. *Microbiologic aspects of endodontic infections*, J Calif Dent Assoc 2004;32: 459-468.
13. Baumgartner JC, Siqueira JF Jr, Xia T, et al: *Geographical differences in bacteria detected in endodontic infections using polymerase chain reaction*, J Endod 2004;30: 141-144.
14. Baumgartner JC, Watkins BJ, Bae KS, et al: *Association of blackpigmented bacteria with endodontic infections*, J Endod 1999;25: 413-415.
15. Baumgartner JC, Xia T. *Antibiotic susceptibility of bacteria associated with endodontic abscesses*, J Endod 2003;29: 44-47.
16. Beighton D, Hardie JM, Whiley RA. *A scheme for the identification of viridans streptococci*, J Med Microbiol 1991;35: 367-372.
17. Beloin C, Valle J, Latour-Lambert P, et al: *Global impact of mature biofilm lifestyle on Escherichia coli K-12 gene expression*, Mol Microbiol 2004;51: 659-674.
18. Bergenholtz G. *Micro-organisms from necrotic pulp of traumatized teeth*, Odontol Revy 1974;25: 347-358.
19. Blome B, Braun A, Sobarzo V, et al: *Molecular identification and quantification of bacteria from endodontic infections using real-time polymerase chain reaction*, Oral Microbiol Immunol 2008;23: 384-390.
20. Bosshard PP, Abels S, Altwegg M, et al: *Comparison of*

Telegram: @dental_k

www.konkur.in

conventional and molecular methods for identification of aerobic catalase-negative gram-positive cocci in the clinical laboratory, J Clin Microbiol 2004;42: 2065-2073.

21. Bosshard PP, Abels S, Zbinden R, et al: *Ribosomal DNA sequencing for identification of aerobic gram-positive rods in the clinical laboratory (an 18-month evaluation)*, J Clin Microbiol 2003;41: 4134-4140.
22. Byström A, Sundqvist G. *The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy*, Int Endod J 1985;18: 35-40.
23. Carr GB, Schwartz RS, Schaudinn C, et al: *Ultrastructural examination of failed molar retreatment with secondary apical periodontitis: an examination of endodontic biofilms in an endodontic retreatment failure*, J Endod 2009;35: 1303-1309.
24. Chavez de Paz L, Svensater G, Dahlen G, et al: *Streptococci from root canals in teeth with apical periodontitis receiving endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 232-241.
25. Chavez de Paz LE, Dahlen G, Molander A, et al: *Bacteria recovered from teeth with apical periodontitis after antimicrobial endodontic treatment*, Int Endod J 2003;36: 500-508.
26. Chavez de Paz LE, Molander A, Dahlen G. *Gram-positive rods prevailing in teeth with apical periodontitis undergoing root canal treatment*, Int Endod J 2004;37: 579-587.
27. Chen V, Chen Y, Li H, et al: *Herpesviruses in abscesses and cellulitis of endodontic origin*, J Endod 2009;35: 182-188.
28. Cheung GS, Ho MW. *Microbial flora of root canal-*

Telegram: @dental_k

www.konkur.in

treated teeth associated with asymptomatic periapical radiolucent lesions, Oral Microbiol Immunol 2001;16: 332-337.

29. Chu FC, Leung WK, Tsang PC, et al: *Identification of cultivable microorganisms from root canals with apical periodontitis following two-visit endodontic treatment with antibiotics/steroid or calcium hydroxide dressings*, J Endod 2006;32: 17-23.
30. Chugal N, Wang JK, Wang R, et al: *Molecular characterization of the microbial flora residing at the apical portion of infected root canals of human teeth*, J Endod 2011;37: 1359-1364.
31. Chugal NM, Clive JM, Spångberg LS. *A prognostic model for assessment of the outcome of endodontic treatment: Effect of biologic and diagnostic variables*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 342-352.
32. Costerton B. *Microbial ecology comes of age and joins the general ecology community*, Proc Natl Acad Sci U S A 2004;101: 16983-16984.
33. Costerton JW. The biofilm primer 2007; Springer-Verlag Berlin, Heidelberg.
34. Costerton JW, Stewart PS, Greenberg EP. *Bacterial biofilms: a common cause of persistent infections*, Science 1999;284: 1318-1322.
35. Cvek M, Cleaton-Jones PE, Austin JC, et al: *Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys*, J Endod 1982;8: 391-397.
36. de Chevigny Dao C, TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study—phase 4: initial treatment*, J Endod 2008;34: 258-2563.
37. EL Ferraz de Sousa, CC, Gomes BP, et al: *Bacteriological study of root canals associated with periapical abscesses*,

Telegram: @dental_k

www.konkur.in

Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 332-339.

38. Dahlen G, Bergenholtz G. *Endotoxic activity in teeth with necrotic pulps*, J Dent Res 1980;59: 1033-1040.
39. Dahlén G, Magnusson BC, Möller A. *Histological and histochemical study of the influence of lipopolysaccharide extracted from Fusobacterium nucleatum on the periapical tissues in the monkey Macaca fascicularis*, Arch Oral Biol 1981;26: 591-598.
40. Delivanis PD, Fan VS. *The localization of blood-borne bacteria in instrumented unfilled and overinstrumented canals*, J Endod 1984;10: 521-524.
41. Dethlefsen L, Eckburg PB, Bik EM, et al: *Assembly of the human intestinal microbiota*, Trends Ecol Evol 2006;21: 517-523.
42. Dewhirst FE, Chen T, Izard J, et al: *The human oral microbiome*, J Bacteriol 2010;192: 5002-5017.
43. Distel JW, Hatton JF, Gillespie MJ. *Biofilm formation in medicated root canals*, J Endod 2002;28: 689-693.
44. Dobell C. Antony van Leeuwenhoek and his “little animals” 1932; Staples Press Limited London.
45. Donlan RM, Costerton JW. *Biofilms: survival mechanisms of clinically relevant microorganisms*, Clin Microbiol Rev 2002;15: 167-193.
46. Drancourt M, Bollet C, Carlioz A, et al: *16S ribosomal DNA sequence analysis of a large collection of environmental and clinical unidentifiable bacterial isolates*, J Clin Microbiol 2000;38: 3623-3630.
47. Eckburg PB, Bik EM, Bernstein CN, et al: *Diversity of the human intestinal microbial flora*, Science 2005;308: 1635-1638.
48. Eckburg PB, Lepp PW, Relman DA. *Archaea and their*

Telegram: @dental_k

www.konkur.in

potential role in human disease, Infect Immun 2003;71: 591-596.

49. Egan MW, Spratt DA, Ng YL, et al: *Prevalence of yeasts in saliva and root canals of teeth associated with apical periodontitis*, Int Endod J 2002;35: 321-329.
50. Engelkirk PG, Duben-Engelkirk J, Dowell VR Jr. Principles and practice of clinical anaerobic bacteriology 1992; Star Publishing Company Belmont, CA.
51. Fabricius L, Dahlén G, Ohman AE, et al: *Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure*, Scand J Dent Res 1982;90: 134-144.
52. Fabricius L, Dahlén G, Sundqvist G, et al: *Influence of residual bacteria on periapical tissue healing after chemomechanical treatment and root filling of experimentally infected monkey teeth*, Eur J Oral Sci 2006;114: 278-285.
53. Farzaneh M, Abitbol S, Friedman S. *Treatment outcome in endodontics: the Toronto study. Phases I and II: Orthograde retreatment*, J Endod 2004;30: 627-633.
54. Ferreira DC, Paiva SS, Carmo FL, et al: *Identification of herpesviruses types 1 to 8 and human papillomavirus in acute apical abscesses*, J Endod 2011;37: 10-16.
55. Foschi F, Cavrini F, Montebugnoli L, et al: *Detection of bacteria in endodontic samples by polymerase chain reaction assays and association with defined clinical signs in Italian patients*, Oral Microbiol Immunol 2005;20: 289-295.
56. Fouad AF, Barry J, Caimano M, et al: *PCR-based identification of bacteria associated with endodontic infections*, J Clin Microbiol 2002;40: 3223-3231.
57. Friedman S. *Expected outcomes in the prevention and*

Telegram: @dental_k

www.konkur.in

treatment of apical periodontitis, Ørstavik D T Pitt Ford Essential endodontology 2008; Blackwell Munksgaard Ltd Oxford, UK 408-469.

58. Garberoglio R, Brännström M. *Scanning electron microscopic investigation of human dentinal tubules*, Arch Oral Biol 1976;21: 355-358.
59. Gatti JJ, Dobeck JM, Smith C, et al: *Bacteria of asymptomatic periradicular endodontic lesions identified by DNA-DNA hybridization*, Endod Dent Traumatol 2000;16: 197-204.
60. George N, Flamiatos E, Kawasaki K, et al: *Oral microbiota species in acute apical endodontic abscesses*, J Oral Microbiol 2016;8: 30989.
61. Gier RE, Mitchell DF. *Anachoretic effect of pulpitis*, J Dent Res 1968;47: 564-570.
62. Glick M, Trope M, Bagasra O, et al: *Human immunodeficiency virus infection of fibroblasts of dental pulp in seropositive patients*, Oral Surg Oral Med Oral Pathol 1991;71: 733-736.
63. Gomes BP, Berber VB, Kokaras AS, et al: *Microbiomes of Endodontic-Periodontal Lesions before and after Chemomechanical Preparation*, J Endod 2015;41: 1975-1984.
64. Gomes BP, Endo MS, Martinho FC. *Comparison of endotoxin levels found in primary and secondary endodontic infections*, J Endod 2012;38: 1082-1086.
65. Gomes BP, Jacinto RC, Montagner F, et al: *Analysis of the antimicrobial susceptibility of anaerobic bacteria isolated from endodontic infections in Brazil during a period of nine years*, J Endod 2011;37: 1058-1062.
66. Gomes BP, Lilley JD, Drucker DB. *Clinical significance of dental root canal microflora*, J Dent 1996;24: 47-55.

Telegram: @dental_k

www.konkur.in

67. Gomes BP, Lilley JD, Drucker DB. *Variations in the susceptibilities of components of the endodontic microflora to biomechanical procedures*, Int Endod J 1996;29: 235-241.
68. Gomes BP, Pinheiro ET, Jacinto RC, et al: *Microbial analysis of canals of root-filled teeth with periapical lesions using polymerase chain reaction*, J Endod 2008;34: 537-540.
69. Green BD, Keller M. *Capturing the uncultivated majority*, Curr Opin Biotechnol 2006;17: 236-240.
70. Griffee MB, Patterson SS, Miller CH, et al: *The relationship of Bacteroides melaninogenicus to symptoms associated with pulpal necrosis*, Oral Surg Oral Med Oral Pathol 1980;50: 457-461.
71. Griffen AL, Beall CJ, Campbell JH, et al: *Distinct and complex bacterial profiles in human periodontitis and health revealed by 16S pyrosequencing*, ISME J 2012;6: 1176-1185.
72. Grossman LI. *Origin of microorganisms in traumatized, pulpless, sound teeth*, J Dent Res 1967;46: 551-553.
73. Haapasalo M, Ørstavik D. *In vitro infection and disinfection of dentinal tubules*, J Dent Res 1987;66: 1375-1379.
74. Haapasalo M, Ranta H, Ranta K.et al: ". *Black-pigmented Bacteroides spp. in human apical periodontitis*, Infect Immun 1986;53: 149-153.
75. Hall-Stoodley L, Costerton JW, Stoodley P. *Bacterial biofilms: from the natural environment to infectious diseases*, Nat Rev Microbiol 2004;2: 95-108.
76. Hall-Stoodley L, Stoodley P. *Evolving concepts in biofilm infections*, Cell Microbiol 2009;11: 1034-1043.
77. Handal T, Caugant DA, Olsen I, et al: *Bacterial diversity*

Telegram: @dental_k

www.konkur.in

in persistent periapical lesions on root-filled teeth, J Oral Microbiol 2009;1: doi:10.3402/jom.v1i0.1946.

78. Happonen RP. *Periapical actinomycosis: a follow-up study of 16 surgically treated cases*, Endod Dent Traumatol 1986;2: 205-209.
79. Hashioka K, Suzuki K, Yoshida T, et al: *Relationship between clinical symptoms and enzyme-producing bacteria isolated from infected root canals*, J Endod 1994;20: 75-77.
80. Henderson B, Poole S, Wilson M. *Bacterial modulins: a novel class of virulence factors which cause host tissue pathology by inducing cytokine synthesis*, Microbiol Rev 1996;60: 316-341.
81. Henriques Tavares LC, de Brito LC, WL, et al: *Microbial ecosystem analysis in root canal infections refractory to endodontic treatment*, J Endod 2016;42: 1239-1245.
82. Higuchi R, Gyllensten U, Persing DH. *Next-generation DNA sequencing and microbiology*, Persing DH Tenover FC Tang YW. Molecular microbiology. Diagnostic, principles and practice 2011; ASM Press Washington, DC 301-312.
83. Hong BY, Lee TK, Lim SM, et al: *Microbial analysis in primary and persistent endodontic infections by using pyrosequencing*, J Endod 2013;39: 1136-1140.
84. Horiba N, Maekawa Y, Abe Y, et al: *Correlations between endotoxin and clinical symptoms or radiolucent areas in infected root canals*, Oral Surg Oral Med Oral Pathol 1991;71: 492-495.
85. Hoshino E, Ando N, Sato M, et al: *Bacterial invasion of nonexposed dental pulp*, Int Endod J 1992;25: 2-5.
86. Hsiao WW, Li KL, Liu Z, et al: *Microbial transformation from normal oral microbiota to acute endodontic*

Telegram: @dental_k

www.konkur.in

infections, BMC Genomics 2012;13: 345.
87. Huston MA. Biological diversity 1994; Cambridge University Press Cambridge, UK.
88. Jacinto RC, Gomes BP, Shah HN, et al: *Quantification of endotoxins in necrotic root canals from symptomatic and asymptomatic teeth*, J Med Microbiol 2005;54: 777-783.
89. Jett BD, Huycke MM, Gilmore MS. *Virulence of enterococci*, Clin Microbiol Rev 1994;7: 462-478.
90. Jung IY, Choi BK, Kum KY, et al: *Molecular epidemiology and association of putative pathogens in root canal infection*, J Endod 2000;26: 599-604.
91. Kakehashi S, Stanley HR, Fitzgerald RJ. *The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral Pathol 1965;20: 340-349.
92. Kaufman B, Spångberg L, Barry J, et al: *Enterococcus spp. In endodontically treated teeth with and without periradicular lesions*, J Endod 2005;31: 851-856.
93. Keijser BJ, Zaura E, Huse SM, et al: *Pyrosequencing analysis of the oral microflora of healthy adults*, J Dent Res 2008;87: 1016-1020.
94. Keren I, Kaldalu N, Spoering A, et al: *Persister cells and tolerance to antimicrobials*, FEMS Microbiol Lett 2004;230: 13-18.
95. Keskin C, Demiryurek EO, Onuk EE. *Pyrosequencing analysis of cryogenically ground samples from primary and secondary/persistent endodontic infections*, J Endod 2017;43: 1309-1316.
96. Khemaleelakul S, Baumgartner JC, Pruksakorn S. *Identification of bacteria in acute endodontic infections and their antimicrobial susceptibility*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 746-755.

Telegram: @dental_k

www.konkur.in

97. Khemaleelakul S, Baumgartner JC, Pruksakom S. *Autoaggregation and coaggregation of bacteria associated with acute endodontic infections*, J Endod 2006;32: 312-318.
98. Kolenbrander PE, Andersen RN, Blehert DS, et al: *Communication among oral bacteria*, Microbiol Mol Biol Rev 2002;66: 486-505.
99. Kumar PS, Griffen AL, Moeschberger ML, et al: *Identification of candidate periodontal pathogens and beneficial species by quantitative 16S clonal analysis*, J Clin Microbiol 2005;43: 3944-3955.
100. Kuriyama T, Karasawa T, Nakagawa K, et al: *Bacteriologic features and antimicrobial susceptibility in isolates from orofacial odontogenic infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 600-608.
101. Lana MA, Ribeiro-Sobrinho AP, Stehling R, et al: *Microorganisms isolated from root canals presenting necrotic pulp and their drug susceptibility in vitro*, Oral Microbiol Immunol 2001;16: 100-105.
102. Langeland K, Rodrigues H, Dowden W. *Periodontal disease, bacteria, and pulpal histopathology*, Oral Surg Oral Med Oral Pathol 1974;37: 257.
103. Lewis MA, MacFarlane TW, McGowan DA. *Quantitative bacteriology of acute dento-alveolar abscesses*, J Med Microbiol 1986;21: 101-104.
104. Li H, Chen V, Chen Y, et al: *Herpesviruses in endodontic pathoses: association of Epstein-Barr virus with irreversible pulpitis and apical periodontitis*, J Endod 2009;35: 23-29.
105. Li L, Hsiao WW, Nandakumar R, et al: *Analyzing endodontic infections by deep coverage pyrosequencing*, J

Telegram: @dental_k

www.konkur.in

Dent Res 2010;89: 980-984.
106. Lin LM, Pascon EA, Skribner J, et al: *Clinical, radiographic, and histologic study of endodontic treatment failures*, Oral Surg Oral Med Oral Pathol 1991;71: 603-611.
107. Lin LM, Ricucci D, Lin J, et al: *Nonsurgical root canal therapy of large cyst-like inflammatory periapical lesions and inflammatory apical cysts*, J Endod 2009;35: 607-615.
108. Lin LM, Skribner JE, Gaengler P. *Factors associated with endodontic treatment failures*, J Endod 1992;18: 625-627.
109. Lleo MM, Bonato B, Tafi MC, et al: *Resuscitation rate in different enterococcal species in the viable but nonculturable state*, J Appl Microbiol 2001;91: 1095-1102.
110. Lleo MM, Bonato B, Tafi MC, et al: *Molecular vs culture methods for the detection of bacterial faecal indicators in groundwater for human use*, Lett Appl Microbiol 2005;40: 289-294.
111. Love RM. *Bacterial penetration of the root canal of intact incisor teeth after a simulated traumatic injury*, Endod Dent Traumatol 1996;12: 289-293.
112. Love RM, Jenkinson HF. *Invasion of dentinal tubules by oral bacteria*, Crit Rev Oral Biol Med 2002;13: 171-183.
113. Machado de Oliveira JC, Siqueira JF, Rôças IN Jr, et al: *Bacterial community profiles of endodontic abscesses from Brazilian and USA subjects as compared by denaturing gradient gel electrophoresis analysis*, Oral Microbiol Immunol 2007;22: 14-18.
114. Mah TF, O'Toole GA. *Mechanisms of biofilm resistance to antimicrobial agents*, Trends Microbiol 2001;9: 34-39.
115. Marsh PD. *Dental plaque as a microbial biofilm*, Caries

Telegram: @dental_k

www.konkur.in

Res 2004;38: 204-211.
116. Matsuo T, Shirakami T, Ozaki K, et al: *An immunohistological study of the localization of bacteria invading root pulpal walls of teeth with periapical lesions*, J Endod 2003;29: 194-200.
117. Michelich V, Pashley DH, Whitford GM. *Dentin permeability: a comparison of functional versus anatomical tubular radii*, J Dent Res 1978;57: 1019-1024.
118. Michelich VJ, Schuster GS, Pashley DH. *Bacterial penetration of human dentin in vitro*, J Dent Res 1980;59: 1398-1403.
119. Miller WD. *An introduction to the study of the bacterio-pathology of the dental pulp*, Dent Cosmos 1894;36: 505-528.
120. Mogensen TH, Paludan SR. *Molecular pathways in virus-induced cytokine production*, Microbiol Mol Biol Rev 2001;65: 131-150.
121. Molander A, Reit C, Dahlen G, et al: *Microbiological status of rootfilled teeth with apical periodontitis*, Int Endod J 1998;31: 1-7.
122. Möller AJR. *Microbial examination of root canals and periapical tissues of human teeth*, Odontol Tidskr 1966;74(Suppl): 1-380.
123. Möller AJR, Fabricius L, Dahlén G, et al: *Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys*, Scand J Dent Res 1981;89: 475-484.
124. Molven O, Olsen I, Kerekes K. *Scanning electron microscopy of bacteria in the apical part of root canals in permanent teeth with periapical lesions*, Endod Dent Traumatol 1991;7: 226-229.
125. Moore WEC, Moore LVH. *The bacteria of periodontal*

Telegram: @dental_k

www.konkur.in

diseases, Periodontol 2000 1994;5: 66-77.
126. Munson MA, Pitt-Ford T, Chong B, et al: *Molecular and cultural analysis of the microflora associated with endodontic infections*, J Dent Res 2002;81: 761-766.
127. Murakami Y, Hanazawa S, Tanaka S, et al: *A possible mechanism of maxillofacial abscess formation: involvement of Porphyromonas endodontalis lipopolysaccharide via the expression of inflammatory cytokines*, Oral Microbiol Immunol 2001;16: 321-325.
128. Nagaoka S, Miyazaki Y, Liu HJ, et al: *Bacterial invasion into dentinal tubules of human vital and nonvital teeth*, J Endod 1995;21: 70-73.
129. Nair PNR. *Non-microbial etiology: foreign body reaction maintaining post-treatment apical periodontitis*, Endod Topics 2003;6: 114-134.
130. Neves MA, Provenzano JC, Rôças IN, et al: *Clinical antibacterial effectiveness of root canal preparation with reciprocating single-instrument or continuously rotating multiinstrument systems*, J Endod 2016;42: 25-29.
131. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health*, Int Endod J 2011;44: 583-609.
132. Noiri Y, Ehara A, Kawahara T, et al: *Participation of bacterial biofilms in refractory and chronic periapical periodontitis*, J Endod 2002;28: 679-683.
133. Okamura K, Maeda M, Nishikawa T, et al: *Dentinal response against carious invasion: localization of antibodies in odontoblastic body and process*, J Dent Res 1980;59: 1368-1373.
134. Okamura K, Tsubakimoto K, Uobe K, et al: *Serum proteins and secretory component in human carious*

Telegram: @dental_k

www.konkur.in

dentin, J Dent Res 1979;58: 1127-1133.

135. Oosthuizen MC, Steyn B, Theron J, et al: *Proteomic analysis reveals differential protein expression by Bacillus cereus during biofilm formation*, Appl Environ Microbiol 2002;68: 2770-2780.
136. Ozok AR, Persoon IF, Huse SM, et al: *Ecology of the microbiome of the infected root canal system: a comparison between apical and coronal root segments*, Int Endod J 2012;45: 530-541.
137. Paiva SS, Siqueira JF Jr, Rôças IN, et al: *Supplementing the antimicrobial effects of chemomechanical debridement with either passive ultrasonic irrigation or a final rinse with chlorhexidine: a clinical study*, J Endod 2012;38: 1202-1206.
138. Paiva SS, Siqueira JF Jr, Rôças IN, et al: *Molecular microbiological evaluation of passive ultrasonic activation as a supplementary disinfecting step: a clinical study*, J Endod 2013;39: 190-194.
139. Parsek MR, Singh PK. *Bacterial biofilms: an emerging link to disease pathogenesis*, Annu Rev Microbiol 2003;57: 677-701.
140. Pashley DH. *Dentin-predentin complex and its permeability: physiologic overview*, J Dent Res Spec No, 1985;64: 613-620.
141. Pashley DH. *Clinical considerations of microleakage*, J Endod 1990;16: 70-77.
142. Pashley DH. *Dynamics of the pulpo-dentin complex*, Crit Rev Oral Biol Med 1996;7: 104-133.
143. Paster BJ, Boches SK, Galvin JL, et al: *Bacterial diversity in human subgingival plaque*, J Bacteriol 2001;183: 3770-3783.
144. Paster BJ, Olsen I, Aas JA, et al: *The breadth of bacterial*

Telegram: @dental_k

www.konkur.in

diversity in the human periodontal pocket and other oral sites, Periodontol 2000 2006;42: 80-87.

145. Peciuliene V, Reynaud AH, Balciuniene I, et al: *Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis*, Int Endod J 2001;34: 429-434.
146. Perez F, Calas P, de Falguerolles A, et al: *Migration of a Streptococcus sanguis strain through the root dentinal tubules*, J Endod 1993;19: 297-301.
147. Peters Buijs LB, van Winkelhoff AJ, JF, et al: *Effects of instrumentation, irrigation and dressing with calcium hydroxide on infection in pulpless teeth with periapical bone lesions*, Int Endod J 2002;35: 13-21.
148. Peters LB, Wesselink PR, Buijs JF, et al: *Viable bacteria in root dentinal tubules of teeth with apical periodontitis*, J Endod 2001;27: 76-81.
149. Petti CA, Polage CR, Schreckenberger P. *The role of 16S rRNA gene sequencing in identification of microorganisms misidentified by conventional methods*, J Clin Microbiol 2005;43: 6123-6125.
150. Pinheiro ET, Gomes BP, Ferraz CC, et al: *Microorganisms from canals of root-filled teeth with periapical lesions*, Int Endod J 2003;36: 1-11.
151. Preza D, Olsen I, Aas JA, et al: *Bacterial profiles of root caries in elderly patients*, J Clin Microbiol 2008;46: 2015-2021.
152. Provenzano JC, Rôças IN, Tavares LF, et al: *Short-chain fatty acids in infected root canals of teeth with apical periodontitis before and after treatment*, J Endod 2015;41: 831-835.
153. Provenzano JC, Siqueira JF Jr, Rôças IN, et al: *Metaproteome analysis of endodontic infections in*

Telegram: @dental_k

www.konkur.in

association with different clinical conditions, PLoS One 2013;8: e76108.

154. Rappe MS, Giovannoni SJ. *The uncultured microbial majority*, Annu Rev Microbiol 2003;57: 369-394.
155. Relman DA. *Microbial genomics and infectious diseases*, N Engl J Med 2011;365: 347-357.
156. Ribeiro AC, Matarazzo F, Faveri M, et al: *Exploring bacterial diversity of endodontic microbiota by cloning and sequencing 16S rRNA*, J Endod 2011;37: 922-926.
157. Ricucci D, Candeiro GT, Bugea C, et al: *Complex apical intraradicular infection and extraradicular mineralized biofilms as the cause of wet canals and treatment failure: report of 2 cases*, J Endod 2016;42: 509-515.
158. Ricucci D, Lin LM, Spångberg LS. *Wound healing of apical tissues after root canal therapy: a long-term clinical, radiographic, and histopathologic observation study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: 609-621.
159. Ricucci D, Loghin S, Gonçalves LS, et al: *Histobacteriologic conditions of the apical root canal system and periapical tissues in teeth associated with sinus tracts*, J Endod 2018;44: 405-413.
160. Ricucci D, Loghin S, Siqueira JF Jr. *Exuberant biofilm infection in a lateral canal as the cause of short-term endodontic treatment failure: report of a case*, J Endod 2013;39: 712-718.
161. Ricucci D, Lopes WSP, Loghin S, et al: *Large bacterial floc causing an independent extraradicular infection and posttreatment apical periodontitis: a case report*, J Endod 2018;44: 1308-1316.
162. Ricucci D, Russo J, Rutberg M, et al: *A prospective cohort study of endodontic treatments of 1,369 root*

Telegram: @dental_k

www.konkur.in

canals: results after 5 years, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 825-842.

163. Ricucci D, Siqueira JF Jr. *Apical actinomycosis as a continuum of intraradicular and extraradicular infection: case report and critical review on its involvement with treatment failure*, J Endod 2008;34: 1124-1129.
164. Ricucci D, Siqueira JF Jr. *Biofilms and apical periodontitis: study of prevalence and association with clinical and histopathologic findings*, J Endod 2010;36: 1277-1288.
165. Ricucci D, Siqueira JF Jr. *Biofilms and apical periodontitis: study of prevalence and association with clinical and histopathologic findings*, J Endod 2010;36: 1277-1288.
166. Ricucci D, Siqueira JF Jr. *Fate of the tissue in lateral canals and apical ramifications in response to pathologic conditions and treatment procedures*, J Endod 2010;36: 1-15.
167. Ricucci D, Siqueira JF Jr, Bate AL, et al: *Histologic investigation of root canal-treated teeth with apical periodontitis: a retrospective study from twenty-four patients*, J Endod 2009;35: 493-502.
168. Ricucci D, Siqueira JF Jr, Lopes WS, et al: *Extraradicular infection as the cause of persistent symptoms: a case series*, J Endod 2015;41: 265-273.
169. Robertson D, Smith AJ. *The microbiology of the acute dental abscess*, J Med Microbiol 2009;58: 155-162.
170. Robinson HBG, Boling LR. *The anachoretic effect in pulpitis. Bacteriologic studies*, J Am Dent Assoc 1941;28: 268-282.
171. Rôças IN, Baumgartner JC, Xia T, et al: *Prevalence of selected bacterial named species and uncultivated*

Telegram: @dental_k

www.konkur.in

phylotypes in endodontic abscesses from two geographic locations, J Endod 2006;32: 1135-1138.

172. Rôças IN, Hülsmann M, Siqueira JF Jr. *Microorganisms in root canal-treated teeth from a German population*, J Endod 2008;34: 926-931.
173. Rôças IN, Jung IY, Lee CY, et al: *Polymerase chain reaction identification of microorganisms in previously root-filled teeth in a South Korean population*, J Endod 2004;30: 504-508.
174. Rôças IN, Provenzano JC, Neves MA, et al: *Disinfecting effects of rotary instrumentation with either 2.5% sodium hypochlorite or 2% chlorhexidine as the main irrigant: a randomized clinical study*, J Endod 2016;42: 943-947.
175. Rôças IN, Siqueira JF Jr. *Detection of novel oral species and phylotypes in symptomatic endodontic infections including abscesses*, FEMS Microbiol Lett 2005;250: 279-285.
176. Rôças IN, Siqueira JF Jr. *Characterization of Dialister species in infected root canals*, J Endod 2006;32: 1057-1061.
177. Rôças IN, Siqueira JF Jr. *Root canal microbiota of teeth with chronic apical periodontitis*, J Clin Microbiol 2008;46: 3599-3606.
178. Rôças IN, Siqueira JF Jr. *Prevalence of new candidate pathogens Prevotella baroniae, Prevotella multisaccharivorax and as-yetuncultivated Bacteroidetes clone X083 in primary endodontic infections*, J Endod 2009;35: 1359-1362.
179. Rôças IN, Siqueira JF Jr. *Identification of bacteria enduring endodontic treatment procedures by a combined reverse transcriptase-polymerase chain reaction and reverse-capture checkerboard approach*, J. Endod

Telegram: @dental_k

www.konkur.in

2010;36: 45-52.
180. Rôças IN, Siqueira JF Jr. *Comparison of the in vivo antimicrobial effectiveness of sodium hypochlorite and chlorhexidine used as root canal irrigants: a molecular microbiology study*, J Endod 2011;37: 143-150.
181. Rôças IN, Siqueira JF Jr. *In vivo antimicrobial effects of endodontic treatment procedures as assessed by molecular microbiologic techniques*, J Endod 2011;37: 304-310.
182. Rôças IN, Siqueira JF Jr. *Characterization of microbiota of root canal-treated teeth with posttreatment disease*, J Clin Microbiol 2012;50: 1721-1724.
183. Rôças IN, Siqueira JF Jr. *Frequency and levels of candidate endodontic pathogens in acute apical abscesses as compared to asymptomatic apical periodontitis*, PLoS One 2018;13: e0190469.
184. Rôças IN, Siqueira JF Jr, Aboim MC, et al: *Denaturing gradient gel electrophoresis analysis of bacterial communities associated with failed endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 741-749.
185. Rôças IN, Siqueira JF Jr, Andrade AFB, et al: *Identification of selected putative oral pathogens in primary root canal infections associated with symptoms*, Anaerobe 2002;8: 200-208.
186. Rôças IN, Siqueira JF Jr, Debelian GJ. *Analysis of symptomatic and asymptomatic primary root canal infections in adult Norwegian patients*, J Endod 2011;37: 1206-1212.
187. Rôças IN, Siqueira JF Jr, Santos KR. *Association of Enterococcus faecalis with different forms of periradicular diseases*, J Endod 2004;30: 315-320.
188. Rolph HJ, Lennon A, Riggio MP, et al: *Molecular*

Telegram: @dental_k

www.konkur.in

identification of microorganisms from endodontic infections, J Clin Microbiol 2001;39: 3282-3289.

189. Saber MH, Schwarzberg K, Alonaizan FA, et al: *Bacterial flora of dental periradicular lesions analyzed by the 454-pyrosequencing technology*, J Endod 2012;38: 1484-1488.
190. Sabeti M, Simon JH, Nowzari H, et al: *Cytomegalovirus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns*, J Endod 2003;29: 321-323.
191. Sabeti M, Simon JH, Slots J. *Cytomegalovirus and Epstein-Barr virus are associated with symptomatic periapical pathosis*, Oral Microbiol Immunol 2003;18: 327-328.
192. Sabeti M, Slots J. *Herpesviral-bacterial coinfection in periapical pathosis*, J Endod 2004;30: 69-72.
193. Sabeti M, Valles Y, Nowzari H, et al: *Cytomegalovirus and Epstein-Barr virus DNA transcription in endodontic symptomatic lesions*, Oral Microbiol Immunol 2003;18: 104-108.
194. Saboia-Dantas Coutrin de Toledo CJ, LF, Sampaio-Filho HR, et al: *Herpesviruses in asymptomatic apical periodontitis lesions: an immunohistochemical approach*, Oral Microbiol Immunol 2007;22: 320-325.
195. Saito de Toledo Leonardo D, R, Rodrigues JLM, et al: *Identification of bacteria in endodontic infections by sequence analysis of 16S rDNA clone libraries*, J Med Microbiol 2006;55: 101-107.
196. Sakamoto M, Rôças IN, Siqueira JF Jr, et al: *Molecular analysis of bacteria in asymptomatic and symptomatic endodontic infections*, Oral Microbiol Immunol 2006;21: 112-122.

Telegram: @dental_k

www.konkur.in

197. Sakamoto M, Siqueira JF Jr, Rôças IN, et al: *Bacterial reduction and persistence after endodontic treatment procedures*, Oral Microbiol Immunol 2007;22: 19-23.
198. Sakamoto M, Siqueira JF Jr, Rôças IN, et al: *Molecular analysis of the root canal microbiota associated with endodontic treatment failures*, Oral Microbiol Immunol 2008;23: 275-281.
199. Sakamoto M, Siqueira JF Jr, Rôças IN, et al: *Diversity of spirochetes in endodontic infections*, J Clin Microbiol 2009;47: 1352-1357.
200. Santos AL, Siqueira JF Jr, Rôças IN, et al: *Comparing the bacterial diversity of acute and chronic dental root canal infections*, PLoS One 2011;6: e28088.
201. Sasaki H, Stashenko P. *Interrelationship of the pulp and apical periodontitis*, Hargreaves KM Goodis HE Tay FR Seltzer and Bender's dental pulp ed 2 2012; Quintessence Publishing Chicago 277-299.
202. Sassone LM, Fidel RA, Faveri M, et al: *A microbiological profile of symptomatic teeth with primary endodontic infections*, J Endod 2008;34: 541-545.
203. Sauer K, Camper AK, Ehrlich GD, et al: *Pseudomonas aeruginosa displays multiple phenotypes during development as a biofilm*, J Bacteriol 2002;184: 1140-1154.
204. Schaudinn C, Carr G, Gorur A, et al: *Imaging of endodontic biofilms by combined microscopy (FISH/cLSM - SEM)*, J Microsc 2009;235: 124-127.
205. Schein B, Schilder H. *Endotoxin content in endodontically involved teeth*, J Endod 1975;1: 19-21.
206. Schonfeld SE, Greening AB, Glick DH, et al: *Endotoxin activity in periapical lesions*, Oral Surg Oral Med Oral Pathol 1982;53: 82-87.

Telegram: @dental_k

www.konkur.in

207. Sedgley C, Nagel A, Dahlen G, et al: *Real-time quantitative polymerase chain reaction and culture analyses of Enterococcus faecalis in root canals*, J Endod 2006;32: 173-177.
208. Sedgley CM, Lennan SL, Appelbe OK. *Survival of Enterococcus faecalis in root canals ex vivo*, Int Endod J 2005;38: 735-742.
209. Seltzer S, Farber PA. *Microbiologic factors in endodontology*, Oral Surg Oral Med Oral Pathol 1994;78: 634-645.
210. Sen BH, Piskin B, Demirci T. *Observation of bacteria and fungi in infected root canals and dentinal tubules by SEM*, Endod Dent Traumatol 1995;11: 6-9.
211. Sen BH, Safavi KE, Spångberg LS. *Colonization of Candida albicans on cleaned human dental hard tissues*, Arch Oral Biol 1997;42: 513-520.
212. Sen BH, Safavi KE, Spångberg LS. *Growth patterns of Candida albicans in relation to radicular dentin*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84: 68-73.
213. Shin JM, Luo T, Lee KH, et al: *Deciphering Endodontic Microbial Communities by Next-generation Sequencing*, J Endod 2018;44: 1080-1087.
214. Siqueira JF Jr. *Microbial causes of endodontic flare-ups*, Int Endod J 2003;36: 453-463.
215. Siqueira JF Jr. *Periapical actinomycosis and infection with Propionibacterium propionicum*, Endod Topics 2003;6: 78-95.
216. Siqueira JF Jr. *Reaction of periradicular tissues to root canal treatment: benefits and drawbacks*, Endod Topics 2005;10: 123-147.
217. Siqueira JF Jr. *Microbiology of apical periodontitis*,

Telegram: @dental_k

www.konkur.in

Ørstavik D T Pitt Ford Essential endodontology ed 2 2008; Blackwell Munksgaard Ltd Oxford, UK 135-196.

218. Siqueira JF Jr. Treatment of endodontic infections 2011; Quintessence Publishing London 43-93.
219. Siqueira JF Jr, Alves FR, Rôças IN. *Pyrosequencing analysis of the apical root canal microbiota*, J Endod 2011;37: 1499-1503.
220. Siqueira JF Jr, Antunes HS, Rôças IN, et al: *Microbiome in the apical root canal system of teeth with posttreatment apical periodontitis*, PLoS One 2016;11: e0162887.
221. Siqueira JF Jr, Barnett F. *Interappointment pain: mechanisms, diagnosis, and treatment*, Endod Topics 2004;7: 93-109.
222. Siqueira JF Jr, de Uzeda M. *Disinfection by calcium hydroxide pastes of dentinal tubules infected with two obligate and one facultative anaerobic bacteria*, J Endod 1996;22: 674-676.
223. Siqueira Fonseca JF Jr, de Uzeda M, ME. *A scanning electron microscopic evaluation of in vitro dentinal tubules penetration by selected anaerobic bacteria*, J Endod 1996;22: 308-310.
224. Siqueira JF Jr, Fouad AF, Rôças IN. *Pyrosequencing as a tool for better understanding of human microbiomes*, J Oral Microbiol 2012;4: doi:10.3402/jom.v4i0.10743.
225. Siqueira JF Jr, Guimarães-Pinto T, Rôças IN. *Effects of chemomechanical preparation with 2.5% sodium hypochlorite and intracanal medication with calcium hydroxide on cultivable bacteria in infected root canals*, J Endod 2007;33: 800-805.
226. Siqueira JF Jr, Jung IY, Rôças IN, et al: *Differences in prevalence of selected bacterial species in primary endodontic infections from two distinct geographic*

Telegram: @dental_k

www.konkur.in

locations, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 641-647.

227. Siqueira JF Jr, Lima KC. *Staphylococcus epidermidis and Staphylococcus xylosus in a secondary root canal infection with persistent symptoms: a case report*, Aust Endod J 2002;28: 61-63.
228. Siqueira JF Jr, Magalhães KM, Rôças IN. *Bacterial reduction in infected root canals treated with 2.5% NaOCl as an irrigant and calcium hydroxide/camphorated paramonochlorophenol paste as an intracanal dressing*, J Endod 2007;33: 667-672.
229. Siqueira JF Jr, Paiva SS, Rôças IN. *Reduction in the cultivable bacterial populations in infected root canals by a chlorhexidinebased antimicrobial protocol*, J Endod 2007;33: 541-547.
230. Siqueira JF Jr, Rôças IN. *Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 85-94.
231. Siqueira JF Jr, Rôças IN. *Exploiting molecular methods to explore endodontic infections: Part 1-current molecular technologies for microbiological diagnosis*, J Endod 2005;31: 411-423.
232. Siqueira JF Jr, Rôças IN. *Exploiting molecular methods to explore endodontic infections: Part 2-redefining the endodontic microbiota*, J Endod 2005;31: 488-498.
233. Siqueira JF Jr, Rôças IN. *Uncultivated phylotypes and newly named species associated with primary and persistent endodontic infections*, J Clin Microbiol 2005;43: 3314-3319.
234. Siqueira JF Jr, Rôças IN. *Bacterial pathogenesis and mediators in apical periodontitis*, Braz Dent J 2007;18:

Telegram: @dental_k

www.konkur.in

267-280.
235. Siqueira JF Jr, Rôças IN. *Clinical implications and microbiology of bacterial persistence after treatment procedures*, J Endod 2008;34: 1291-1301.e3.
236. Siqueira JF Jr, Rôças IN. *Diversity of endodontic microbiota revisited*, J Dent Res 2009;88: 969-981.
237. Siqueira JF Jr, Rôças IN. *The microbiota of acute apical abscesses*, J Dent Res 2009;88: 61-65.
238. Siqueira JF Jr, Rôças IN. *As-yet-uncultivated oral bacteria: breadth and association with oral and extra-oral diseases*, J Oral Microbiol 2013;5: doi:10.3402/jom.v5i0.21077.
239. Siqueira JF Jr, Rôças IN. *Microbiology and treatment of acute apical abscesses*, Clin Microbiol Rev 2013;26: 255-273.
240. Siqueira JF Jr, Rôças IN. *Molecular analysis of endodontic infections*, Fouad AF Endodontic microbiology 2017; John Wiley & Sons Hoboken, NJ 81-128.
241. Siqueira JF Jr, Rôças IN, Alves FR, et al: *Bacteria in the apical root canal of teeth with primary apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107: 721-726.
242. Siqueira JF Jr, Rôças IN, Baumgartner JC, et al: *Searching for Archaea in infections of endodontic origin*, J Endod 2005;31: 719-722.
243. Siqueira JF Jr, Rôças IN, Cunha CD, et al: *Novel bacterial phylotypes in endodontic infections*, J Dent Res 2005;84: 565-569.
244. Siqueira JF Jr, Rôças IN, Debelian GJ, et al: *Profiling of root canal bacterial communities associated with chronic apical periodontitis from Brazilian and Norwegian subjects*, J Endod 2008;34: 1457-1461.

Telegram: @dental_k

www.konkur.in

245. Siqueira JF Jr, Rôças IN, Lopes HP. *Patterns of microbial colonization in primary root canal infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 174-178.
246. Siqueira JF Jr, Rôças IN, Lopes HP, et al: *Fungal infection of the radicular dentin*, J Endod 2002;28: 770-773.
247. Siqueira JF Jr, Rôças IN, Marceliano-Alves MF, et al: *Unprepared root canal surface areas: causes, clinical implications, and therapeutic strategies*, Braz Oral Res 2018;32: e65.
248. Siqueira JF Jr, Rôças IN, Moraes SR, et al: *Direct amplification of rRNA gene sequences for identification of selected oral pathogens in root canal infections*, Int Endod J 2002;35: 345-351.
249. Siqueira JF Jr, Rôças IN, Paiva SS, et al: *Bacteriologic investigation of the effects of sodium hypochlorite and chlorhexidine during the endodontic treatment of teeth with apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 122-130.
250. Siqueira JF Jr, Rôças IN, Paiva SSM, et al: *Cultivable bacteria in infected root canals as identified by 16S rRNA gene sequencing*, Oral Microbiol Immunol 2007;22: 266-271.
251. Siqueira JF Jr, Rôças IN, Rosado AS. *Investigation of bacterial communities associated with asymptomatic and symptomatic endodontic infections by denaturing gradient gel electrophoresis fingerprinting approach*, Oral Microbiol Immunol 2004;19: 363-370.
252. Siqueira JF Jr, Rôças IN, Souto R, et al: *Checkerboard DNA-DNA hybridization analysis of endodontic infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod

Telegram: @dental_k

www.konkur.in

2000;89: 744-748.
253. Siqueira JF Jr, Rôças IN, Souto R, et al: *Microbiological evaluation of acute periradicular abscesses by DNA-DNA hybridization*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 451-457.
254. Siqueira JF Jr, Sen BH. *Fungi in endodontic infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 632-641.
255. Siqueira JF, Rôças IN. *Present status and future directions in endodontic microbiology*, Endod Topics 2014;30: 3-22.
256. Siqueira JF, Rôças IN, Ricucci D. *Biofilms in endodontic infection*, Endod. Topics 2010;22: 33-49.
257. Siren EK, Haapasalo MP, Ranta K, et al: *Microbiological findings and clinical treatment procedures in endodontic cases selected for microbiological investigation*, Int Endod J 1997;30: 91-95.
258. Sjögren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int Endod J 1997;30: 297-306.
259. Sjögren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498-504.
260. Slots J. *Rapid identification of important periodontal microorganisms by cultivation*, Oral Microbiol Immunol 1986;1: 48-57.
261. Slots J, Sabeti M, Simon JH. *Herpesviruses in periapical pathosis: an etiopathogenic relationship?*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 327-331.
262. Socransky SS, Gibbons RJ, Dale AC, et al: *The microbiota of the gingival crevice in man. 1. Total*

Telegram: @dental_k

www.konkur.in

microscopic and viable counts and counts of specific organisms, Arch Oral Biol 1963;8: 275-280.

263. Socransky SS, Haffajee AD. *Dental biofilms: difficult therapeutic targets*, Periodontol 2000 2002;28: 12-55.
264. Song Y, Liu C, McTeague M, et al: *16S ribosomal DNA sequence-based analysis of clinically significant gram-positive anaerobic cocci*, J Clin Microbiol 2003;41: 1363-1369.
265. Stashenko P, Wang CY, Riley E, et al: *Reduction of infection-stimulated periapical bone resorption by the biological response modifier PGG glucan*, J Dent Res 1995;74: 323-330.
266. Stewart PS, Costerton JW. *Antibiotic resistance of bacteria in biofilms*, Lancet 2001;358: 135-138.
267. Stoodley P, Sauer K, Davies DG, et al: *Biofilms as complex differentiated communities*, Annu Rev Microbiol 2002;56: 187-209.
268. Suau A, Bonnet R, Sutren M, et al: *Direct analysis of genes encoding 16S rRNA from complex communities reveals many novel molecular species within the human gut*, Appl Environ Microbiol 1999;65: 4799-4807.
269. Subramanian K, Mickel AK. *Molecular analysis of persistent periradicular lesions and root ends reveals a diverse microbial profile*, J Endod 2009;35: 950-957.
270. Sunde PT, Olsen I, Debelian GJ, et al: *Microbiota of periapical lesions refractory to endodontic therapy*, J Endod 2002;28: 304-310.
271. Sunde PT, Olsen I, Gobel UB, et al: *Fluorescence in situ hybridization (FISH) for direct visualization of bacteria in periapical lesions of asymptomatic root-filled teeth*, Microbiology 2003;149: 1095-1102.
272. Sunde PT, Tronstad L, Eribe ER, et al: *Assessment of*

Telegram: @dental_k

www.konkur.in

periradicular microbiota by DNA-DNA hybridization, Endod Dent Traumatol 2000;16: 191-196.

273. Sundqvist G. *Bacteriological studies of necrotic dental pulps* [Odontological Dissertation no.7] 1976; University of Umea Umea, Sweden.
274. Sundqvist G. *Endodontic microbiology*, Spangberg LSW Experimental endodontics 1990; CRC Press Boca Raton 131-153.
275. Sundqvist G. *Associations between microbial species in dental root canal infections*, Oral Microbiol Immunol 1992;7: 257-262.
276. Sundqvist G. *Taxonomy, ecology, and pathogenicity of the root canal flora*, Oral Surg Oral Med Oral Pathol 1994;78: 522-530.
277. Sundqvist G, Figdor D, Persson S, et al: *Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 86-93.
278. ter Steeg PF, van der Hoeven JS. *Development of periodontal microflora on human serum*, Microb Ecol Health Dis 1989;2: 1-10.
279. Tang G, Samaranayake LP, Yip HK. *Molecular evaluation of residual endodontic microorganisms after instrumentation, irrigation and medication with either calcium hydroxide or Septomixine*, Oral Dis 2004;10: 389-397.
280. Tang YW, Ellis NM, Hopkins MK, et al: *Comparison of phenotypic and genotypic techniques for identification of unusual aerobic pathogenic gram-negative bacilli*, J Clin Microbiol 1998;36: 3674-3679.
281. Tani-Ishii N, Wang CY, Tanner A, et al: *Changes in root canal microbiota during the development of rat periapical*

Telegram: @dental_k

www.konkur.in

lesions, Oral Microbiol Immunol 1994;9: 129-135.

282. Tanner A, Lai CH, Maiden M. *Characteristics of oral gramnegative species*, Slots J Taubman MA Contemporary oral microbiology and immunology 1992; Mosby St Louis 299-341.
283. Tronstad L, Barnett F, Cervone F. *Periapical bacterial plaque in teeth refractory to endodontic treatment*, Endod Dent Traumatol 1990;6: 73-77.
284. Tronstad L, Barnett F, Riso K, et al: *Extraradicular endodontic infections*, Endod Dent Traumatol 1987;3: 86-90.
285. Trowbridge HO, Emling RC. Inflammation. A review of the process ed 5 1997; Quintessence Chicago.
286. Tzanetakis GN, Azcarate-Peril MA, Zachaki S, et al: *Comparison of bacterial community composition of primary and persistent endodontic infections using pyrosequencing*, J Endod 2015;41: 1226-1233.
287. van Amersfoort van Berkel ES, TJC, Kuiper J. *Receptors, mediators, and mechanisms involved in bacterial sepsis and septic shock*, Clin Microbiol Rev 2003;16: 379-414.
288. van Winkelhoff Carlee AJ, AW, de Graaff J. *Bacteroides endodontalis and others black-pigmented Bacteroides species in odontogenic abscesses*, Infect Immun 1985;49: 494-498.
289. Vengerfeldt V, Spilka K, Saag M, et al: *Highly diverse microbiota in dental root canals in cases of apical periodontitis (data of illumina sequencing)*, J Endod 2014;40: 1778-1783.
290. Vera J, Siqueira JF Jr, Ricucci D, et al: *One- versus two-visit endodontic treatment of teeth with apical periodontitis: a histobacteriologic study*, J Endod 2012;38: 1040-1052.

Telegram: @dental_k

www.konkur.in

291. Vianna ME, Conrads G, Gomes BPFA, et al: *Identification and quantification of archaea involved in primary endodontic infections*, J Clin Microbiol 2006;44: 1274-1282.
292. Vianna ME, Horz HP, Gomes BP, et al: *In vivo evaluation of microbial reduction after chemo-mechanical preparation of human root canals containing necrotic pulp tissue*, Int Endod J 2006;39: 484-492.
293. Vickerman MM, Brossard KA, Funk DB, et al: *Phylogenetic analysis of bacterial and archaeal species in symptomatic and asymptomatic endodontic infections*, J Med Microbiol 2007;56: 110-118.
294. Voelkerding KV, Dames SA, Durtschi JD. *Next-generation sequencing: from basic research to diagnostics*, Clin Chem 2009;55: 641-658.
295. Wadachi R, Hargreaves KM. *Trigeminal nociceptors express TLR-4 and CD14: a mechanism for pain due to infection*, J Dent Res 2006;85: 49-53.
296. Waltimo T, Trope M, Haapasalo M, et al: *Clinical efficacy of treatment procedures in endodontic infection control and one year follow-up of periapical healing*, J Endod 2005;31: 863-866.
297. Waltimo T, Trope M, Haapasalo M, et al: *Clinical efficacy of treatment procedures in endodontic infection control and one year follow-up of periapical healing*, J Endod 2005;31: 863-866.
298. Waltimo TM, Ørstavik D, Siren EK, et al: *In vitro susceptibility of Candida albicans to four disinfectants and their combinations*, Int Endod J 1999;32: 421-429.
299. Waltimo TM, Ørstavik D, Siren EK, et al: *In vitro yeast infection of human dentin*, J Endod 2000;26: 207-209.
300. Waltimo TM, Siren EK, Ørstavik D, et al: *Susceptibility*

Telegram: @dental_k

www.konkur.in

of oral Candida species to calcium hydroxide in vitro, Int Endod J 1999;32: 94-98.

301. Wara-Aswapati N, Boch JA, Auron PE. *Activation of interleukin 1beta gene transcription by human cytomegalovirus: molecular mechanisms and relevance to periodontitis*, Oral Microbiol Immunol 2003;18: 67-71.
302. Ward DM, Weller R, Bateson MM. *16S rRNA sequences reveal numerous uncultured microorganisms in a natural community*, Nature 1990;345: 63-65.
303. Wattal C, Oberoi JK, Goel N, et al: *Matrixassisted laser desorption ionization time of flight mass spectrometry (MALDI-TOF MS) for rapid identification of micro-organisms in the routine clinical microbiology laboratory*, Eur J Clin Microbiol Infect Dis 2017;36: 807-812.
304. Wayman BE, Murata SM, Almeida RJ, et al: *A bacteriological and histological evaluation of 58 periapical lesions*, J Endod 1992;18: 152-155.
305. Williams BL, McCann GF, Schoenknecht FD. *Bacteriology of dental abscesses of endodontic origin*, J Clin Microbiol 1983;18: 770-774.
306. Wittgow WC Jr, Sabiston CB Jr. *Microorganisms from pulpal chambers of intact teeth with necrotic pulps*, J Endod 1975;1: 168-171.
307. Yamasaki M, Kumazawa M, Kohsaka T, et al: *Pulpal and periapical tissue reactions after experimental pulpal exposure in rats*, J Endod 1994;20: 13-17.
308. Yoshida M, Fukushima H, Yamamoto K, et al: *Correlation between clinical symptoms and microorganisms isolated from root canals of teeth with periapical pathosis*, J Endod 1987;13: 24-28.
309. Zandi H, Kristoffersen AK, Ørstavik D, et al: *Microbial analysis of endodontic infections in root-filled teeth with*

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apical periodontitis before and after irrigation using pyrosequencing, J Endod 2018;44: 372-378.

310. Zandi H, Rodrigues RC, Kristoffersen AK, et al: *Antibacterial effectiveness of 2 root canal irrigants in root-filled teeth with infection: a randomized clinical trial*, J Endod 2016;42: 1307-1313.
311. Zehnder M, Guggenheim B. *The mysterious appearance of enterococci in filled root canals*, Int Endod J 2009;42: 277-287.
312. Zoletti GO, Siqueira JF Jr, Santos KR. *Identification of Enterococcus faecalis in root-filled teeth with or without periradicular lesions by culture-dependent and -independent approaches*, J Endod 2006;32: 722-726.

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16: Pathobiology of apical periodontitis

Louis M. Lin, George T.-J. Huang

CHAPTER OUTLINE

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Periradicular tissues consist of cementum, periodontal ligament, and alveolar bone. *Cementum* is a mineralized, avascular connective tissue and consists of three different types. Acellular afibrillar cementum covers the teeth at and along the cementoenamel junction. Acellular extrinsic fiber cementum is confined to the coronal half of the root. Cellular intrinsic fiber cementum is present on the apical half of the root where no acellular extrinsic fiber cementum has been laid down.[198] Many growth factors, such as insulin-like growth factor-1 (IGF-1), fibroblast growth factors (FGFs), epidermal growth factor (EGF), bone morphogenetic proteins (BMPs), transforming growth factor-β (TGF-β), and platelet-derived growth factor (PDGF), are contained

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in the cementum matrix.[50,91,170] These growth factors may be released under certain conditions, as they have been shown to be associated with cementoblast proliferation, migration, and differentiation during cementum wound healing.[91]

The *periodontal ligament* is a soft, specialized connective tissue that connects the cementum to the alveolar bone. Periodontal ligament contains heterogeneous cell populations and extracellular matrix (ECM).[149,198] The cells of the periodontal ligament include osteoblasts, fibroblasts, epithelial cell rests of Malassez (ERM), macrophages, cementoblasts, and undifferentiated mesenchymal cells (stem cells).[198] Fibroblasts, osteoblasts, and epithelial cells are differentiated cells that have retained the ability to undergo limited cell divisions and proliferation upon stimulation by appropriate signals. Multipotent mesenchymal stem cells of the periodontal ligament are capable of differentiating into cementoblast-like cells and periodontal ligament cells as well as osteoblasts.[115,188,243] The ECM of the periodontal ligament consists of collagen fibers, fibronectin, elastin, other noncollagenous proteins, and proteoglycans. The ECM serves as stratum for cell adhesion and promotes cell spreading and cytoskeletal organization. The collagen fibers (Sharpey fiber) of the periodontal ligament connect the tooth with the alveolar bone. The periodontal ligament is highly vascularized and innervated. The tissue apical to the dentinocemental junction should be considered as part of the periodontal ligament because cementum is not a normal component of the pulp tissue.

ERM, the remnants of the Hertwig epithelial root sheath that disintegrates after tooth development, are present in the periodontal ligament near the root surface in all teeth after root formation. [217,218] They are nests of epithelial cells connected as a network and surrounded by a basal lamina.[198,240] ERM are quiescent in normal periodontal ligament[198] but can be stimulated to proliferate in apical periodontitis.[163] They are believed to be the cellular source that when properly stimulated can form radicular cysts in certain apical periodontitis lesions.[163,206]

Alveolar bone or *alveolar process* is that part of bone of the jaws housing the sockets for the teeth. It consists of outer cortical plate, a central spongy or cancellous bone, and bone lining the sockets.[198] Bone matrix contains growth factors, IGFs, TGF-β, BMPs, FGF, and PDGF.[43,261] These growth factors are

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essential for osteoblast progenitor cell proliferation, migration, and differentiation during bone wound healing.[166]

The response of the periradicular tissues to various injuries is similar to that of other connective tissues elsewhere in the body. The response is manifested as an inflammatory reaction regulated by both innate and adaptive immune mechanisms. Although microbial infection of the pulp in the root canals is the primary cause of apical periodontitis,[122,184,190] the pathologic changes of the periapical tissues in apical periodontitis are usually not directly caused by microbes themselves, but rather by their toxins, noxious metabolic by-products, and disintegrated pulp tissue in the root canal system. These irritants are capable of inducing both innate and adaptive immune responses. The subsequent immunoinflammatory responses are diverse and can involve changes in microvasculature, transmigration of blood-borne cells and plasma proteins out of the blood circulation into the tissue space, and activation of sensory nerves. In addition, endothelial cells, mast cells, platelets, fibroblasts, neutrophils, macrophages, dendritic cells, innate and adaptive immune cells, immunoglobulins, inflammatory mediators, proinflammatory cytokines, chemokines, and neuropeptides are also involved in the inflammatory response. Apical periodontitis can be protective or destructive, depending on the dynamic interaction between microbial insult and the host's defenses in the periapical tissues.[176] Unfortunately, the bacterial biofilm formed in the root canal system with necrotic pulp is protected from host's defenses and antibiotic therapy because of a lack of blood circulation in the root canal system. Consequently, any attempt of wounded periradicular tissues to repair/regenerate is futile, because bacterial toxins and noxious metabolic by-products in the root canal system continuously egress into the periapical area and irritate the periapical tissues. Emerging lines of evidence suggest that under most conditions, bacterial biofilms in the complex root canal system can be greatly reduced, but not eliminated, by conventional endodontic procedures such as mechanical instrumentation, antiseptic irrigation, and intracanal medication. If microbes in the root canal system are effectively eliminated or entombed within the root canal by filling material, and the root canal system is adequately sealed and protected from coronal microleakage, then periradicular tissues have the ability to restore their original structures by means of a repair/regeneration

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process. Nevertheless, the presence of posttreatment apical periodontitis may be due to persistent microbial biofilms in the root canal,[190,224] and this recognition has spurred considerable research into treating biofilms (see Chapters 14 and 15).

Apical periodontitis

Prevalence

Epidemiologic study of apical periodontitis documents that the prevalence of apical periodontitis varies among patients aged 20 to 30 (33% prevalence of apical periodontitis), 30 to 40 (40%), 40 to 50 (48%), 50 to 60 (57%), and older than 60 years of age (62%).[79,209] Most studies on the prevalence of apical periodontitis are from European and Scandinavian countries.[73,120,247] According to a survey by the American Dental Association in 2005–06, an estimated 15.1 million root canal treatments were performed annually in the United States alone.[10] Apical periodontitis is a prevalent health problem.[74]

Etiology

The etiology, pathogenesis, and histopathology of apical periodontitis are similar to that of marginal periodontitis (see also Chapter 25). Both diseases are caused by bacterial infection and involve pathologic changes of alveolar bone, periodontal ligament, and cementum. Marginal periodontitis affects coronal periodontal tissues, whereas apical periodontitis affects apical periodontal tissues. Bone loss is one of the characteristic features in both diseases: crest bone is lost in marginal periodontitis, and apical bone undergoes resorption in apical periodontitis.

Apical periodontitis can be caused by both exogenous and endogenous factors. Exogenous factors include microbes and their toxins and noxious metabolic by-products, chemical agents, mechanical irritation, foreign bodies, and trauma. Endogenous factors include the host's metabolic products, such as urate and cholesterol crystals,[194] as well as cytokines or other inflammatory mediators.[263] These irritants can activate nonantigenic pathway or antigenic pathway to induce innate and adaptive immunoinflammatory

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responses, respectively.

Infection of the pulp tissue caused by caries or other pathways is the primary cause of apical periodontitis.[122,190,224] The classic study by Kakehashi and colleagues[122] demonstrated that pulp necrosis and periradicular inflammation developed in conventional rats when the pulps of teeth were surgically exposed to oral microorganisms. However, in germ-free laboratory rats, no pulp necrosis and periradicular inflammation occurred even when the pulps of teeth were surgically exposed to the oral environment and packed with sterile food debris. A similar response occurs in humans. Using bacterial culturing, it has been demonstrated that traumatized human teeth with intact crowns and necrotic pulps *without bacterial contamination* did not show radiographic evidence of periapical bone destruction. In contrast, if bacteria were isolated from traumatized teeth with intact crowns and necrotic pulps, then radiographic evidence of periradicular bone destruction was observed.[272] Similar important findings have been replicated in nonhuman primate experiments. When the pulps of intact vital teeth were intentionally devitalized under aseptic conditions and left in the root canals with bacteria-tight, sealed coronal restoration for 6 months to 1 year, no periradicular inflammatory reaction was observed.[161,184] Taken together, there is considerable evidence that bacteria constitute a major etiologic factor in the development of apical periodontitis.

Bacterial toxins (e.g., lipopolysaccharide [LPS], lipoteichoic acid [LTA]) and noxious metabolic by-products that egress from the root canal system into the periapical tissues are capable of inducing a periapical immunoinflammatory reaction.[55,57,67,236,322] These irritants can activate the innate immune system via receptors that recognize the stereotypic pathogen-associated molecular patterns (PAMPs) that are found in the structure of these toxins. Different classes of microbes express different molecular patterns that are recognized by different pattern recognition receptors (PRRs) or Toll-like receptors (TLRs) on host cells, such as phagocytes, dendritic cells, and B lymphocytes.[1,177,178] PRRs or TLRs are encoded in the germline. In mammalian species, there are at least 10 TLRs, and each appears to have a distinct function in innate immune recognition.[178] For example, LPS can stimulate sensory nerve fibers to release calcitonin gene–related peptide (CGRP) and substance P (SP)[64,110] to cause vasodilation and increased

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vascular permeability. LPS and lipoproteins can also activate TLRs on dendritic cells to stimulate T-lymphocyte differentiation.[5] Certain subtypes of TLRs recognize the common shared structural features of various toxins (i.e., PAMPS). Because the TLRs are synthesized before an infection, they are classified as part of the innate immune system.

Apical periodontitis can be caused either by entry into the periapical tissues of bacterial toxins, enzymes, and noxious metabolic by-products or by direct invasion of the periapical tissues by microbes originating from the root canal system. It is important to differentiate between apical inflammation and apical infection. *Apical inflammation* is the periapical tissue reaction to irritants emerging from the root canal system that manifests as vasodilation, increased vascular permeability, and exudation. In contrast, *apical infection* is due to the physical presence of pathogenic microorganisms in the periapical tissues that subsequently produce tissue damage. There can be infection without inflammation, for instance, in a severely immunocompromised patient. There can also be inflammation without infection, such as in a myocardial infarct, cerebral infarct, and physical or chemical injury.[171] In diseases caused by infection, bacteria are usually present in the involved tissues or organs,[305] such as acute necrotizing gingivitis, marginal periodontitis, actinomycosis, tuberculosis, and bacterial bronchitis. Although apical periodontitis is primarily an infectious disease, bacteria are usually not present in the periapical tissues but in the root canal system,[145,190,307] except in certain types of apical periodontitis associated with abscess formation,[207,302,314] or with a draining sinus tract,[93,207,312] or extraradicular endodontic infection.[271,296] One major current hypothesis is that apical periodontitis is triggered by entry into the periapical tissues of bacterial toxins, enzymes, and noxious metabolic by-products.[280] The mere presence of bacterial contamination in some apical periodontitis lesions does not necessarily denote a periradicular infection. Periapical infection is related to both virulence and the number and specific combinations of microorganisms and the host's immune defense in the periapical tissues. Bacteria may be temporarily present in the inflamed periradicular tissues only to be killed by the host's defense mechanisms when the focus of infection in the root canal system is effectively eliminated by mechanical instrumentation, antiseptic irrigation, and intracanal medication. For instance, the majority of apical

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periodontitis lesions with abscess formation or draining sinus tracts heal satisfactorily after nonsurgical root canal treatment without the need for systemic antimicrobial therapy.[209]

Primary root canal infection in untreated root canals is a polymicrobial mix with approximately equal proportions of gram-positive and gram-negative bacterial species, dominated by obligate anaerobes (see Chapter 15).[273,275] In root-filled teeth with apical periodontitis, gram-positive microorganisms, with a relatively equal distribution of facultative and anaerobic species, appear to dominate other microorganisms.[182,276] A high prevalence of *E. faecalis* is frequently observed in filled root canals associated with persistent apical periodontitis.[97,127,182,213,254] These issues are described in greater detail in Chapter 15.

Physical (overinstrumentation, overfilling) and chemical (irrigants, intracanal medication, root canal filling materials) insults,[240] as well as traumatic injury[11,12] to the periapical tissues, can also cause apical periodontitis, depending on the severity of injury and cytotoxicity of the chemicals. Foreign bodies, such as root canal filling materials, have been shown to cause persistent periapical inflammation.[136,193,211,324] However, the possibility of bacterial contamination in foreign body–induced apical periodontitis lesions was not carefully ruled out in many studies, therefore it is possible that the foreign bodies might serve as carriers for the microorganisms. In addition, foreign bodies have the odd property of favoring infection,[325] as they can lower the infectious dose of bacteria and cause granulocytes to develop phagocytic defect or loss of ammunition.[326] Although most root canal filling materials are not inert and are capable of inducing certain degrees of inflammation, in general they are biocompatible and well tolerated by periapical tissues.[99,102]

It has also been demonstrated histologically that periodontal disease could cause inflammatory pulpal and periapical disease.[146]

Infection: A conflict between host and parasites

Every infection is a race between the capabilities of the microorganism to

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multiply, spread, and cause disease and the ability of the host to control and finally eliminate the microorganisms.[305] The host has physical barriers—surface epithelium, enamel, and dentin—as well as innate and adaptive immune defenses to prevent establishment of infection. Nevertheless, microbes also possess weapons, leading to inhibition of phagocytosis, inhibited lysosomal function, reduced killing by phagocytes, inactivation of complement system and immunoglobulins, and specific mechanisms that permit invasion of the host's physical barriers.[305] Infection of a tissue can manifest different histopathologic features as a result of specific host-microbe interactions that occur. Many infections are asymptomatic in more than 90% of individuals.[305] For instance, pulp necrosis and chronic apical periodontitis caused by root canal infection are usually asymptomatic, and patients are often surprised to find out that this infection has been present for a sufficient time to lead to destruction of periapical bone. There is no simple correlation between infection and clinical symptoms of apical periodontitis, except in cases of symptomatic apical periodontitis and acute apical abscess.

Pathogenesis

It is a general belief that the development of apical periodontitis follows total pulp necrosis. This belief is based on (1) the pulpal strangulation theory due to a generalized increase in pulpal interstitial pressure inside the uncompromised pulp space during pulpal inflammation that causes collapse of venules and cessation of blood flow[99] and (2) animal and human studies that concluded that uncontaminated necrotic pulps that are intentionally devitalized or accidentally traumatized are generally incapable of inducing periapical inflammation unless they are infected.[161,184,272] However, if the vital pulps become infected due to caries or other pathways, periapical inflammation can develop even when inflamed, but vital tissue is still present in the apical portion of the root canal.[159,224] Most of our information related to the histopathology of apical periodontitis comes from analysis of longstanding, chronic human lesions caused by caries or from time-course studies of development of apical periodontitis induced by artificial root canal infection in animals. In these instances, the moment of transition from pulpitis to apical periodontitis was not captured. In fact, apical periodontitis

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has been demonstrated to be a direct extension of apical pulpitis into the periapical tissues before total pulp necrosis caused by root canal infection (Fig. 16.1).[138,159,160,222,223,279] For example, Kovacevic and colleagues[138] studied the transition from pulpitis to apical periodontitis in dogs' teeth by artificial exposure of pulps to the oral cavity and observed that pulpitis was coupled with an acute apical periodontitis. Similarly, Kimberly and Byers[131] demonstrated that periapical changes, including sprouting of nerve fibers, appeared 3 to 5 weeks following establishment of irreversible pulpitis subsequent to pulp exposure lesions in animals. Yamasaki and coworkers[321] and Stashenko and colleagues[266] also showed that periapical inflammatory infiltrates, increased osteoclast numbers, and bone destruction were apparent well in advance of total pulpal necrosis, with vital pulp tissue still present in the apical portion of the root canal. The biologic basis for these observations appears to hinge on the apical development of pulpal infection/inflammation leading to the diffusion of many inflammatory mediators, proinflammatory cytokines, chemokines, and bacterial toxins into the periapical area prior to total pulpal necrosis.

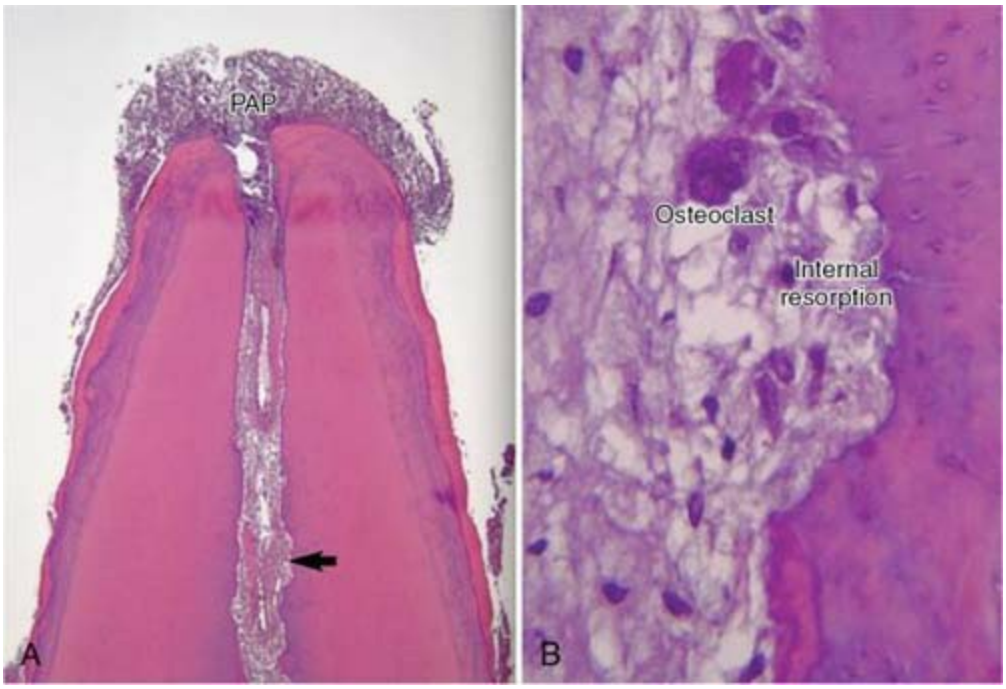

FIG. 16.1 **A,** Inflammation of the pulp tissue in the apical root canal extends into the periapical tissues in a mature tooth (H&E, magnification: c100). **B,** Arrow in **A**. High magnification of the pulp

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tissue in the apical root canal in **A**. The pulp tissue is vital and infiltrated with chronic inflammatory cells. Note resorption of canal wall and multinucleated clast cells (H&E magnification: ×200).

A) Stained micrograph shows tooth, marked PAP, at the root apex. A rightward arrow points toward build up tissue in the root canal.

B) Stained micrograph shows pulp tissue with two labels osteoclast and internal resorption.

Source: (Courtesy Dr. Domenico Ricucci, Rome, Italy.)

When pulps are infected/inflamed, many innate and adaptive immune cells are activated and release elevated amounts of various inflammatory mediators, including cytokines, chemokines, and neuropeptides. As the pulpal inflammation spreads, the inflammatory mediators begin to alter the pathobiology of the periapical tissues, manifesting as vasodilation, increased vascular permeability, infiltration of immunoinflammatory cells, and bone resorption. Clinically, the observable changes on radiographic examination of apical periodontitis are widening of the periodontal ligament space or development of apical osteolytic lesions due to bone resorption. The loss of bone is mainly caused by activated osteoclasts. Many cytokines, such as interleukin (IL)-1, IL-6, IL-11, IL-17, and tumor necrosis factor α (TNF-α), are found to have the ability to induce osteoclast progenitor cell differentiation and activation.[27,263] The inflammation-induced bone resorption in the periapical tissues is accompanied by recruitment of immunoinflammatory cells that essentially build a defensive line against the spread of microbial invasion from the root canal.[176]

The pathogenesis of apical periodontitis involves innate and adaptive immune responses as well as sensory nerve response in the periapical tissues. Immunoinflammatory cells present in human periradicular lesions consist of lymphocytes, macrophages, plasma cells, neutrophils, dendritic cells, and natural killer (NK) cells with the former two types as the majority.[156,175] The characteristic features of innate and adaptive immunity are summarized in Table 16.1.

Table 16.1

Features of Innate and Adaptive Immunity

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Property	Innate	Adaptive
Recognition	Structures shared by groups of related microbes (conserved molecular patterns)	Antigens of microbes and of nonmicrobial antigens (details of molecular structure)
Diversity	Limited	Very large
Memory	None	Yes
Receptors	Encoded in the genome	Encoded in gene segments (somatic recombination)
Blood proteins	Complement	Antibodies
Cells	Macrophages	Lymphocytes
	Neutrophils	Antigen-presenting cells
		NK cells
Action time	Immediate activation of effectors	Delayed activation of effectors
Response	Costimulatory molecules, cytokines	
(IL-1, IL-6)		
IL-2	Clonal expansion	
Chemokines (IL-8)	Effector cytokines (IL-4, IFN*r*)	

IFN, Interferon; *IL,* interleukin; *NK,* natural killer.

Data from Janeway CA, Medzhitov R: Innate immune recognition, *Annu Rev Immunol* 20:197, 2002; Abbas AK, Lichtman AH, Pober JS: *Cellular and molecular immunology*, ed 5, Philadelphia, 2003, Saunders.

Innate immune response

Specificity of innate immune response

In recent years, the concept of the nonspecific nature of innate immunity has changed since identification of a network of germline-encoded receptors, PRRs, which recognize specific molecular motifs of microorganisms.[5,6,178] PRRs can be expressed on the cell surface (macrophages, dendritic cells, neutrophils, NK cells, B cells), in intracellular compartments, or secreted into the blood and tissue fluids.[118] There are numerous microbial constitutive and conserved products such as PAMPs also noted earlier. Importantly, the PRRs of the innate immune system recognize PAMPs.[5,178]

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The specificity of innate immunity is due to the recognition of PAMPs of microorganisms by PRRs, such as TLRs of the host's cells. Activation of PRRs triggers numerous host responses, including opsonization, activation of complement and coagulation cascades, phagocytosis, activation of proinflammatory signaling pathways, and induction of apoptosis.[118] For example, TLR4/CD14 is the receptor for the gram-negative bacterial LPS. TLR4-mutated C3H/HeJ mice (LPS hyporesponsive) have reduced response to gram-negative bacteria and are highly susceptible to infection by *Salmonella typhimurium* or *Neisseria meningitidis*.[51] Importantly, there is a reduced expression of IL-1 and IL-12 and decreased periradicular bone destruction in TLR-4 deficient mice when teeth are subjected to pulpal exposures and infection with a mixture of four anaerobic pathogens: *Prevotella intermedia*, *Fusobacterium nucleatum*, *Streptococcus intermedius* (G+), and *Peptostreptococcus micros* (G+).[108] In addition, LPS is shown to be capable of inducing pain via direct activation of TLR4/CD14 expressed on nociceptive sensory neurons.[304] Thus, the TLR4 PRR receptor is importantly involved in odontogenic infections.

Components such as LTA of gram-positive bacterial cell walls can also stimulate innate immunity in a way similar to LPS. TLR2 plays a major role in detecting gram-positive bacteria and is involved in the recognition of a variety of microbial components, including LTA, lipoproteins, and peptidoglycan. The importance of TLR2 in the host defense against gram-positive bacteria has been demonstrated using TLR2-deficient ($TLR2^{-/-}$) mice, which were found to be highly susceptible to challenge with *Staphylococcus aureus* or *Streptococcus pneumoniae*.[69,281] LTA also stimulates leukocytes to release inflammatory mediators, including TNF-α, IL-1β, IL-6, IL-8, and prostaglandin (PG) E2, which are known to play a role in various phases of the inflammatory response. All of these inflammatory mediators have been detected in periapical samples, and each has a well-known tissue-damaging effect by activating various host responses.

The innate immune response to bacterial infection induces expression of proinflammatory cytokines (IL-1, IL-6, IL-8, IL-17, TNFα, IFN-γ), chemokines (CXCR1, CXCR2, TGF-β), and costimulators, which are essential for activation and influence of the nature of adaptive immune response.[5,177] The innate immune system is capable of recognizing non-self

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and self-antigens, whereas the adaptive immune system is not; thus, many autoimmune diseases are disorders of adaptive immunity.[177]

Nonspecific innate immune response

The primary nonspecific innate immune defense mechanism in apical periodontitis is phagocytosis of microbes by specialized phagocytes such as polymorphonuclear leukocytes (PMNs) and macrophages. This type of immune response does not require specific recognition of invaders by phagocytes. Tissue inflammation leads to the recruitment of PMNs from the blood circulation into the periradicular tissue. Activated PMNs exhibit an abrupt increase in oxygen consumption, the well-known respiratory burst, resulting in the release of oxygen radicals, a family of extremely destructive, short-lived substances that destroy nearby microorganisms and host cells.[17] Phagocytized microbes or foreign particles are exposed to a toxic environment containing specific azurophil granules and oxygen-derived free radicals and are eventually degraded.[200] PMNs also possess an extracellular killing mechanism via neutrophil extracellular traps (NETs), which are extracellular structures composed of chromatin with specific proteins from the neutrophilic granules. Upon activation (e.g., by IL-8, LPS, bacteria, fungi, activated platelets), neutrophils start a cellular program called *apoptosis* that leads to their death and the formation of NETs, which have antimicrobial activities.[33,80] Besides their role in the innate immunity as professional phagocytes, macrophages also serve as APC by expressing MHC (major histocompatibility complex) class II molecules that interact with antigen-specific clones of T-helper lymphocytes. Circulating monocytes are the precursors of both tissue macrophages and many dendritic cell subsets.[1,129,244] The details of the immunologic activities of neutrophils and macrophages in periapical pathosis are described in Chapters 13 and 14.

Adaptive/specific immune response

Continuous or severe infections at levels beyond the defense capacity of innate immunity are mediated by adaptive immunity, which is much more specific toward exogenous antigens. Adaptive immunity possesses the ability to memorize and respond to repeated exposure to the same antigen. The specificity of adaptive immunity is regulated at genetic levels in B and T

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lymphocytes through a complex process leading to the generation of molecules that recognize and bind to foreign or self-antigens. These molecules are specific receptors on T cells (T-cell antigen receptors or TCRs) and on B cells (B-cell antigen receptors or BCRs; also termed *immunoglobulins*). TCRs on T cells interact with antigens that are presented by antigen presenting cells (APC) expressing MHC II molecules along with other accessory molecules, whereas BCRs on B cells interact with antigens directly. BCRs may be secreted in the blood circulation or in the tissues as antibodies. The variable region of both TCR and BCR proteins are rearranged at the genomic level via genetic recombination of the V(D)J segments. The estimated total diversity after this recombination for TCR is approximately 10^{21} and for BCR is approximately 10^{15} that generates the repertoire of different individual T- and B-cell clones.[1,119] Each T- or B-cell clone generated in the bone marrow carries a specific TCR and BCR. They undergo a positive and negative selection process through which most clones are deleted via apoptosis because they bind to self-antigens. This initial "negative screening" process reduces the potential for autoimmune disorders. Only those that do not interact with self-antigens are released into the lymphatic system and blood circulation. The naive T cells circulate back and forth between the lymphatic system and blood circulation until they encounter foreign antigens presented by APC. About 97% of T cells undergo apoptosis, and only a small percentage of these cells are exported to the periphery as mature T cells.[1]

The interaction between TCR and the antigen peptide/MHC complex and co-stimulators activates T cells, leading to the synthesis of T-cell growth factor, IL-2, and its receptor that causes T-cell clonal expansion/proliferation. Some of these T cells differentiate into armed, effector T cells, and others become memory cells. There are a number of T-cell subpopulations, categorized by their functions: (1) T helper cells (T_H), (2) T regulatory cells (T_{reg}), (3) T suppressor cells (T_S), and (4) T cytotoxic (cytolytic) (T_C) cells.[1,60,301] Some of them can be distinguished by their cell surface markers, cytokine profiles, or transcriptional factors. See also Chapter 14 for additional details.

The development of CD4 T_H cells involves the encounter of APCs in association with class II MHC. All tissue cells express MHC class I, but only

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certain cells express class II MHC. Class II MHC–expressing cells constitute the body's population of APCs and consist of (1) dendritic cells, (2) macrophages, (3) B cells, (4) vascular endothelial cells, and (5) epithelial cells. The former three are considered "professional" APCs, because they are dedicated to this function. The latter two APCs are quiescent under normal conditions but can be induced to express class II MHC when exposed to elevated concentrations of IFN-γ.[1,119]

Dendritic cells and macrophages phagocytose foreign antigens, whereas B cells utilize the membrane-bound immunoglobulin to bind and internalize antigens. Other APCs endocytose foreign antigens into the cytoplasm for antigen processing and the processed antigens are partially degraded into small peptides. Many of them are 10 to 30 amino acids long and capable of binding onto the newly synthesized class II MHC molecules before the antigen/class II MHC complex is transported to the surface of cells and presented to TCRs of $CD4^+$ T cells.

Although controversial, evidence has suggested that $CD8^+$ T_S and $CD8^+$ CTLs represent distinct subpopulations of $CD8^+$ T cells. T_S are MHC class I–restricted $CD8^+/CD28^-$ T_S cells, which act on APC by a contact-dependent manner, rendering them tolerogenic to T_H cells. They inhibit the proliferation of T_H cells.[53,327] T cytotoxic cells ($CD8^+T_C$), also known as *cytolytic T lymphocytes* (CTLs), are a subset of T cells that kill target cells expressing MHC-associated peptide antigens. The majority of T_C cells express CD8 and recognize antigens degraded in the cytosol and expressed on the cell surface in association with class I MHC molecules of the target cells. Functional T_C acquire specific membrane-bound cytoplasmic granules, including a membrane pore-forming protein called *perforin* or *cytolysin* and enzymes called *granzymes*.[1]

Upon antigen stimulation, naive CD4 T cells can proliferate and differentiate into T_H0 cells, which are subsequently committed to develop into T_H1 or T_H2 cells.[1] Monocytoid DC (dendritic cell) (DC1) induces T_H1-type responses; plasmacytoid DC (DC2) selectively induces T_H2 responses. Each subset of T_H cells has distinct functions and cytokine profiles. T_H1 cells develop from T_H0 activated by IL-12 and IFN-γ and mainly produce IL-2,

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interferon (IFN)-γ, and TNF-α, which activate macrophages. T_H2 cells develop from T_H0 activated by IL-4 and IL-10 and produce IL-4, IL-5, IL-10, and IL-13, which activate B cells to make antibody. T_H0 can also develop into T_H17 under the activation of IL-6 and TGF-β and produce IL-17, a powerful proinflammatory cytokine. Macrophages, dendritic cells, and T cells produce antiinflammatory cytokine, IL-10. Overall, T_H1 and T_H2 have mutually inhibitory effects.[1]

The role of B cells in adaptive immunity is mainly the production of antibodies that constitute the host humoral immune response. The V(D)J gene recombination occurs in both heavy and light (κ and λ) chains. A recombinase system, which is an enzymatic complex consisting of several enzymes—including recombination activating gene 1 and 2 (RAG-1, RAG-2), terminal deoxynucleotidyl transferase (TdT), DNA ligase IV, Ku proteins, and XRCC4—is essential for successful recombination. This recombinase system is also used for TCR recombination. Mature IgM/IgD coexpressing B cells undergo isotype switching via a process called *switch recombination* after encountering antigen. The rearranged V(D)J gene segment recombines with a downstream C region gene (γ, ε, or α), and the intervening DNA sequence is deleted. This gives rise to other classes of immunoglobulins (IgG, IgE, IgA) besides IgM. In addition to isotype switching, activated B cells also undergo somatic mutation in the V region gene, leading to affinity maturation of antibodies and alternative splicing of VDJ RNA to membrane or secreted immunoglobulin mRNA. A large quantity of antibody is secreted when B cells terminally differentiate into plasma cells.[1,119] The ability of antigens to selectively stimulate the differentiation of plasma cells supports the clinical finding that plasma cells isolated from periapical lesions secrete antibodies specific for the particular bacteria found in the adjacent root canal system.

The immune response and the role of lymphocyte subpopulations in apical periodontitis lesions were investigated by employing lymphocyte-deficient rodents as study models. T-cell deficiency appears to accelerate bone loss at the early phase of apical periodontitis lesions (2 weeks) but does not affect the overall course of lesion development.[282,306] Using RAG-2 SCID mice (both T- and B-cell-deficient), it was found that approximately a third of the RAG-2 mice developed endodontic abscesses, whereas no immunocompetent

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controls had abscesses.[284] In another study, specific RAG-2 knockout (k/o) mice were used to determine which immune element was important for the defense mechanism in endodontic infection. The results demonstrated that B cells, but not T cells, played a pivotal role in preventing dissemination of endodontic infection.[109] Therefore, both T and B cells mediate the observed immune responses in apical periodontitis lesions.

Neurogenic inflammation

Certain primary afferent sensory nerve fibers, upon stimulation by various irritants, release neuropeptides, which cause vasodilation, protein extravasation, and recruitment/regulation of immune cells such as macrophages, neutrophils, mast cells, and lymphocytes. This phenomenon is termed *neurogenic inflammation*.[29,32] Pivotal neuropeptides in the induction of neurogenic inflammation are calcitonin gene-related peptide (CGRP) for vasodilation, and SP for the induction of protein extravasation. Neuropeptides and their receptors are widely distributed throughout the body. During inflammation, there is a sprouting of afferent fibers[41,42] and local increases in inflammatory mediators that trigger neuropeptide release, leading to neurogenic inflammation.[29,46] Besides the cardinal functions of the key neuropeptides that cause the first sign of inflammation—vasodilation and increased vascular permeability—the role of these neuropeptides in the process of inflammation is now known to be far more complex. In the development of chronic apical periodontitis lesions, neuropeptides are also involved in immune regulation, bone resorption, and wound healing. At sufficient concentrations, SP increases the secretion of IL-1, TNF-α, and IL-6 from macrophages and stimulates T-lymphocyte proliferation and enhances antigen-induced IFN-γ production by T cells.[264] Certain neuropeptides, such as SP, up-regulate immune and inflammatory responses, whereas other neuropeptides, such as CGRP, vasoactive intestinal peptide (VIP), and neuropeptide Y (NPY), inhibit inflammatory responses. Synergistic interactions among neuropeptides, such as CGRP and other inflammatory mediators, eicosanoids, and bradykinin, suggest a complex interplay between these molecules in the immune response.[86,219,264]

In chronic apical periodontitis lesions, specific receptors for SP and CGRP are expressed in certain immune cells, including macrophages and

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lymphocytes. Both CGRP and VIP may play a role in inhibiting bone resorption by suppressing osteoclastic functions. The level of VIP in apical periodontitis lesions is inversely related to lesion size. Osteoclast cell culture studies have demonstrated that the presence of greater concentrations of VIP leads to a decrease in their ability to form lacunae of osseous resorption, causing rapid cytoplasmatic contraction and reduced cell mobility. VIP exerts an effect on macrophages to block the production of TNF-α, IL-6, and IL-12, suggesting that VIP could have a role in controlling growth of apical periodontitis lesions.[46]

The major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis caused by root canal infection are illustrated in Fig. 16.2.

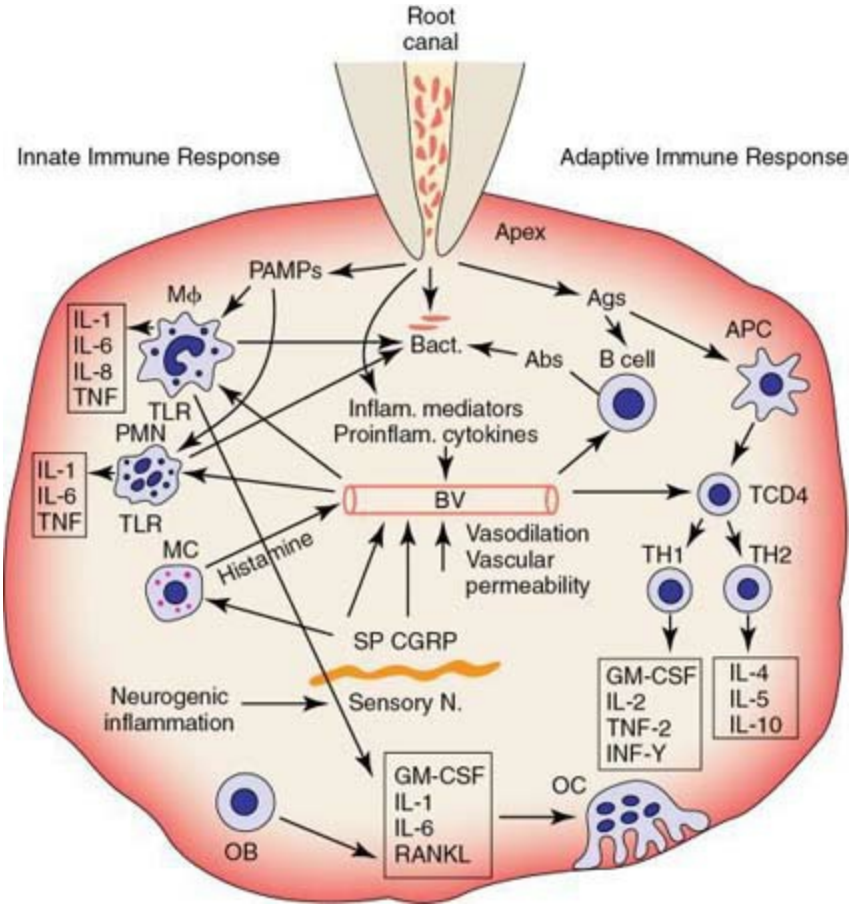

FIG. 16.2 Major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis. *APC,* Antigen presenting cell; *BV,* blood vessels; *CGRP,* calcitonin gene–related peptide; *GM-CSF,* granulocyte/monocyte colony-

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stimulating factor; *INF*, interferon-*r*; *MC,* mast cell; *Mφ,* macrophage; *OB,* osteoblast; *OC,* osteoclast; *PAMPs,* pathogen-associated molecular patterns; *PMN,* polymorphonuclear leukocyte; *RANKL,* receptor activator of nuclear factor κB ligand; *TLR,* Toll-like receptor; *TNF*, tumor necrosis factor.

A schematic diagram shows innate immune response and adaptive immune response caused by root canal infection.

Innate immune response: PAMPs leads to TLR in PMN and TLR in M phi followed by IL-1, IL-6, IL-8, TNF. PMN further leads to bacteria along with IL-1, IL-6, and TNF, whereas M phi leads to bacteria as well as GM-CSF, IL-1, IL-6, RANKL from OB that further leads to OC during neurogenic inflammation. Beside this, SP CGRP leads to MC and BV. MC along with histamine further leads to BV. BV ultimately leads to Mm phi and PMN.

Adaption immune response: Ags from apex leads to APS and B cell. APS further leads to TCD4 further leads to TH1 and TH2. TH1 leads to GM-CSF, IL-2, TNF-2, INF-Y and TH2 leads to IL-4, IL-5, IL-10. SP CGRP from sensory nerve leads to BV through vasodilation vasculator permeability that further leads to TCD4 and B cell followed by Abs then bacteria from root canal infection. Inflam. mediators proinflam. cytokines leads to BV.

Source: (Courtesy Dr. Louis M. Lin, NY.)

Diagnosis

Correlation between clinical and histologic findings

Clinical diagnosis of inflammatory periapical disease is mainly based on clinical signs or symptoms, duration of disease, pulp tests, percussion, palpation, and radiographic findings (see also Chapter 1). In contrast, a histologic diagnosis is a morphologic and biologic description of cells and ECM of diseased tissues. The clinical diagnosis represents a provisional diagnosis of disease based on signs, symptoms, and testing results, whereas the histologic diagnosis is a definitive diagnosis of the disease.

Apical periodontitis can be symptomatic or asymptomatic. Similar to pulpitis, apical periodontitis is not always symptomatic or painful.[241,242,248] Although many inflammatory mediators (histamine, bradykinin, prostaglandins) and proinflammatory cytokines (IL-1, IL-6, TNF, nerve growth factor [NGF]), as well as neuropeptides (SP, CGRP), are capable of

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sensitizing and activating nociceptors of sensory nerve fibers,[99,100] other mediators such as endogenous opioids and somatostatin released by inflammatory cells during inflammation are able to inhibit firing of sensory nerve fibers.[105,246] Activation of nociceptors of sensory nerve fibers may also be related to concentrations of inflammatory mediators. The complexity of these findings supports the clinical observation that there is no good correlation between clinical symptoms and histopathologic findings of apical periodontitis.[30,164] For example, many teeth with apical periodontitis are free of symptoms. In addition, pain is mediated at molecular level and hard to identify histologically.[21]

Correlation between radiographic and histologic findings

Radiography is designed to detect pathologic changes at tissue and not cellular level. Even using sensitive imaging systems such as ultrasound, cone-beam computed tomography, and other imaging technologies, it is impossible to detect the presence of inflammatory cells or other subtle changes in the periapical tissues. Using conventional radiographic and histologic methods in the same cadavers, evidence of inflammation was often observed in the periapical tissues of endodontically treated teeth with histologic examination but not with normal radiographic examination.[19,39,88] It has also been demonstrated that lesions localized in the cancellous bone may not be visible radiographically unless they involve cortical bone (see also Chapter 2).[23,25,112] In addition, radiographic images are unable to differentiate apical granuloma and apical cyst.[144,225,278] Accordingly, radiographic findings and histopathologic features of apical periodontitis have a poor correlation. Histologic examination remains the gold standard of diagnosis of inflammatory periapical lesions. Cone beam computed tomography is more sensitive than conventional periapical radiography to detect periapical osteolytic lesions.[212]

The absence of clinical symptoms and negative periapical radiographic findings of endodontically involved teeth does not necessarily indicate absence of apical periodontitis.[19] By the same token, clinical success of endodontically involved teeth (i.e., absence of signs and symptoms and

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negative periapical radiographic findings) after nonsurgical root canal therapy does not necessarily imply complete histologic healing of periapical lesions. Thus, currently available diagnostic methods used in endodontics, such as clinical symptoms, percussion, palpation, pulp sensibility tests (cold, heat, electric), and radiographic imaging are not sensitive enough to provide accurate diagnosis of inflammatory periapical lesions. Until we have more advanced and sophisticated clinical diagnostic technologies, we will continue to face the problem of clinical diagnosis of inflammatory periapical lesions. Nevertheless, the treatment of various types of apical periodontitis lesions is basically the same: nonsurgical root canal therapy.

Histopathology

Histopathology focuses on cellular biology of diseased tissues and organs. Biochemical disorders occur inside the cells before histologically observable morphologic changes of cell injury. Cell death (protein denaturation) occurs before observable morphologic changes of cell necrosis. Gene transformation or mutation occurs before observable morphologic changes of neoplastic cells.[171]

Based on etiology, clinical signs and symptoms, and duration of the disease process, the World Health Organization (WHO) classifies disease of periapical tissues into many categories.[318] There are also many classifications of inflammatory periradicular disease in several endodontic textbooks[113,209,293] and by the American Board of Endodontists, depending on clinical manifestations and histologic appearances. In addition, the American Association of Endodontists held a consensus conference on diagnostic terms in the fall of 2008. Traditionally, there has been a lack of consensus on clinical diagnostic terminology of pulpal and periapical disease in the endodontic community because of the paucity of studies with high levels of evidence. This chapter will focus on pathobiology of apical periodontitis.

The histopathologic analysis of a diseased tissue or organ only shows structural changes of cells and ECM at the time the tissue or organ is removed. Therefore, it does not represent the complete kinetics or spectrum of disease development. Histologic classification of apical periodontitis is

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based on types of cells participating in inflammatory responses in the periapical tissues in this chapter. In general, inflammation can be divided into acute and chronic responses, depending on types of cells present at the site of injured tissue.[141,171,259,310] Acute inflammatory response is characterized by participation of neutrophilic leukocytes and chronic inflammatory response by participation of macrophages and lymphocytes. However, many factors, such as severity of tissue injury, specific etiology, host's resistance, and particular tissue involved, can modify the course and morphologic variations as well as cell biology of both acute and chronic inflammatory responses.[141,171] Acute and chronic inflammatory responses are in fact not rigid phases of a single programmed event but two overlapping responses with partially different triggering mechanisms and programs.[171] Sometimes apical periodontitis may be associated with abscess formation. *Abscess* is a localized collection of purulent exudate in a tissue.[141] The development of an abscess in apical periodontitis lesions is probably caused by colonization of a combination of specific pyogenic pathogens in the inflamed periapical tissues.[35,207,274,302] Abscess begins as a furious battle between highly virulent pathogens and an army of neutrophilic leukocytes. The pathogens produce massive toxins to kill neutrophils. As neutrophils attack the pathogens, they secrete lysosomal enzymes that digest not only the dead cells but also some live ones. Many neutrophils die fighting against pathogens. The resulting purulent exudate is poorly oxygenated and has a low pH. The bactericidal ability of leukocytes appears to be impaired because of the deprivation of oxygen and interference of respiratory burst. The purpose of respiratory burst is to generate bactericidal agents.[17,171] However, the phagocytic activity of leukocytes is not impaired in anaerobic conditions.[171] To avoid confusion between histologic and clinical diagnosis, readers are encouraged to read the chapters related to clinical examination, radiographic interpretation, and clinical diagnosis of inflammatory periapical disease (see Chapters 1, 2, and 5).

Acute apical periodontitis

The development of acute apical periodontitis largely reflects the innate immune response and is the first line of active defense against irritants from

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the root canal. Acute apical periodontitis is an immediate defense reaction to irritants and does not require exquisite specificity and memory. Characteristic features of acute apical periodontitis are similar to the typical acute inflammatory reaction in all connective tissues and consist of vasodilation, increased vascular permeability, and transmigration of leukocytes from the blood vessels into perivascular tissue space. The beneficial actions of acute inflammation are (1) infiltration of leukocytes to the injured tissue to phagocytose and kill microbial agents; (2) accumulation and activation of humoral factors such as immunoglobulins, complement factors, plasma proteins, and innate immune cytokines (IL-1, TNF) in the injured tissue to recruit more neutrophils and macrophages; and (3) neutralization or degradation of bacterial toxins and their harmful metabolic by-products.[141,171]

Cell biology

The inflammatory response is a dynamic interaction between host defense mechanisms and microbial insults. The interlacing activation and control pathways of cellular and humoral components involved in the inflammatory response are complex. The cells involved—neutrophils, monocytes/macrophages, platelets, mast cells, T lymphocytes, B lymphocytes, NK cells, dendritic cells, endothelial cells, fibroblasts, eosinophils, and basophils—each have numerous functions that are activated and modulated by a multiplicity of biochemical messengers.[1,141,171] *Cell activation* means that the cell acquires the ability to perform one or more new functions or to perform normal functions at a higher rate[1]; it often results in transcription of new genes and synthesis of new proteins.[1]

Mast cells

Histamine stored in the cytoplasmic granules of mast cells is the mediator that first appears in acute inflammation to induce vasodilation and increased vascular permeability; mast cells are the designated triggers of acute inflammation. They are widely distributed in perivascular tissue spaces and originate in the bone marrow from precursor cells. Mature mast cells contain numerous cytoplasmic granules, which are the source of vasoactive mediators. The preformed histamine is released by mast cell degranulation

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and can be triggered by (1) physical stimuli such as cold, heat, and mechanical trauma; (2) binding of IgE-specific antigen to mast cells and membrane-bound IgE antibodies; (3) binding of complement components (C3a and C5a) to their complementary receptors on mast cells; and (4) stimulation by neuropeptide (SP) and cytokines (IL-1, IL-8).[1,32,141,171] In addition, activated mast cells secret cytokines (TNF-α, IL-1, IL-3, IL-4, IL-5, IL-6, IL-13), prostaglandins, and leukotrienes to enhance inflammatory defense mechanisms.[1,40,129]

Endothelial cells

Endothelial cells are important players in the inflammatory response. Without the participation of endothelial cells, the host is unable to deliver its cellular and humoral defense components from the circulating blood to the site of tissue injury. Inflammatory mediators, complement components, proinflammatory cytokines, nitric oxide, neuropeptides, and bacterial toxins can all affect endothelial cells, resulting in vasodilation and increased vascular permeability.[1,141,171] IL-1 and TNF released by activated macrophages and NK cells can stimulate endothelial cells to express intercellular adhesion molecules (ICAMs), such as ICAM-1, ICAM-2, ICAM-3, vascular cell adhesion molecule (VCAM), and platelet endothelial cell adhesion molecule (PECAM), which enhance leukocyte adhesion to endothelial cells and transmigration through the blood vessels.[1,141,153,171] IL-1, TNF, and LPS also can activate endothelial cells to synthesize chemokine (IL-8), a potent chemotactic mediator for neutrophils.[1]

Polymorphonuclear neutrophilic leukocytes

Polymorphonuclear neutrophilic leukocytes (PMNs) are the principal effector cells in acute apical periodontitis. They are derived from bone marrow hematopoietic stem cells. Neutrophils have a lobulated nucleus and contain primary or azurophil (elastase and myeloperoxidase) and secondary or specific (lysozyme and other protease) granules in their cytoplasm.[129,141,171] Neutrophils are only present in the blood circulation. They are the first leukocytes to transmigrate through the blood vessels into perivascular tissue space and then are directed toward the wound or irritants, peaking at 24 to 48 hours. The transmigration of neutrophilic leukocytes from the blood vessels

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into perivascular space involves complex cellular and molecular biology. Following vasodilation and increased vascular permeability, leukocyte margination, rolling, capture, and activation in the blood vessel and then transmigration through the blood vessel are mediated by an intricate interaction of cell adhesion molecules expressed on leukocytes (L-selectin, integrins) and on endothelial cells (P- and E-selectins, ICAM, VCAM, PECAM-1). Neutrophil rolling is mediated by interaction between leukocyte selectin ligands and P-selectins on endothelial cells. Leukocyte sticking is mediated by interaction between leukocyte integrins and ICAMs and VCAMs on endothelial cells. Leukocyte transmigration is mediated by interaction between PECAM-1 on both leukocytes and endothelial cells.[171] Chemokines (IL-8) increase the affinity of leukocyte integrins for their ligands on endothelial cells.[1] Once transmigrating through the junction between endothelial cells and basement membrane into the perivascular tissue space, neutrophilic leukocytes are directed toward stimuli by chemotactic factors or chemotaxins, such as bacterial products (fLMP), C3a, C5a, leukotriene B4 (LTB4), platelet-activating factor (PAF), fibrinopeptides, dead cells, and chemokines (IL-8) by receptor-mediated mechanisms.[1,141,171,259]

Neutrophils can be activated by bacteria and PAMPs (also known as the TLRs, described earlier). They can also be stimulated by IL-1, TNF, and chemokines produced by activated macrophages and NK cells to enhance the phagocytic activity of infectious agents and synthesis of defensins, which are broad-spectrum antibiotics.[1] Neutrophilic leukocytes are terminally differentiated cells and short lived—within hours to a few days. Most neutrophilic leukocytes in acute inflammatory response die as a result of apoptosis or programmed cell death. The apoptotic neutrophils are phagocytosed by macrophages.[92,141,171] However, some neutrophilic leukocytes die after a furious battle against microbial infection and release intracellular proteolytic lysosomal enzymes, oxygen-derived active metabolites, nitric oxide, proinflammatory cytokines, eicosanoids, and matrix metalloproteinases into tissue to intensify inflammation and tissue damage.[141,171] The release of lysosomal enzymes by neutrophils and macrophages can also occur by lysosomal suicide due to rupture of phagolysosome in the cytosol, regurgitation during phagocytosis of irritants, or frustrated phagocytosis of indigestible foreign bodies. [141,171] The main

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effector functions of neutrophilic leukocytes are phagocytosis, killing of microbes, and release of inflammatory mediators (including proinflammatory cytokines) to recruit more leukocytes to prevent spread of infection.

Macrophages

Macrophages make their appearance as a second wave in acute apical periodontitis within 48 to 96 hours. Macrophages are blood-borne but have a counterpart in the connective tissue.[171] Blood monocytes transmigrate through the blood vessel into the tissues and become macrophages. Mature macrophages have an irregular-shaped nucleus and contain abundant lysosomes and many phagocytic vesicles (phagosomes). Monocytes use mechanisms similar to neutrophils to adhere to endothelial cells in high endothelial venule–expressing adhesion molecules for mononuclear leukocytes (ICAM for macrophages and VCAM for lymphocytes); they then transmigrate through the blood vessel and are directed toward the site of tissue injury by chemotactic factors.[171]

Macrophages can be activated by bacteria, PAMPs, and interferon-γ (INF-γ) and produce numerous products: lysosomal enzymes, coagulation factors, bioactive lipids, reactive oxygen species, chemokines, cytokines/growth factors, and angiogenesis factors.[141,171,259] Macrophages are the most dynamic and versatile leukocytes. The main functions of macrophages are numerous. They include phagocytosis of microbes and foreign bodies, production of inflammatory mediators, initiation of the immune response, cleanup operation of necrotic cells and tissue, induction of systemic effects (fever, acute phase reaction, cachexia), synthesis of molecules or cytokines affecting cell and vascular growth in wound healing, as well as antibacterial and antiviral defenses.[171] Tissue macrophages are not terminally differentiated cells and are able to undergo mitosis. Their life span is from several weeks to months.

Activated neutrophilic leukocytes and macrophages are capable of phagocytizing and killing pathogens. They recognize microbes through TLRs and receptors for the Fc fragment of immunoglobulin IgG as well as complement component C3b on their cell surface. C3b opsonization and antibody coating of microbes enhance recognition and phagocytosis of pathogens by activated neutrophilic leukocytes and macrophages.[1,141,171]

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Activated neutrophilic leukocytes are effective in phagocytosing and killing extracellular microbes, whereas macrophages activated by IFN-*r* produced by NK cells and T_H1 cells are more effective in phagocytosing and killing intracellular microbes.[1] Killing of phagocytosed microbes by neutrophilic leukocytes and macrophages is mediated through oxygen-dependent and oxygen-independent mechanisms. Oxygen-dependent mechanisms are more effective in killing all kinds of bacteria than oxygen-independent mechanisms.[1,141,259] The effector molecules of an oxygen-dependent system are hydrogen peroxide, superoxide anion, hydroxyl radical, singlet oxygen, and hypochlorite. An oxygen-independent system is also important in killing microbes and is dependent on lysozymes, defensins, and lactoferrin contained in phagocyte granules.[1,141,171,259] Some antimicrobial peptides and other oxygen-independent mechanisms possessed by phagocytes are specialized for killing of certain groups of microbes.[1,259]

Microbes phagocytosed by phagocytes are enclosed in membrane-bound phagosomes in the cytosol. The phagosomes fuse with lysosomes to form phagolysosomes. Irritants such as microbes, foreign protein antigens, and dead cells inside the phagolysosome are destroyed or degraded by proteolytic enzymes stored in lysosomes.[1] There are also granules that are fused with the nascent phagosome and release their contents into the phagosome. Some of these granules have direct antimicrobial action, such as defensins and the bactericidal permeability-increasing protein azurocidin. Others are proteases, such as elastase and cathepsins, lactoferrin, and peroxidase myeloperoxidase. Lysosomal enzymes, reactive oxygen intermediates, and nitric oxide released by neutrophils and macrophages indiscriminately kill not only bacteria but also tissue cells. Much of periapical tissue damage that occurs during acute inflammation can be attributed to release of proteolytic lysosomal enzymes and MMPs from disintegrated neutrophilic leukocytes and macrophages rather than to bacteria and their toxins.[1,171]

Platelets

Platelets normally circulate in the blood but also play an important role in inflammation. They are small cytoplasmic fragments derived from the megakaryocyte.[129] Platelets are essential for blood clotting, hemostasis, and fibrinolysis. Platelets produce vasoactive amines (PAF, serotonin),

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chemokines, and growth factors (PGDF, FGF, TGF) during inflammation.[171]

Natural killer cells

NK cells are also players in acute apical periodontitis. They are a subset of lymphocytes found in blood and lymphoid tissues. NK cells are derived from the bone marrow hematopoietic stem cells but lack the specific T-cell receptor for antigen recognition.[1] NK cells also possess TLRs for microbial constitutive and conserved products. The effector functions of NK cells are to lyse virus-infected cells without expressing class 1 MHC molecules and to secret IFN-*r* to activate macrophages. Antibody-coated cells, cells infected by viruses, some intracellular bacteria, and IL-2 released by activated macrophages can activate NK cells. Viruses have been isolated in apical periodontitis lesions.[228,260] NK cells kill target cells by antibody-dependent cell-mediated cytotoxicity because NK cells express receptor for the Fc fragment of IgG.[1] NK cells provide a link between the innate and adaptive immune systems.[118]

Inflammatory mediators

Numerous biochemical mediators are involved in the acute innate inflammatory response. They are mainly derived from plasma and inflammatory cells. The main biologic functions of inflammatory mediators are to cause vasodilation and increased vascular permeability and recruit inflammatory cells, mainly neutrophilic leukocytes and macrophages from blood circulation to the site of tissue injury. Some mediators can also cause tissue damage. The major mediators involved in vascular changes, cell recruitment, and tissue damage in the acute inflammatory response are listed in Table 16.2.

Table 16.2

Major Mediators in Inflammation

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Mediator	Source	Effector Cells and Tissues
VASODILATOR		
Histamine	Mast cells	Endothelial cells
	Platelets	
Prostaglandins	Leukocytes	Endothelial cells
	Mast cells	
Nitric oxide	Endothelial cells	Vascular smooth muscle
	Macrophages	
CGRP	Sensory nerve	Endothelial cells
Fibrin degradation products	Plasma	Endothelial cells
INCREASED VASCULAR PERMEABILITY		
Bradykinin	Plasma	Endothelial cells
Leukotrienes C4, D4, E4	Leukocytes	Endothelial cells
	Mast cells	
PAF	Leukocytes	Endothelial cells
	Mast cells	
	Endothelial cells	
C3a, C5a	Plasma	Endothelial cells
Fibrinopeptides	Plasma	Endothelial cells
Substance P	Sensory nerve	Endothelial cells
INCREASED EXPRESSION OF ENDOTHELIAL ADHESION MOLECULES (SELECTIN, ICAM, VCAM, PEAM)		
TNF	Activated macrophages	Endothelial cells
	NK cells	
IL-1	Activated macrophages	Endothelial cells
	NK cells	
Chemokines	Macrophages	Endothelial cells
	Neutrophils	
	Endothelial cells	
	Fibroblasts	
LEUKOCYTE ACTIVATION AND CHEMOTAXIS		
C3a, C5a	Plasma	Leukocytes
Leukotriene B4	Mast cells	Leukocytes
	Leukocytes	
Chemokines (IL-8)	Macrophages	Leukocytes
	Neutrophils	
	Endothelial cells	
	Fibroblasts	
Fibrinopeptides	Plasma	Leukocytes
Bacterial products (fMLP)	Bacteria	Leukocytes
TNF	Activated macrophages	Leukocytes

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	Dead cells	
OPSONINS		
C3b, C5b, immunoglobulins	Plasma	Microbes
TISSUE DAMAGE		
Lysosomal enzymes	Neutrophils	Cells and tissues
	Macrophages	
Free oxygen radicals	Activated leukocytes	Cells and tissues
Nitric oxide	Macrophages	Cells and tissues
Bacterial products (LPS)	Bacteria	Cells and tissues

CGRP, Calcitonin gene–related peptide; *fMLP,* formyl-methionyl-leucyl-phenylalanine; *ICAMs*, intercellular adhesion molecules; *IL,* interleukin; *LPS,* lipopolysaccharide; *PAF,* platelet activation factor; *PECAM*, platelet endothelial cell adhesion molecule; *NK cell,* natural killer cell; *TNF,* tumor necrosis factor; *VCAM*, vascular cell adhesion molecule.

Data from Kumar V, Abbas AK, Fausto N, et al: *Robbins and Cotran pathologic basis of disease*, ed 8, Philadelphia, 2010, Saunders; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, 2002, Mosby; Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, 2004, Oxford University Press; Abbas AK, Lichtman, Pober JS: *Cellular and molecular immunology*, ed 5, Philadelphia, 2003, Saunders.

All listed inflammatory mediators have been shown to be present in apical periodontitis.[289,290] Bradykinin is the product of kinin-system activation; fibrinopeptides, the products of blood-clotting system activation; and fibrin degradation products, the products of fibrinolytic-system activation. Kinin, fibrinolytic, and clotting systems are initiated by activated Hageman factor (coagulation factor XII). Prostaglandins and leukotrienes are the products of arachidonic acid metabolism. Activated phospholipase A2 splits cell membrane phospholipid molecules into arachidonic acid and platelet activating factor. Arachidonic acid molecules can be processed along two pathways: the cyclooxygenase pathway, which leads to production of prostaglandins and thromboxanes, and the lipoxygenase pathway, which produces leukotrienes.[141,171]

Complement components, such as C3a, C3b, C5a, C5b, and C5–C9, are products of the complement cascade, which can be activated by two pathways. The classic pathway is initiated by activation of C1 by multimolecular aggregates of IgG or IgM antibody complexed with specific antigen. The alternate pathway is activated by microbial cell components (LPS, teichoic acid) and plasmin. C3a and C5a are anaphylatoxins that stimulate mast cells and basophils to release histamine. They also cause phagocytes to release lysosomal enzymes. C3b is an opsonin and can coat

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bacteria to enhance phagocytosis by phagocytes. In addition, C3b can also bind to antibody bound to antigen or microbes. C5a is a strong chemotaxin for neutrophils. C5b-C9 is a membrane attack complex and able to cause cell lysis if activated on the host cell and bacterial cell membrane.[141,171]

Besides inflammatory mediators, the inflammatory response is also dependent upon the timing and extent of various cytokine and chemokine secretions. IL-1, TNF, IL-6, IL-12, and IFN-γ are present in the acute inflammatory response.[1,94] IL-6 is produced by activated mononuclear phagocytes, endothelial cells, and fibroblasts in response to microbial infection and other cytokines, such as IL-1 and TNF. IL-6 stimulates the synthesis of acute-phase proteins by hepatocytes.[1] The major source of IL-12 is activated mononuclear phagocytes and dendritic cells. IL-12 provides a link between innate and adaptive immune responses.[1] IFN-*r* is produced by activated NK cells, T_H1 cells, and cytotoxic T cells and can activate macrophages and enhance their microbial killing ability. IL-1β and TNF are associated with apical bone resorption during chronic apical periodontitis.[263]

Chemokines are a large family of structurally homologous cytokines that stimulate leukocyte movement and regulate the transmigration of leukocytes from the blood vessel to tissue space.[1] They are produced by leukocytes, endothelial cells, and fibroblasts. The secretion of chemokines is induced by microbial infection, TNF, and IL-1.[1,94,249] Different types of leukocytes express different chemokine receptors.[1]

Neuropeptides are released via axon reflexes of afferent sensory neurons in response to various stimuli. Neuropeptides, as mediators of the inflammatory process, are described in detail in the Neurogenic Inflammation section of this chapter. Inflammatory mediators such as histamine, kinins, prostaglandins, and proinflammatory cytokines are capable of sensitizing and activating sensory nerves to release neuropeptides.[32,100,221,235]

Histopathology

The acute inflammatory response is practically immutable in all vascularized living tissues, largely due to the programmed actions of the innate immune system. Initially, blood vessels are engorged by a local infiltration of inflammatory cells, mainly activated neutrophilic leukocytes and some

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macrophages in the apical periodontal ligament of the infected/inflamed root canal. In addition, sprouting of sensory nerve fibers has been shown early in the inflamed periapical tissues.[42,131] Several studies have also demonstrated the presence of inflamed vital pulp tissue with intact nerve fibers in the apical portion of the root canal in association with apical periodontitis.[159,222,223,321] This explains the clinical observation that a patient may experience some pain if an instrument is introduced into the canal short of the apex in some teeth with apical periodontitis lesions. Acute apical periodontitis with abscess formation may show localized collection of dead and live neutrophilic leukocytes, disintegrating tissue cells, and dead and live pyogenic pathogens (Fig. 16.3).

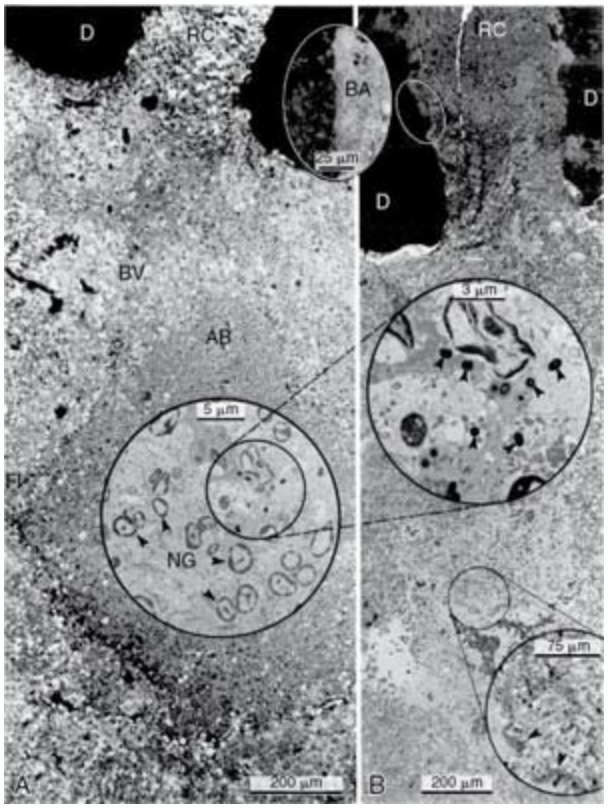

FIG. 16.3 Structure of a secondary periapical abscess. **A,** Axial section of an abscessed apical periodontitis. The microabscess *(AB)* contains a focus of neutrophils (*NG* inset in **A**). Note the phagocytosed bacteria in one of the neutrophils (further magnified in large inset in **B**). A secondary abscess forms when bacteria (*BA* in oval inset) from the apical root canal *(RC)* advance into the chronic apical periodontitis lesion (**B**). Note the tissue necrosis immediately in front of the apical foramen and the bacterial front in the body of the lesion *(arrowheads in*

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lower inset). BV, Blood vessels; *D,* dentin. (**A,** Magnification: ×130; **B,** magnification: ×100; oval inset, magnification: ×400; inset in **A**, magnification: ×2680; upper inset in **B**, magnification: ×4900; lower inset in **B**, magnification: ×250.)

A) Electron micrograph depicted on the scale of 200 micrometers shows D, RC, inset of BA, AB, and NG. NG has irregular and oval shaped cells. NG and BA are depicted on the scale of 5 and 25 micrometers, respectively.

B) Electron micrograph depicted on the scale of 200 micrometers shows two insets. The first inset, NG, on the scale of 3 micrometers has large irregular structure with a nucleus in it along with small circular and oval cells in the surrounding. Another inset depicted on the scale of 75 micrometers has normal cells.

Source: (From Nair PNR: Apical periodontitis: a dynamic encounter between root canal infection and host response, *Periodontol 2000* 13:121, 1997.)

In primary acute apical periodontitis, apical bone destruction is usually not observed by conventional periapical radiography because the duration of acute response is short, and activated neutrophilic leukocytes and macrophages are not able to resorb bone. Only osteoclasts are capable of resorbing bone, and they have to differentiate from bone marrow mesenchymal stem cells. However, in a rat model experiment, Stashenko and colleagues[266] showed histologically that periapical inflammatory cell infiltration increases osteoclast numbers, and that bone destruction was apparent well in advance of total pulpal necrosis. Bacteria are usually not present in acute apical periodontitis lesions.

Clinical features

As discussed earlier, there is no correlation between clinical and radiographic findings and histologic appearance of inflammatory periapical disease. Teeth with acute apical periodontitis are usually symptomatic and painful to bite and percussion, which results from mechanical allodynia and hyperalgesia.[99,128] Pain is induced by sensitization and activation of nociceptors of sensory nerve fibers by inflammatory mediators, proinflammatory cytokines, nerve growth factor, and pressure.[99,128] Sprouting of sensory nerve fibers in inflamed periapical tissues could also increase receptive field size in teeth with apical periodontitis.[41,42] If acute

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apical periodontitis is associated with abscess formation, intraoral or extraoral swelling may develop. Radiographic examination usually does not show periapical bone destruction of the involved tooth in acute apical periodontitis, although occasional slight widening of the apical periodontal ligament space and loss of the apical lamina dura of the involved tooth may be present.

Outcomes

The fundamental purpose of the acute inflammatory response is to restore the structural and functional integrity of damaged tissue by eliminating irritants as soon as possible.[141,171,310] Tissue damage can also occur in acute inflammation by release of lysosomal enzymes, toxic oxygen radicals, and nitric acid from disintegrated neutrophilic leukocytes and macrophages into tissue.[171] Depending on the dynamic interaction between host defenses and microbial insults, acute apical periodontitis can result in (1) restitution of normal periapical tissues if irritants are immediately eliminated by root canal therapy, (2) abscess formation if massive invasion of periapical tissues by highly pyogenic pathogens occurs, or (3) progression to chronic apical inflammation if irritants continue to persist.

If purulent exudate in apical abscess can be evacuated through the root canal (or through incision and drainage when indicated) during root canal therapy, the patient usually experiences an immediate relief of acute pain. In most cases, if the source of infection in the root canal is eliminated by root canal therapy, the abscess will heal by reabsorption of the pus by periapical tissues. Phagocytes will kill bacteria in the abscess. The continued influx of leukocytes stops because the chemotactic stimuli have been removed, and the existing neutrophilic leukocytes die of apoptosis. Finally, macrophages move in to clean up necrotic neutrophilic leukocytes and disintegrated tissue cells. However, if bacterial virulence and numbers of pathogens overwhelm the host's defenses, the abscess may break through the cortical bone, periosteum, and oral mucosa or facial skin to develop an intraoral or extraoral draining sinus tract. Sometimes, the uncontrolled abscess may spread along the fascial planes of the face and neck to develop serious cellulitis (see also Chapter 19).[147,240]

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Chronic apical periodontitis

If pathogens in the root canal are not eliminated, the acute apical periodontitis may progress to become a chronic apical periodontitis. Chronic apical periodontitis is characterized by the persistence of inflammatory stimuli, adaptation of the host's response to stimuli, presence of adaptive immune responses, and initiation of the repair process.[141,171,259,310] Chronic inflammation is good news and bad news. The good news is that the host's defenses are able to maintain an active defense against the invading microorganisms and toxins; the bad news is that the host response is inadequate to eliminate these factors.[171]

Chronic apical periodontitis is a form of adaptive immune response that requires exquisite specificity and memory. The adaptive immune response enhances bacterial killing compared with the innate immune response. Traditionally, the terms *chronic apical periodontitis* and *periapical granuloma* are used interchangeably. A granuloma is a focal area of *granulomatous inflammation*, which is a histologic term for a chronic inflammatory reaction.[141,171,305] Granulomatous inflammation is characterized by the presence of activated macrophages with modified epithelioid cells in diseases such as tuberculosis, leprosy, syphilis, cryptococcosis, sarcoidosis, rheumatic fever, and foreign body granuloma.[104,141,171,315] The presence of poorly digestible irritants (nonantigenic or antigenic), T cell–mediated immunity to irritants, or both appears to be necessary for granuloma formation.[1,141,171] A granuloma is relatively avascular,[141,171] whereas a chronic apical periodontitis is very vascular. Histologically, some but not all chronic apical periodontitis lesions may show some features of granulomatous inflammation,[141,171] so the terms *apical granuloma* and *chronic apical periodontitis* should not be used interchangeably. A *granuloma* is best considered a histologic term used to describe a specific form of chronic inflammation such as foreign body granuloma or immune granuloma.[141,171]

The foreign body reaction is a specific subtype of chronic inflammation.[141,171,310] Foreign materials such as root canal filling materials, paper points, cotton fibers, and surgical sutures can trigger a foreign body giant cell granuloma.[136,193,324] If activated macrophages are unable to engulf

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large indigestible foreign particles, they can fuse to form giant cells on the surface of the particles and continuously release lysosomal enzymes, inflammatory mediators, and proinflammatory cytokines as a result of frustrated phagocytosis. Giant cells appear to be at least as active metabolically as a regular macrophage.[171] In addition, foreign bodies can favor infection in several ways, as they can be a source of bacterial biofilm[54,203] and they lower the infectious dose of bacteria to induce infection. For example, if a small, sterile plastic cage is implanted under the skin of a guinea pig, as few as 100 *S. aureus* are sufficient to infect the tissue, whereas even 10^9 bacteria (i.e., a million-fold increase in dose) fail to produce an abscess in normal guinea pig skin.[326] Finally, foreign bodies can make the infection hard to treat because bacteria in biofilms can switch on appropriate genes and cover themselves with a thick layer of biopolymer that is resistant to both host defense mechanisms and antimicrobial agents.[121,171]

Cell biology

Macrophages and lymphocytes

Macrophages and lymphocytes are the primary players in asymptomatic apical periodontitis.[4,55,82,125,135,269,270,291,323] Lymphocytes are blood-borne and have counterparts in the connective tissue.[171] Macrophages play a dual role in host defenses. In the innate immune response, activated macrophages phagocytose microbes, dead cells, and foreign bodies and produce inflammatory mediators and proinflammatory cytokines to enhance the host defense against stimuli. In the adaptive immune response, activated macrophages function as APCs. They phagocytose and present the processed foreign antigens in association with MHC to T cells. Thus, activated macrophages are effector cells of adaptive immune response.

Lymphocytes are the only cells in the body capable of specifically recognizing and distinguishing different antigenic determinants; they are responsible for the two defining characteristics of the adaptive immune response: specificity and memory. The functions of lymphocytes were described earlier in the section on the adaptive immune response.

Dendritic cells

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Dendritic cells play a vital role in asymptomatic apical periodontitis. They have been shown to be present in apical periodontitis lesions in rats.[123,208] Dendritic cells are accessory immune cells derived from bone marrow hematopoietic stem cells and may be related to the mononuclear phagocyte lineage.[1] They function as APC for naive T lymphocytes and are important for the initiation of adaptive immune responses to protein antigen.[1] Activated dendritic cells produce IL-12, which is a key inducer of cell-mediated immunity.

Osteoclasts

Periapical bone destruction is a hallmark of asymptomatic apical periodontitis. During the chronic stage of apical periodontitis, both osteoclast and osteoblast activity decrease,[308] so the periapical osteolytic lesion remains stationary. Based on magnified radiographic and automated image analysis, periapical bone destruction was observed at 7 days after the pulps of experimental teeth were exposed to oral microorganisms in animal studies. A period of rapid bone destruction took place between 10 and 20 days, with slower bone resorption thereafter.[308] The stationary phase of bone resorbing activity was correlated to asymptomatic apical periodontitis.[308] Increased expression of bone resorptive cytokines such as IL-1, IL-6, and TNF was related to the period of active bone resorption.[309] T_H cells appear to outnumber T_S cells during the active stage of periapical bone destruction in induced rat periapical lesions, but T_S cells dominate T_H cells during the stationary stage of bone destruction.[267]

Bone resorption is caused by osteoclasts and involves a cross-talk among osteocytes, osteoblasts, and osteoclasts.[215] The formation of osteoclasts involves differentiation of the osteoclast precursors from mesenchymal stem cells in bone marrow. Several cytokines and growth factors, such as granulocyte/macrophage colony-stimulating factor (GM-CSF), RANKL (receptor activator of nuclear factor κB ligand), osteoprotegerin (OPG), IL-1, IL-6, TNF, as well as prostaglandins, bradykinin, kallidin, and thrombin, have been shown to mediate osteoclast progenitor cell differentiation.[27,43,68,152,157,186,215,216,283] Parathyroid hormone is capable of stimulating osteoblasts to synthesize GM-CSF and RANKL. Bone stromal

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cells and T cells also produce RANKL. Osteoclast progenitor cells express receptor activator of nuclear factor κB (RANK). Osteoprotegerin (OPG), a decoy receptor for RANKL secreted by osteoblasts, negatively regulates the differentiation of osteoclasts by absorbing RANKL and reducing its ability to activate the RANK pathway.[129,215] RANKL activates the RANK pathway on osteoclast progenitor cells, resulting in differentiation of these cells along the osteoclast lineage. Proinflammatory cytokines, IL-1, TNF, and IL-6 also mediate the differentiation of osteoclast progenitor cells to osteoclasts. The differentiation of the mononuclear osteoclast progenitor cells terminates with fusion to latent multinucleated osteoclasts, which are finally activated to become bone-resorbing osteoclasts. The mature osteoclasts then attach to mineralized bone surface after osteoblasts have primed the unmineralized bone surface and released chemotactic factor to attract osteoclasts.[196,215] Osteoclasts attach to bone by the vitronectin receptor (integrin superfamily), expressed preferentially in the sealing zone. Vitronectin has binding sites for the arginine-glycine-aspartic amino acid (RDG) sequences present in many ECM proteins, including osteopontin, bone sialoprotein, and fibronectin, on the surface of the exposed mineralized bone.[84] When bound to bone ECM, osteoclasts develop a ruffled border. Inside the ruffled border, osteoclasts use ATP to drive H^+ pumps, leading to the acidification of the extracellular compartment. They subsequently secrete proteolytic lysosomal enzymes and carbonic anhydrase to degrade both the mineralized and unmineralized components of bone.[18,20,31,283] (See Fig. 16.5 for the mechanism of bone resorption by osteoclasts in apical periodontitis.)

The resorption of root cementum or dentin in apical periodontitis lesions is less well understood than resorption of bone. Bone is constantly remolding (resorption and deposition) through physiologic and functional processes and thus is much easier to study. In contrast, cementum and dentin are more stable. The cells responsible for dental hard-tissue resorption are called *odontoclasts*.[229] It has been shown in ultrastructure and gene expression studies that odontoclasts and osteoclasts are similar.[230,231] Therefore, it is believed that the cellular mechanisms of bone, cementum, and dentin resorption are similar.[231] Nevertheless, little is known about how the precursors of the odontoclasts appear and what causes odontoclast differentiation and activation to resorb dentin and cementum. Bone resorption

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by osteoclasts in apical periodontitis is illustrated in Fig. 16.4.

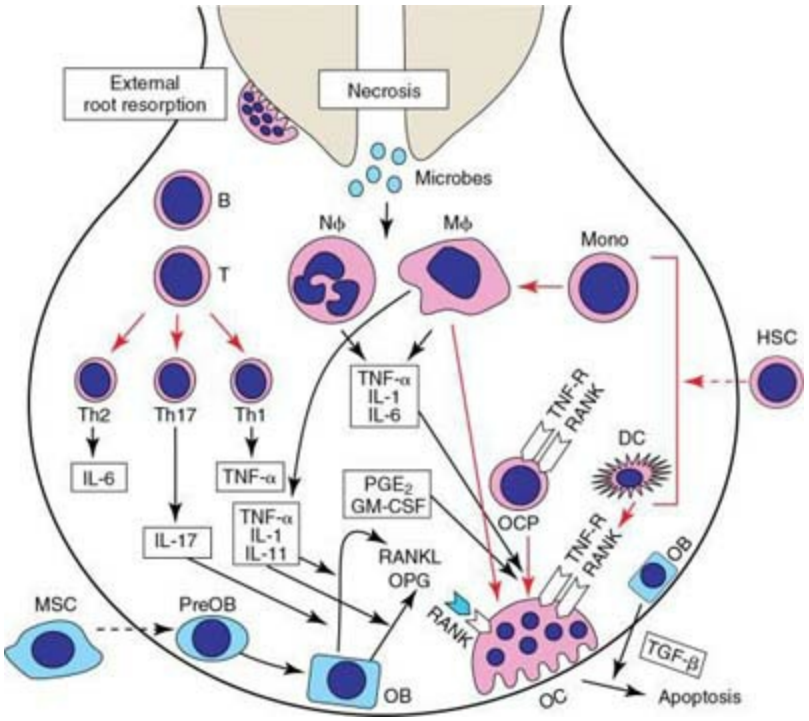

FIG. 16.4 Bone resorption by osteoclasts in apical periodontitis. *DC,* Dendritic cell; *GM-CSF,* granulocyte/monocyte colony-stimulating factor; *HSC,* hematopoietic stem cell; *IL*, interleukin; *Mφ,* macrophage; *MSC,* mesenchymal stromal cell; *Nφ,* neutrophil; *OB,* osteoblast; *OC,* osteoclast; *OCP,* osteoclast precursor; *OPG,* osteoprotegerin; *PGE,* prostaglandin E; *RANK,* receptor activator of nuclear factor κB; *RANKL,* receptor activator of nuclear factor κB ligand; *TGF*-β, transforming growth factor-β; *TNF*, tumor necrosis factor.

The process of bone resorption is as follows:

Microbes from necrosis lead to N phi and M phi, which leads to TNF-alpha, IL-1, IL-6. They along with PGE sub 2, GM-CSF, TNF-R and RANK in OCP as well as DC, lead to OC. OC and OB in the presence of TGF-beta cause apoptosis. HSC leads to mono and DC. Mono further leads to M phi. Two cells on the left below the text external root resorption, B and T, lead to Th2, Th17, and Th1. Th2 further leads to IL-6, Th17 further leads to iL-17, and Th1 leads to TNF-alpha. IL-17 along with TNF-alpha, IL-1, and IL-11 collectively leads to RANKL and OPG from OB. MSC leads to PreOB further leads to OB.

Source: (Courtesy Dr. George T.-J. Huang, TN.)

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Epithelial cell rests of malassez

ERM are present in the periodontal ligament of all teeth. Proliferation of ERM is evident in approximately 52% of inflammatory periapical lesions collected from extracted teeth.[192] It is a form of pathologic (inflammatory) hyperplasia. This type of hyperplasia is caused by stimulation of growth factors and cytokines produced during the inflammatory response. Hyperplasia is a self-limiting process and is reversible when the causative stimulus is eliminated.[141,171,259] During periapical inflammation, innate and adaptive immune cells and stromal cells (e.g., activated macrophages, neutrophilic leukocytes, NK cells, T cells, fibroblasts) in the periapical tissues produce many inflammatory mediators (e.g., prostaglandin, histamine), proinflammatory cytokines (e.g., IL-1, IL-6, TNF), and growth factors (e.g., PDGF, EGF, keratinocyte growth factor [KGF], FGF). These inflammatory mediators, proinflammatory cytokines,[38,49,90,179,233] and growth factors[81,114,165,183,204,287] are capable of stimulating epithelial cell rests to proliferate (Fig. 16.5). The extent of cellular proliferation appears to be related to the degree of inflammatory cell infiltration.[222,223]

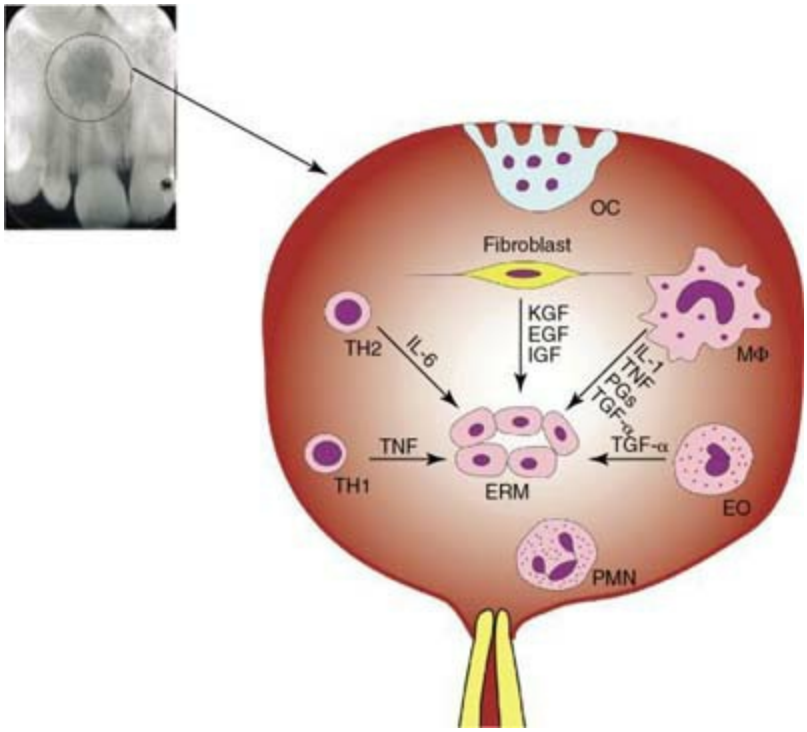

FIG. 16.5 Schematic illustration of the major mechanisms that activate

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proliferation of epithelial cell rests in apical periodontitis. *EGF,* Epidermal growth factor; EO, eosinophil; *ERM*, epithelial cell rests of Malassez; *FGF,* fibroblast growth factor; *KGF*, keratinocyte growth factor; *IGF,* insulin-like growth factor; *IL*, interleukin; *Mφ,* macrophage; *OC,* osteoclast; *PMN,* polymorphonuclear leukocyte; *TGF,* transforming growth factor; *TNF*, tumor necrosis factor.

Radiograph shows a large dark patch under the tooth. An enlarged view of the dark patch has fibroblast under OC. Fibroblast in the presence of KGF, EGF, and IGF leads to ERM. M phi (IL-1, TNF, PGs, TGF), TH2 (IL-6), EO (TGF-alpha), and TH1 (TNF) also lead to ERM. The lower right corner has PMN.

Source: (From Lin LM, Huang GT-J, Rosenberg P: Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after periapical wound healing, *J Endod* 33:908, 2007.)

Fibroblasts

Fibroblasts are important cells in chronic inflammation and wound healing. They are derived from undifferentiated mesenchymal cells and exist in all connective tissues. They synthesize and secret proteoglycans, glycoproteins, and precursor molecules of various types of collagens and elastin.[126] In chronic inflammation, fibroblast migration and proliferation are triggered by multiple growth factors (TGF-β, PDGF, EGF, FGF) and the fibrogenic cytokines, IL-1 and TGF-α, produced by activated platelets, macrophages, endothelial cells, and inflammatory cells.[141,171] In turn, activated fibroblasts produce an array of cytokines such as IL-1, IL-6, and GM-CSF, which influence leukocyte development. Fibroblasts stimulated by inflammation also produce matrix metalloproteinase to degrade the proteins that comprise the ECM.[171,298]

Fibrovascular granulation tissue is also a prominent feature of chronic apical periodontitis as a repair process. Migration and proliferation of endothelial cells from preexisting capillaries and venules into the site of chronic inflammation is called *neovascularization*. It is mediated by angiogenesis factors, such as vascular endothelial growth factor (VEGF) and TGF-β, produced by activated macrophages, platelets, and endothelium.[171,259] Neovascularization supplies oxygen and nutrients for the support of metabolically active macrophages and fibroblasts during wound healing.

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Inflammatory mediators

Many inflammatory mediators present in symptomatic apical periodontitis are also expressed in asymptomatic apical periodontitis. In addition, several different cytokines and growth factors are secreted by cells such as activated macrophages, lymphocytes, dendritic cells, and fibroblasts, in the chronic/adaptive inflammatory response described earlier.

Histopathology

Macrophages and lymphocytes are predominant cells in asymptomatic apical periodontitis lesions (Fig. 16.6). Occasionally, foamy macrophages and giant cells are seen, especially associated with cholesterol crystal deposits, which are products of disintegrated cell membranes. Cholesterol crystals are present in approximately 18% to 44% of all apical periodontitis lesions.[36,297] Bone resorption is a hallmark of asymptomatic apical periodontitis. Multinucleated osteoclasts may sometimes be seen in resorptive Howship lacunae. Proliferation of epithelial cell rests is often present in asymptomatic apical periodontitis lesions. ERM proliferate in three dimensions and form irregular strands or islands of epithelium, which are often infiltrated by varying degrees of inflammatory cells (see Fig. 16.6, *B*). A key feature of asymptomatic apical periodontitis is a proliferation of fibrovascular granulation tissue that is an attempt to prevent further spread of infection/inflammation and to repair wounded periapical tissues.

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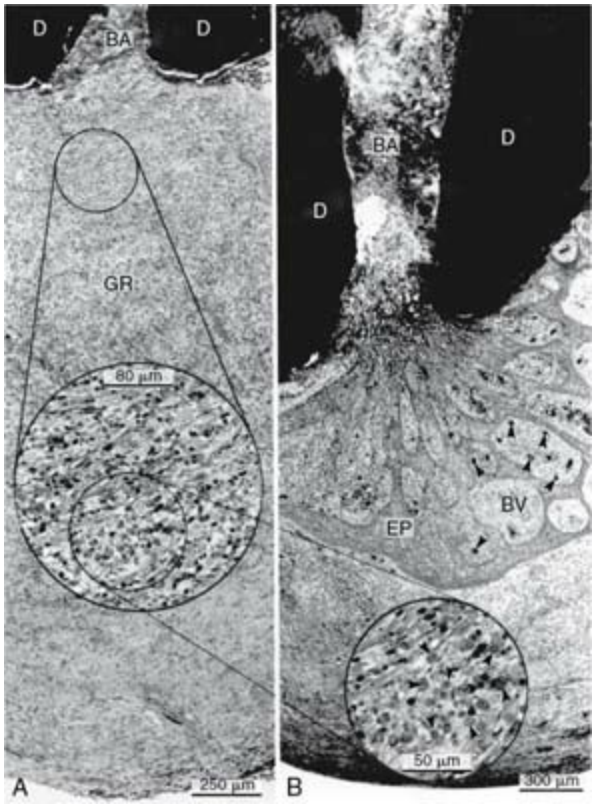

FIG. 16.6 Chronic asymptomatic apical periodontitis without **(A)** and with **(B)** epithelium *(EP)*. The root canal contains bacteria *(BA)* in **A** and **B.** The lesion in **A** has no acute inflammatory cells, even at the mouth of the root canal with visible bacteria at the apical foramen *(BA)*. Note the collagen-rich maturing granulation tissue *(GR)* infiltrated with plasma cells and lymphocytes (inset in **A** and **B**). *BV,* Blood vessels; *D,* dentin. (**A,** Magnification: ×80; **B,** magnification: ×60; inset in **A,** magnification: ×250; inset in **B,** magnification: ×400.)

Two electron micrographs are marked A and B.

A) The labels read, D, BA, and GR. An inset of 80 micrometers contains fluid along with ground tissue, dark, and slightly light spots. The micrograph is depicted on the scale of 250 micrometers.

B) The labels read, BA, D, EP, and BV. An inset of 50 micrometers from the micrograph A shows several arrow heads pointing toward nearly less-dense oval-shaped cells. The micrograph is depicted on the scale of 300 micrometers.

Source: (From Nair PNR: Apical periodontitis: a dynamic encounter between root canal infection and host response, *Periodontol 2000* 13:121, 1997.)

Little information is available concerning cemental changes in apical

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periodontitis. Using a scanning electron microscope, Simon and associates[252] observed that projections, depressions, and fibers of cementum were haphazardly arranged in root canal infection under scanning electron microscopy. There was an increase of mineralized projections, cementum lacunae, and surface resorptions and a decrease in fibers. All these changes may provide a more favorable condition for attachment of bacterial biofilm on the apical external root surface in extraradicular infection.

It is a general belief that asymptomatic apical periodontitis lesions are usually devoid of innervation, so local anesthesia may not be necessary if teeth with asymptomatic apical periodontitis require root canal therapy. However, light and transmission electron microscopic studies have demonstrated that teeth with chronic apical periodontitis were well innervated (Fig. 16.7).[158,174] This explains why instruments accidentally introduced into inflamed periapical tissues without local anesthesia can cause the patient pain.

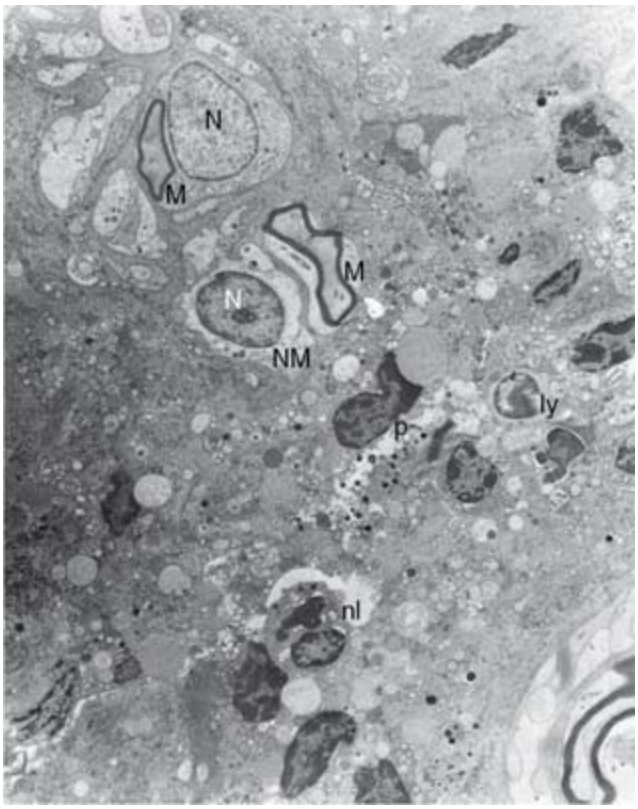

FIG. 16.7 Presence of intact myelinated and nonmyelinated nerve fibers in chronic apical periodontitis lesion. *ly,* Lymphocyte; *M,*

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myelinated nerve fiber; *N,* nucleus of Schwann cell; *nl,* neutrophilic leukocyte; *NM,* nonmyelinated nerve fiber; *p,* plasma cell. (Electron micrograph, magnification: ×1600.)

Electron micrograph shows nearly oval nuclei surrounded by nonmyelinated nerve fibers at the base and myelinated nerve fibers on the right. Plasma cells present nearby circular lymphocyte and white patches of neutrophilic leukocyte.

Source: (From Lin L, Langeland K: Innervation of inflammatory periapical lesions, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 51:535, 1981.)

External root resorption that involves cementum or both cementum and dentin usually occurs in asymptomatic apical periodontitis lesions.[148,303] Fortunately, cementum and dentin appear to be less readily resorbed than bone by inflammatory processes.[95] However, the radiographic presence of root "blunting" should be interpreted by the astute clinician as evidence for resorption of dentin and cementum, and the working length of the instrumentation and obturation phases of nonsurgical endodontic treatment should be adjusted by a corresponding amount.

Using light and transmission electron microscopy and microbiologic culturing, bacteria have been shown to be present in many asymptomatic apical periodontitis lesions.[2,117,296,311] Inadvertent contamination is a major concern when culturing bacteria from these lesions. Under meticulous transmission electron microscopic examination, Nair[189] was unable to observe bacteria in most asymptomatic apical periodontitis lesions. If bacteria are present in the inflamed periapical tissues, they are usually found inside the phagocytes (Fig. 16.8).[159] Importantly, the mere presence of bacteria in the inflamed periapical tissues (colonization) does not necessarily imply periapical infection. Bacteria have to be able to establish an infectious process, such as survival and tissue destruction, in the inflamed periapical tissues to be considered as etiologic factors for the resulting infection. Most apical periodontitis lesions are not infected; nonsurgical root canal therapy of teeth with apical periodontitis can achieve a high success rate, provided root canal infection is controlled.

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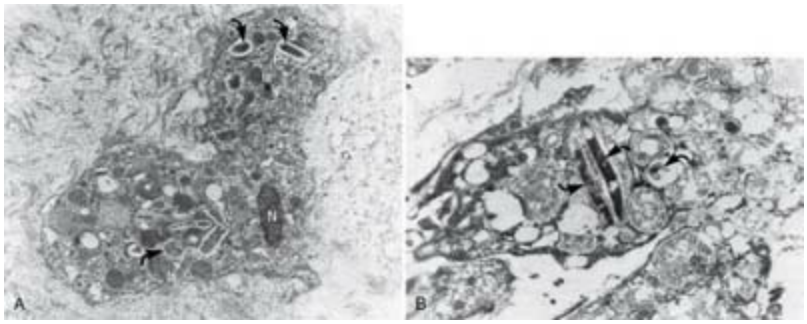

FIG. 16.8 Bacteria phagocytosed by phagocytes in chronic apical periodontitis lesion. **A,** Bacteria *(arrows)* in phagosome of a neutrophil. *Is,* Lysosome; *N,* part of nucleus; *Pv,* phagocytic vacuole. (EM, magnification: ×6300.) **B,** Part of the cell structure of a macrophage. Note longitudinally and cross-cut bacteria cells with unit membrane *(arrows)* in phagocytic vacuoles. (EM, magnification: ×10,000).

Two electron micrographs are marked A and B.

A) Phagocytic vacuole has an elongated oval shape with a light periphery. Part of nucleus is shown as oval-shaped cell, whereas lysosome shows dark circular patches.

B) Arrows from oval structures point toward longitudinal cells and an arrow from small dark patch point toward inverted U-shaped structure with dark patch at the base.

Source: (From Lin L, Langeland K: Light and electron microscopic study of teeth with carious pulp exposures, *Oral Surg Oral Med Oral Pathol* 51:292, 1981.)

Clinical features

Involved teeth are usually asymptomatic and show an ill-defined or well-defined periapical radiolucent lesion radiographically. Chronic apical periodontitis lesion with abscess formation always shows a draining sinus tract. Occasionally, the sinus tract from an apical abscess may drain along the root surface and opens into the gingival sulcus. This leads to the development of a deep, narrow pseudopocket that often mimics a periodontal pocket or a vertical root fracture (see Chapter 25 for details).

Outcomes

Chronic apical periodontitis may result in (1) regeneration or repair of the periapical tissues after root canal therapy, (2) acute exacerbation, (3)

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development of an abscess with an intraoral or extraoral draining sinus tract, or (4) development of a serious cellulitis.

Chronic apical periodontitis with cyst formation

The radicular cyst is unique because no cysts in the body have similar pathogenesis. It is believed that the radicular cyst is likely formed by inflammatory proliferation of epithelial cell rests in the inflamed periodontal ligament in chronic apical periodontitis lesions.[163,195,206,285] The radicular cyst is a pathologic cavity completely lined by nonkeratinized stratified squamous epithelium of variable thickness in a three-dimensional structure in an apical periodontitis lesion. A radicular cyst can be a “pocket cyst” (i.e., attached to the apical foramen) or a “true cyst” (no attachment to the root structure),[191,251] but it cannot form by itself. Therefore, a radicular cyst should not be considered a separate disease entity from chronic apical periodontitis. A radicular cyst (pocket or true) is classified as an inflammatory and not an abnormal developmental or a neoplastic lesion in the WHO *Histological Typing of Odontogenic Tumors, Jaw Cysts and Allied Lesions*.[139] The prevalence of radicular cysts in apical periodontitis lesions from extracted teeth ranges from 15% to 20%.[192]

Cell biology

In addition to the presence of all chronic inflammatory cells, the epithelial cells are the most prominent cell type found in chronic apical periodontitis with cyst formation. ERM can be considered unipotent or restricted-potential stem cells. They can be stimulated to divide symmetrically and asymmetrically into basal cells (stem cells) and suprabasal squamous cells of lining epithelium of radicular cysts.[163] Many theories about apical cyst formation have been proposed. The nutritional deficiency theory assumes that when islands of epithelium keep growing, the central cells of the epithelial island will move farther away from their source of nutritional supply and undergo necrosis and liquefaction degeneration. The products accumulated attract neutrophils and granulocytes into the necrotic area. Microcavities then

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coalesce to form a cyst cavity lined by stratified squamous epithelium.[285] The abscess theory postulates that when an abscess is formed in connective tissue, epithelial cells proliferate and line the abscess cavity because of their inherent tendency to cover exposed connective-tissue surface.[195,206] The theory of merging of epithelial strands proposes that proliferating epithelial strands merge in all directions to form a three-dimensional sphere-like structure composed of fibrovascular connective tissue, with varying degrees of inflammatory cells trapped, that gradually degenerates due to lack of blood supply. This leads to formation of a cyst cavity.[163] Regardless of how a radicular cyst (pocket or true) is formed, it is likely caused by inflammatory proliferation (hyperplasia) of epithelial cell rests in the apical periodontitis lesions.

It has been speculated that radicular cyst expansion is caused by increased osmotic pressure in the cyst cavity,[288] but this hypothesis overlooks the cellular aspects of cyst growth and the biochemistry of bone destruction.[75,179] Radicular cyst expansion is likely caused by degradation of fibrous connective tissue capsule by MMPs, which are produced by activated neutrophils, fibroblasts, and macrophages[286] and bone resorption. The ERM have also been shown to be capable of secreting a bone resorbing factor.[28] Most inflammatory mediators and proinflammatory cytokines that stimulate proliferation of epithelial cell rests also mediate bone resorption in apical periodontitis lesions.

Inflammatory mediators

Mediators are basically similar to those present in chronic apical periodontitis.

Histopathology

Two types of cysts have been described in chronic apical periodontitis lesions. The pocket cyst's lumen opens into the root canal of the involved tooth (Fig. 16.9).[191,192,251] The true cyst is completely enclosed by a lining epithelium, and its lumen has no communication with the root canal of the involved tooth (Fig. 16.10).[191,192,251] Apical cysts are lined by hyperplastic, nonkeratinized stratified squamous epithelium of variable thickness, which is

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separated from the fibrovascular connective tissue capsule by a basement membrane. Both the lining epithelium and the connective tissue capsule are usually infiltrated with inflammatory cells,[224,245] indicating that inflammatory cells are attracted to these tissues by chemotactic irritants, either in the root canal system or in the periapical tissues. In nonproliferating epithelium, there is less inflammatory cell infiltration.[47,240] Occasionally, mucous cell metaplasia or ciliated respiratory-type cells are present in cystic epithelial lining.[201] The epithelial cells of lining epithelium do not show any characteristic features of neoplastic changes, such as pleomorphism, lack of polarity, nuclear enlargement, large nucleoli, abnormal nuclear/cytoplasmic ratio, hyperchromatism, or abnormal mitosis. Unlike odontogenic keratocysts and calcifying odontogenic cysts, the basal cells of radicular cystic lining epithelium are incapable of proliferation by themselves without stimulation of growth factors or cytokines released by innate and adaptive immune cells during periapical inflammation. The lumen of cysts may contain inflammatory exudates, cholesterol crystals, clear fluid, or bacterial colonies (Figs. 16.11 and 16.12).[192,224]

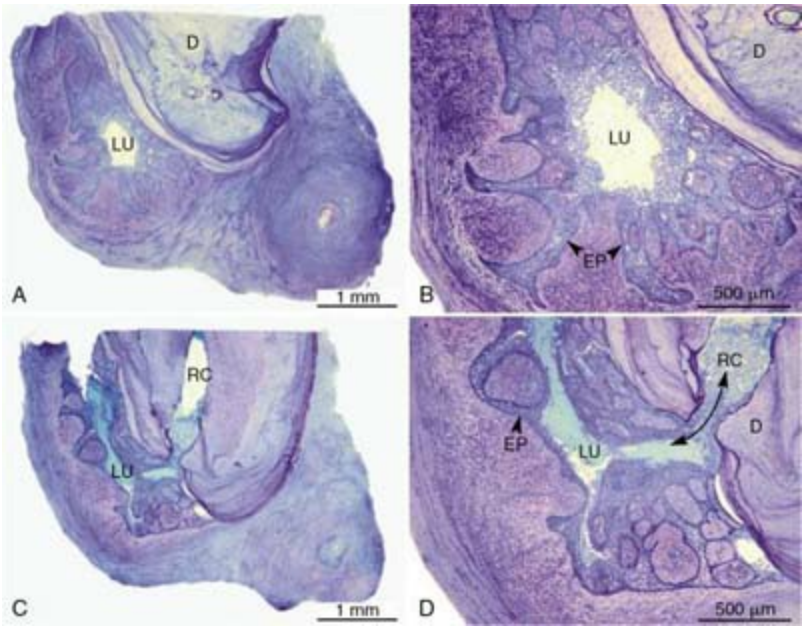

FIG. 16.9 **A,** A well-developed pocket cyst in apical periodontitis lesion. **B,** Note the saclike epithelial lumen. **C** and **D,** Sequential axial serial section passing through the apical foramen in the root canal plane *(RC)* shows the continuity of the lumen with the root canal. *D,*

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Dentin; *EP,* epithelium; *LU,* Lumen. (**A** and **C,** Magnification: ×16; **B** and **D,** magnification: ×40.)

Four stained micrographs marked A through D.

A) The micrograph depicted on the scale of 1 millimeters shows lumen on the left side of root apex.

B) The micrograph depicted on the scale of 500 micrometers has large lumen cavity surrounded by well-defined EP cells.

C) The micrograph depicted on the scale of 1 millimeter has lumen cavity filled with tissue of root canal. EP cells are diffused.

D) The micrograph depicted on the scale of 500 micrometers has a double-headed curved arrow between RC and lumen. A roughly circular structure is marked EP above the lumen.

Source: (From Nair PNR: Non-microbial etiology: foreign body reaction maintaining post-treatment apical periodontitis, *Endod Topics* 6:96, 2003.)

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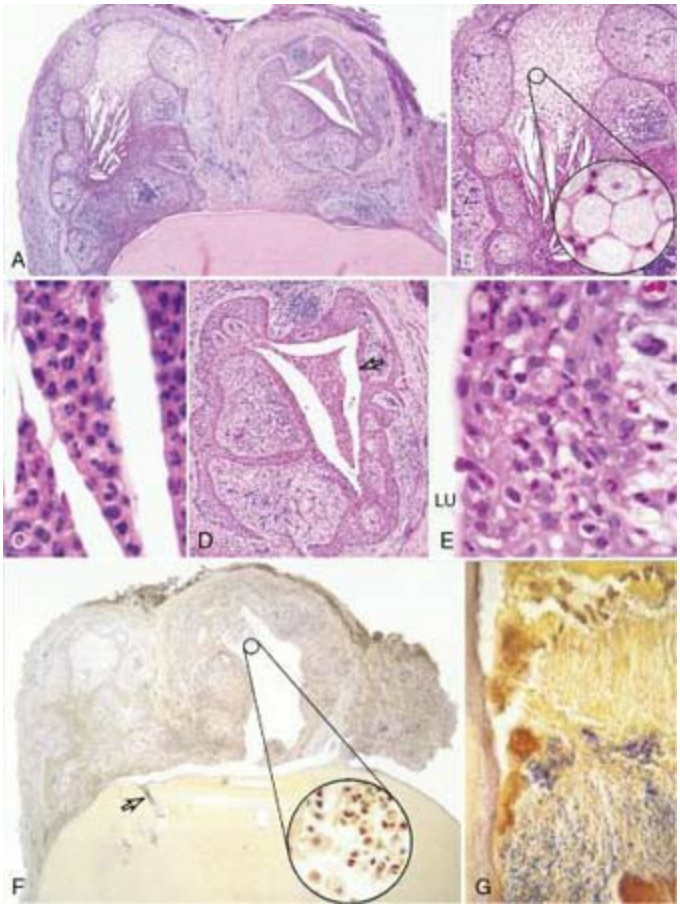

FIG. 16.10 **A,** Two distinct cysts lined by epithelium in apical periodontitis lesion. There is no evidence of communication with the foramen in serial sections (H&E, magnification: ×25). **B,** Cyst cavity on the left side in **A.** Accumulation of foamy macrophages (H&E, magnification: ×50; inset magnification: ×1000). **C,** Lower part of cavity in **B.** Dense infiltration of neutrophilic leukocytes (H&E, magnification: ×1000). **D,** Cyst cavity on the right side in **A.** Necrotic tissue debris in the lumen; infiltration of inflammatory cells in the epithelium (H&E, magnification: ×50). **E,** Cystic wall in **D.** Cyst's lining epithelium is infiltrated with acute and chronic inflammatory cells (H&E, magnification: ×1000). **F,** Foamy macrophages, neutrophilic leukocytes, and necrotic tissue inside the cyst *(inset)*, but no bacteria (Brown & Brenn, magnification: ×25, inset magnification: ×1000). **G,** Area indicated by an *open arrow* in **F.** Bacterial colonies in the apical foramen (Brown & Brenn, magnification: ×1000). *LU,* Lumen.

Seven stained micrographs marked A through G.

A) It shows vertical white strips below the cavity on the left and a triangular white patch on the right.

B) The inset of cavity on the left shows circular cells with ground tissue. One cell has a nucleus inside.

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C) Two vertical strips are shown.

D) An arrow points toward the enlarged triangular structure with dense tissue inside.

E) The wall of lumen cavity shows nearly oval and dark patches surrounded by white periphery and less-density fluid on the right.

F) An arrow points toward vertical invagination. An inset shows fatty plaques.

G) It shows bacterial colonies entering the open root canal in tooth.

Source: (From Ricucci D, Pascon EA, Pitt Ford TR, Langeland K: Epithelium and bacteria in periapical lesions, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 101:241, 2006.)

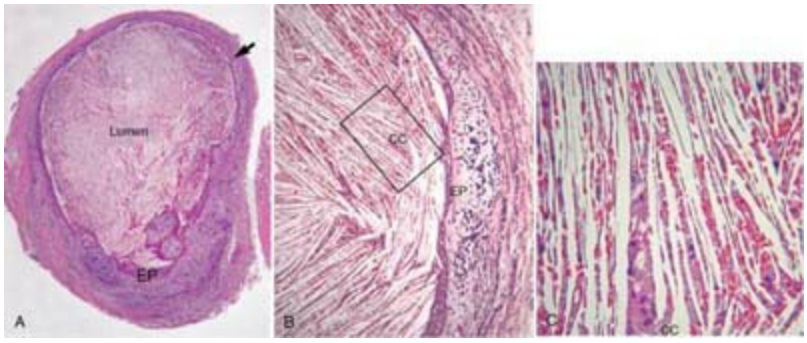

FIG. 16.11 **A,** A radicular cyst in which the lumen is completely filled with cholesterol crystals *(CC)* (H&E, magnification: ×25). **B,** High magnification of cholesterol crystals in **A** (H&E, magnification: ×100). **C,** High magnification of rectangular area in **B.** Multinucleated giant cells associated with cholesterol crystals (H&E, magnification: ×400). *EP,* Epithelium.

Three stained micrographs are marked A through C.

A) Large lumen cavity is shown with an oval and a circular structure at the base.

B) The condensed needle shaped cholesterol crystal cells near epithelium.

C) The enlarged view of cholesterol crystals shows the empty spaces between cholesterol crystal cells.

Source: (Courtesy Dr. Domenico Ricucci, Rome, Italy.)

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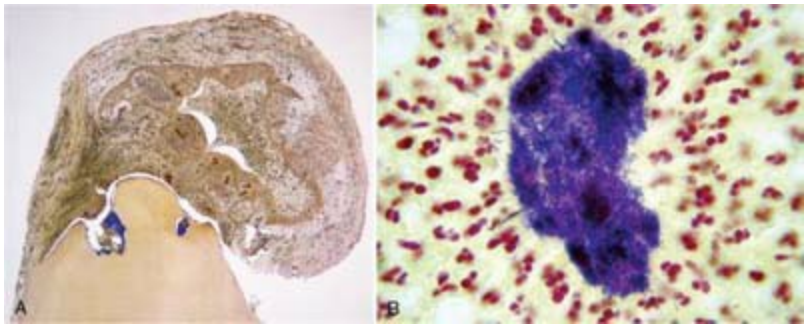

FIG. 16.12 **A,** An apical true cyst in apical periodontitis lesion. Bacterial colonies are seen in the foraminal areas (Brown & Brenn, magnification: ×25). **B,** Bacterial colonies in the cyst lumen, surrounded by inflammatory cells (Brown & Brenn, magnification: ×400).

A) Microscopic image shows a sac-like pocket near the root apex.

B) Microscopic image shows dark patches in the pocket.

Source: (From Ricucci D, Bergenholtz G: Histologic features of apical periodontitis in human biopsies, *Endod Topics* 8:68, 2004, Fig. 6.)

Clinical features

The involved tooth is usually asymptomatic. Periapical osteolytic lesions of endodontically involved teeth may sometimes show a well-demarcated radiolucency surrounded by a rim of radiopaque border radiographically.

Outcomes

There is no direct evidence demonstrating whether chronic apical periodontitis with cyst formation (pocket and true) can or cannot regress after nonsurgical root canal therapy. Unfortunately, apical cysts cannot be diagnosed clinically and can only be diagnosed after surgical biopsy or extraction of teeth with apical periodontitis. Clinical outcome studies have shown that after proper nonsurgical root canal therapy, teeth with apical periodontitis lesions heal in 78% of cases.[258] Therefore, it is speculated that some cysts, especially pocket cysts, may heal after nonsurgical root canal therapy.[191] Apical true cysts are less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature; surgical intervention is necessary.[191] Nonetheless, similar to the periapical pocket cyst, a periapical true cyst is also formed within an apical periodontitis lesion and is not a

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neoplastic lesion. Any disease caused by inflammation/infection should be able to heal if the causative irritant/irritants are removed, unless the irritant/irritants are neoplasm-inducing agents or a carcinogen.

Chronic apical periodontitis with reactive bone formation: Focal condensing osteitis

Chronic apical periodontitis with reactive bone formation is analogous to chronic osteomyelitis with proliferative periostitis. The etiology and pathogenesis of these two diseases are not well understood. It is generally believed that both lesions are caused by a long-term, low-grade inflammation/infection or a high resistance of local tissue to inflammation/infection.[201,218] Instead of bone resorption, the inflammation induces reactive bone formation of alveolar trabecular or spongy bone around the periapex of endodontically involved teeth.

Cell biology

As described previously, during the chronic stage of apical periodontitis, both osteoclast and osteoblast activity decrease.[308] However, in chronic apical periodontitis with reactive bone formation, osteoblasts appear to be stimulated to produce more bone. It is not clear what factor/factors stimulate osteoblasts to produce more bone mass. It could be due to increased expression of growth factors/cytokines, such as TGF-β, BMP, PDGF, and transcription factor Cbfa1 (core-binding factor family).[129]

Histopathology

There is an excessive apposition of bone mass without bone resorption in the apical area. As the bone marrow spaces become smaller and obliterated, the bone resembles compact bone that is infiltrated by a small number of lymphocytes. The compact bone has few lacunae, and many of them are empty of osteocytes. It has many prominent resting and reversal lines, similar to an idiopathic osteosclerosis or a Pagetoid appearance.[47,201,218]

Clinical features

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The lesion is usually observed in young patients, and the mandibular first molar is most commonly involved. The teeth often have gross carious lesions and can be vital or nonvital. They are usually asymptomatic. Radiographically, the lesion may have a well-defined or ill-defined radiopaque mass associated with the apex of an endodontically involved tooth. The lamina dura around the root apex is usually intact.

Outcomes

Most chronic apical periodontitis lesions with reactive bone formation demonstrate healing after nonsurgical root canal therapy. The excessive compact bone in most cases will be remodeled to normal appearance.[72]

Periapical lesions of nonendodontic origin

Many periapical lesions are not of endodontic origin and should be considered in the differential diagnosis of apical periodontitis. These lesions include (but are not limited to) trauma,[11,12] foreign bodies,[136,193,324] host metabolic by-products,[194] advanced periodontal disease,[146] fibro-osseous lesions, and benign and malignant tumors.[201] A complete description of these lesions is beyond the scope of this chapter; the interested reader is encouraged to seek the cited references.

Extraradicular endodontic infection

Extraradicular endodontic infection implies that bacteria have established an infectious process outside the root canal system in the periapical area.[257,271,296] Extraradicular endodontic infection can only be diagnosed after surgical biopsy or microbiologic sampling or molecular detection of therapy-resistant periapical lesions during apical surgery. The method of sample collection should be carefully reviewed, because contamination is a problem in bacteriologic study of apical periodontitis lesions during apical surgery.

Extraradicular endodontic infection can be a part of intraradicular infection or an independent entity without an intraradicular component. To be considered an independent extraradicular infection, the presence of pathogens

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in the root canal system must be completely ruled out, because intraradicular bacteria pose more potential problems than extraradicular bacteria in periapical inflammation. Bacteria in the complex root canal system are well protected from root canal treatment procedures (i.e., mechanical instrumentation, antiseptic irrigation, intracanal medication), host defenses, and antimicrobial therapy because of a lack of blood circulation and areas resistant to access (e.g., isthmi; see Chapter 8). The independent extraradicular infection probably does not occur often and is usually associated with apical actinomycosis.[98,277] Bacteria have been observed as biofilm on the apical external root surface.[257,295] In addition, viruses have also been isolated from apical periodontitis lesions.[154,228,260] If microbiologic root canal cultures are not completely reliable (because bacteria may remain in the complex root canal system),[232] then interpretation of independent extraradicular infections as endodontic treatment failures should be carefully evaluated.

Although virulence and number of pathogens, and the host's immune defense mechanisms. determine whether extraradicular infection will develop or not, the development of extraradicular infection is still not fully understood. In order for planktonic pathogens to establish infection in the periapical tissues, most likely the host's immune defense mechanisms have to be compromised. What are the predisposing factors to extraradicular infection? How are planktonic bacterial cells able to evade the host's powerful defenses in order to colonize and establish an infectious process in the periapical tissues? Are all extraradicular infections resistant to nonsurgical root canal therapy? The preponderance of evidence does not support the consistent presence of an independent periapical infection in endodontic infections.

Apical periodontitis and systemic diseases

Information concerning apical periodontitis and systemic diseases is limited. Clinical and radiographic studies have shown that there is a greater prevalence of periapical lesions in diabetics than in nondiabetics.[24] Type 2 diabetes mellitus (DM) is significantly associated with an increased prevalence of apical periodontitis.[239] Other findings demonstrated that type 2

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diabetes is associated with increased risk of a poor response by the periradicular tissues to odontogenic pathogens.[34] In rodent models, diabetic conditions enhance the development of periradicular lesions and increase mortality.[76,116,134] Mice deficient in P/E-selectin are shown to be more prone to infection-mediated early-onset periodontal disease.[202]

Genetic polymorphisms may also play a role in the individual's response to various diseases. Haplotype analyses have indicated that a variant of the IL-1β gene was likely to be most important for early-onset periodontitis risk.[65] In a Chinese male population, it was reported that the polymorphisms of *IL1A*+4845 and *IL1B*-511 might play an important role in determining generalized aggressive periodontitis susceptibility. A possible combined effect of the polymorphism of *IL1B*-511 and smoking on this type of periodontitis susceptibility was suggested.[155] In a study on patients from Chile, the heterozygous inheritance of the *IL1B*+3954 gene was found significantly higher in periodontitis cases than in control patients. The prevalence of a positive genotype (at least one allele of the two variants present at each locus) is significantly higher in cases (26%) than in controls (10%) and significantly associated with periodontitis, irrespective of smoking status and periodontitis severity.[167] Those displaying high-producer *IL6* genotype, intermediate and high-producer *IL1B* genotypes, and low-producer *TNFA* genotype tend to exhibit symptomatic dental abscesses.[56]

Apical periodontitis was once considered a focus of infection, and the microorganisms from the focus could disseminate through blood circulation to remote parts of the body, where secondary disease occurred.[111] Even though bacteremias do occur during root canal treatment of teeth with apical periodontitis when instrumentation is carried through the apical foramen into the periapical tissues, the incidence and magnitude of bacteremias is not clinically significant for a healthy person and in fact appears to be lower than that observed by flossing.[22,61,62] Microorganisms in the blood circulation are rapidly eliminated by the host's humoral and cellular defense components within minutes.[22,62] However, bacteremia may pose a potential danger to immunocompromised patients or patients with congenital heart valve disease.[61] No good correlation between apical periodontitis and coronal heart disease and rheumatoid arthritis has been observed.[44,187] There is insufficient evidence to support that apical periodontitis could serve as a focus of

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infection and cause significant systemic diseases.[71,210]

Genetic and systemic disease risk factors of persistent apical periodontitis

Genetic risk factors

Genetic polymorphism in the population plays an important role in the variation of the susceptibility and development of diseases among individuals. Knowledge on the polymorphism of key genes that are involved in disease development will help us to understand and establish prevention or treatment strategies for the diseases. Because of the completion of the Human Genome Project, many advanced molecular technologies have been developed including high-throughput screening and sequencing, which further help gather the information on polymorphism in human populations. There are different types and causes of genetic polymorphisms. Single nucleotide polymorphisms (SNPs) that have a single base variation resulting from insertion or deletion of a base are considered the most common type of genetic polymorphism.[45]

Occurrence of SNP within the coding region of a gene may produce an altered protein, which may lead to altered function (termed functional SNPs). Occurrence of SNP within the promoter region of a gene may alter gene regulation, which may cause reduction/inhibition or overexpression of gene expression. Functional SNPs alter amino acid sequence or interfere with the transcriptional factor binding, whereas nonfunctional SNPs do not affect the regulation of protein expression. Many SNPs have no effect on cell function, but some could predispose people to disease or influence their response to drugs. Besides SNPs, there are single amino-acid polymorphisms (SAAPs or SAPs) of proteins, which may result from RNA editing independent of SNPs.[319]

A genetic polymorphism is defined as alleles or variants that appear in at least 1% of the population. The 1% cutoff excludes mutations that may have occurred in a single family. In most polymorphisms, one allele dominates and appears with < 99% frequency (for example, 65%) in the population. In a biallelic situation, some authors term the dominant allele as normal (N-allele

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or allele 1) and it occurs in < 99%, whereas the rarer allele (R-allele or allele 2) is present in >1% in the population.[237,256] It has been long known that genetic polymorphisms are associated with increased severity of inflammatory diseases.[101] Both endodontic and periodontic diseases are mainly an infectious disease caused by microbial infection. They share common features in terms of inflammation and bone resorption. Studies have implicated association of periodontitis or apical periodontitis with several gene polymorphisms such as *IL-1, IL-6, IL-8, Fc*γR, TNF receptor superfamily member 1B (*TNFRSF1B*), (selenoprotein S) *SEPS1*, and *MMPs*.[8,48,132,137,180,181,320]

It has long been observed that the proinflammatory cytokine interleukin-1 *(IL-1)* genotype is a severity factor in adult periodontal disease.[137] The specific periodontitis-associated IL-1 genotype has a variant in the *IL-1B* gene that is associated with high levels of IL-1 production. Gene polymorphism studies suggest that the frequency of *IL-1*β genotypes (*IL-1*β +3953 restriction fragment length biallelic polymorphism) is increased in patients with advanced adult periodontitis compared to early and moderate combined, and to healthy controls.[87] A more recent study showed that IL-1β levels in gingival crevicular fluid is increased with severity of disease and correlates well with clinical signs of incipient disease.[265] This suggests that some individuals, when challenged by bacterial accumulations, may respond with a higher level of inflammatory activities, therefore leading to more severe periodontitis. Both FcγRIIa (CD32) and FcγRIIIb (CD16) influence polymorphonuclear leukocyte phagocytic function and therefore their polymorphisms may also influence inflammatory disease development. It was reported that the *Fc*γR IIIb-NA2 allotype represents a risk factor for recurrence of adult periodontitis,[132] and the *Fc*γRIIIa-158V allele and possibly *Fc*γRIIIb-NA2 may be associated with severity of chronic periodontitis in a Japanese population,[133] whereas the *Fc*γRIIa-H/H131 genotype may be associated with chronic periodontitis risk (and disease severity) in Caucasian smokers.[320] *Fc*γRIIIa and *Fc*γRIIIb genotypes may impose an additional risk of periodontal bone loss in a German population.[180]

The polymorphisms of two other genes involved in inflammation, *SEPS1* and *TNFRSF1B*, have been found to be involved in determining host individual susceptibility to aggressive periodontitis, and there is the potential

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association between *IL-6* and *Fc*γR polymorphisms and the aggressive periodontitis in a Caucasian-Italian population.[234]

The genetic polymorphisms have been involved in the development of apical periodontitis reports. Two genetic conditions—carriage of allele H131 of the *Fc*γRIIa gene and a combination of this allele with allele NA2 of the *Fc*γRIIIb gene—have been reported to be associated with posttreatment apical periodontitis,[256] whereas polymorphism in the *Fc*γRIIIa does not influence the patient's response to endodontic treatment of teeth with apical periodontitis in a Brazilian population.[255] So far there has been only one report on *IL-1* polymorphism association with apical periodontitis in an American population.[185] In this study, patients with persistent apical periodontitis showed an increased prevalence in the genotype composed of allele 2 of *IL-1*β polymorphism when compared with those who experienced complete healing after acceptable root canal therapy (70.6% vs. 24.6%). This outcome suggests that specific genetic markers associated with IL-1β production may predict increased susceptibility to persistent apical periodontitis. An earlier paper that studied a Brazilian population did not find an association between IL1 polymorphism and persistent apical periodontitis; however, allele 1 (for *IL-1A* and *IL-1B*) was always detected higher in diseased than in healthy/healing individuals, although not statistically significant.[256] The lack of statistical difference could be due to small sample size or different population pools.

The polymorphism of the IL-8 gene, a potent chemokine that attracts neutrophils to the site of infection, has also been shown to influence the development of different forms of apical periodontitis.[8] *IL8/CXCL8-251 T* allele, which is associated with higher production of IL8/CXCL8, is also associated with a higher risk of developing acute suppurative form of apical periodontitis. *IL8/CXCL8-251 A* allele, which is associated with lower production of IL8/CXCL8, is associated with the chronic nonsuppurative form of apical periodontitis. SNP of other inflammation-associated genes, MMP2 and MMP3, also show influence on periapical lesion formation.[181] Thus, markers in MMP3 and MMP2 genes could also help predict host susceptibility to developing periapical lesions and the healing response. It is clear that genetic polymorphisms are involved in pathogenesis of apical periodontitis.[140]

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Systemic disease risk factors

Many systemic diseases have been linked to periodontal disease[130,205] because they contribute to either a decreased host's resistance to infection or dysfunction in the connective tissue of periodontium, increasing patient susceptibility to immuno-inflammation-induced destruction.[16] However, the similar systemic diseases do not appear to link to apical periodontitis, possibly because periodontitis is an open lesion and apical periodontitis is a closed lesion.

DM, a systemic disease, has been well documented to be associated with a high prevalence of apical periodontitis.[24,77,116,168,173,238] DM is a chronic disorder of carbohydrate, fat, and protein metabolism, and a defective or deficient insulin secretory response.[141] Hyperglycemia is characteristic of DM and has profound effects on cell metabolisms, especially endothelial cells such as increased polyol pathway flux, increased formation of advanced glycation end products, hyperglycemia-induced activation of protein kinase C, and increased hexosamine pathway flux.[37] The tissue most frequently affected by hyperglycemia is microvasculature, whereby endothelial cells are not able to transport intracellular glucose effectively.[37] Because of damage to vasculature (atherosclerosis), blood circulation, innate and adaptive immuno-inflammatory mechanism, and phagocyte function are impaired,[37,141] thus leading to pulp necrosis and predisposition to pulpal infection and subsequent apical periodontitis.[24]

Sickle cell anemia is the prototype of hereditary globinopathies, characterized by the production of structurally abnormal hemoglobulin.[141] The red blood cells are sickle shaped. Accumulation of distorted red cells could cause vasoocclusion, which could lead to anoxia, infarcts, and necrosis of tissue.[103] The occurrence of asymptomatic pulp necrosis in clinically intact permanent teeth has been reported.[14,126,250] Pulp necrosis could predispose to pulpal infection and apical periodontitis because of a lack of defense mechanism.

Sjögren syndrome is a chronic, systemic autoimmune disorder.[201] It involves salivary glands resulting in xerostomia. The lack of salivary cleansing action predisposes the patient to dental caries. If the caries is not prevented or treated early, it would impose risk of pulpal infection and apical

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periodontitis.

Radiation therapy of head and neck malignant lesions is associated with a high occurrence of caries.[107] If untreated, the radiation caries could lead to pulpal infection and subsequent apical periodontitis. It has been shown that a radiation dose 66 to 72.2 Gy (gray) in the head and neck region was associated with a high prevalence of caries and apical periodontitis because of a change in microflora favorable for carious development.[106]

However, there is no clear-cut direct causation or mechanism between systemic disease risk factors and the incidence or persistence of apical periodontitis without prior caries and pulp infection.

Wound healing of apical periodontitis

Periapical wound healing after nonsurgical root canal therapy

Understanding wound healing is as important as knowing the pathogenesis of disease, because satisfactory wound healing is the ultimate goal of treatment. If we are able to understand the mechanisms of periapical wound healing, we can design treatment approaches that maximize favorable conditions for wound healing to occur, such as effective disinfection of the root canal system in nonsurgical root canal therapy, control of periapical inflammation by medication, or incorporation of growth factors in bone grafts in surgical endodontic therapy (see also Chapters 13 and 22).

Wound healing begins as soon as inflammation starts. When irritants (microbial and nonmicrobial) in the canal systems or in the periapical tissues are eliminated by nonsurgical or surgical endodontic therapy, inflammatory mediators are no longer produced in the periapical tissues because of the reduction of inflammatory cells. Inflammatory mediators already present are inactivated by the body's control mechanisms to prevent an inflammatory reaction from going unchecked. This process precedes wound healing. Although a great deal of information is known about what turns on the inflammation, relatively little is known about what turns off the inflammatory system after elimination of irritants. Examples of host antiinflammatory control mechanisms are (1) enzyme destruction of inflammatory activators;

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(2) natural inhibitors of inflammatory mediators (opioids, somatostatin, glucocorticoids); (3) relative balance between intracellular levels of cyclic AMP (adenosine monophosphate) and cyclic GMP (guanosine monophosphate); (4) the antiphlogistic role of histamine; (5) inhibitors of the complement system[298]; and (6) antiinflammatory cytokines, such as IL-4, IL-10, IL-13, and TGF-β.[96,199] In addition, the major cellular inducer of inflammation, neutrophilic leukocytes, undergo apoptosis,[92] and the major cellular player of wound healing, macrophages, secrete antiinflammatory molecules such as lipoxins, resolvins, and protectins.[141,171,199]

The process of wound healing is tightly regulated by cell-to-cell cross-talk, cell-to-ECM interactions, and cell surface receptors, as well as the temporal and spatial expression of a variety of cytokines, growth factors, and neuropeptides and apoptosis (Table 16.3).[32,89,92,268,313] All these cellular and humoral factors operate together in either an antagonistic or synergistic manner and are precisely orchestrated during wound healing. This results in a highly organized response permitting regeneration of the original tissue architecture. Wound healing appears to be a programmed event. Much information concerning the pathogenesis of apical periodontitis has been gained from animal studies.[78,122,292] Although studies of wound healing of teeth with apical periodontitis after nonsurgical root canal therapy and endodontic surgery are available in a few animal experiments and a human study,[13,150,151] no studies of wound healing of apical periodontitis lesions with cyst formation after nonsurgical root canal therapy exist in the literature.

Table 16.3

Important Growth Factors/Cytokines in Wound Healing

Cytokines	Major Source	Target Cells and Major Effects
EGF	Macrophages, platelets, fibroblasts	Epithelial cells, fibroblasts, endothelial cells
FGF	Macrophages, endothelial cells	Endothelial cells (angiogenesis), mesenchymal cells
TGF-α	Macrophages, platelets, keratinocytes	Angiogenesis, fibroblasts

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TGF-β	Macrophages, platelets	Similar to EGF
PDGF	Macrophages, platelets, endothelial cells	Chemoattractant for macrophages, fibroblasts
VEGF	Macrophages, epithelial cells	Angiogenesis
IGF	Fibroblasts, epithelial cells	Granulation tissue formation, reepithelialization
CSF	Multiple cells	Macrophages, granulation tissue formation
SP, CGRP	Sensory nerve	Endothelial cells, fibroblasts, keratinocytes

CGRP, Calcitonin gene–related peptide; *CSF,* colony-stimulating factor; *EGF,* epidermal growth factor; *FGF,* fibroblast growth factor; *IGF,* insulin-like growth factor; *PDGF,* platelet-derived growth factor; *TGF,* transforming growth factor; *VEGF,* vascular endothelial growth factor.

Modified from Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, 2004, Oxford University Press; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, 2002, Mosby; Werner S, Grose R: Regulation of wound healing by growth factors and cytokines, *Physiol Rev* 83:835, 2003.

Wound healing of apical periodontitis lesions after proper nonsurgical root canal therapy follows the general principle of wound healing of connective tissues elsewhere in the body, with the formation of fibrovascular granulation tissue, removal of necrotic tissue and dead bacteria by activated macrophages, and finally repair or regeneration of the wounded tissue. Healing of apical periodontitis lesions is largely accomplished by regeneration and to some degree by repair. Local tissue resident cells involved in periapical wound healing are osteocytes, osteoblasts, and bone marrow mesenchymal stem cells in alveolar bone and stem cells in the periodontal ligament.[243] During periapical wound healing, many unwanted hyperplastic cells (e.g., endothelial cells, fibroblasts, epithelial cells) are deleted by apoptosis,[63,89,92] and the ECM is remodeled by metalloproteinases. Pathologic processes such as extensive fibrosis do not occur often, and the damaged periapical tissues can be restored mostly to their original structure by the process of regeneration.

Mesenchymal stem cells have immunomodulatory and antiinflammatory properties.[3,169,253] Recently, mesenchymal stem cells were shown to play an important role as prohealing and immunosuppressive agents in the pathogenesis of inflammatory periapical lesions.[15] During periapical

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inflammation, proinflammatory cytokines such as IL-1β and TNFα are capable of inhibiting osteogenic differentiation from mesenchymal stem cells.[143] However, during periapical wound healing, mesenchymal stem cells are capable of inhibiting immune response and producing antiinflammatory cytokines.[15]

The temporal and spatial relationship between alveolar bone, cementum, and periodontal ligament during periapical wound healing after nonsurgical root canal therapy cannot be clearly delineated. Nevertheless, wound healing appears to recapitulate the embryonic morphogenesis of damaged tissues or organs in many instances. The process of periapical wound healing after nonsurgical root canal therapy may be similar to wound healing following guided tissue regeneration in periodontal therapy: regeneration of new cementum, new alveolar bone, and new periodontal ligament.[70,294] Both nonsurgical root canal therapy and guided tissue regeneration therapy in periodontal disease are intended to remove irritants and provide a favorable microenvironment conducive to regeneration of periodontal tissues damaged by apical periodontitis and marginal periodontitis, respectively.

During periapical wound healing, the cells of viable periodontal ligament from adjacent root surfaces proliferate to cover the root surfaces in which the periodontal ligament was damaged by apical periodontitis and removed by macrophages. Proteins derived from the Hertwig epithelial root sheath (i.e., enamel matrix proteins) are required for cementoblast differentiation from ectomesenchymal stem cells in the dental follicle during tooth development.[262] However, the cells of the Hertwig epithelial root sheath are absent in mature teeth.[198,240] Nevertheless, the ECM and growth factors of cementum (i.e., IGF-1, FGFs, EGF, BMP, TGF-β, PDGF) sequestered after cemental resorption in mature teeth are capable of inducing proliferation, migration, attachment, and differentiation of multipotent stem cells in the periodontal ligament into cementoblast-like cells and produce cementoid tissue on the root surface denuded of periodontal ligament.[50,91,243] This is similar to reparative dentin formation by pulp stem cells in vital pulp therapy where growth factors such as TGF-β are released from dentin matrix binding sites by capping materials, such as calcium hydroxide or mineral trioxide aggregate.[197,227,300] Root resorption that involves cementum or both cementum and dentin can only be repaired by cementoid tissue, because

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multipotent stem cells of the periodontal ligament are incapable of differentiating into odontoblasts that produce dentin.[243]

Bone has a remarkable capacity for regeneration in response to injury. During periapical wound healing, the osteoprogenitor cells lining the surfaces of endosteum—stimulated by TGF-β, BMPs, IGFs, PDGF, VEGF, and cytokines released by stromal cells, osteoblasts, platelets, and bone matrix after bone resorption—can undergo proliferation and differentiation into osteoblasts and produce bone matrix.[7,166] The osteoprogenitor cells in the inner layer of periosteum beneath the oral mucosa—stimulated by TGF-β, BMPs, IGFs, PDGF, and VEGF—are also capable of proliferation and differentiation into osteoblasts and produce bone matrix.[126,166] If both buccal and lingual/palatal cortical bone plates are destroyed by large apical periodontitis lesions, it is possible that the lesion may be repaired with fibrous scar tissue formation because of extensive destruction of the periosteum beneath the oral mucosa.[13] Accordingly, a guided tissue regeneration procedure using membrane barriers and bone grafts may be recommended to prevent ingrowth of fibroblasts from periosteum or submucosa into the bony defect and to enhance periapical wound healing if periapical surgery is necessary.[58,59] The cellular and molecular mechanisms of excess scar tissue formation in periapical wound healing are not completely understood. Growth factors/cytokines may play an important role in regulating fibroblast gene expression and excess scar tissue formation.[299]

The newly regenerated periodontal ligament will finally undergo remodeling into a mature periodontal ligament, with one group of collagen fibers (Sharpey fibers) inserted into the newly formed cementum and another group of collagen fibers inserted into the newly formed alveolar bone. Thereby, regeneration of damaged periapical tissues, cementum, periodontal ligament, and alveolar bone is completed.

Periapical wound healing after surgical endodontic therapy

The mechanism of periapical wound healing after nonsurgical and surgical endodontic therapy is similar, but the kinetics of healing of the periapical wound after endodontic surgery is faster than nonsurgical endodontic

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therapy.[142] In surgical endodontic therapy, a clinician removes irritants, such as necrotic cells, tissue debris, and bacteria in the periapical lesions, which is called *surgical debridement*.[162,171] In contrast, in nonsurgical endodontic therapy, activated macrophages perform bacterial killing and cleanup of periapical lesions, which is called *biologic debridement*.[171] Surgical debridement is very effective and of course quite rapid, whereas biologic debridement takes time. Surgical treatment may result in more rapid periapical bone healing, but also may imply a higher risk of late failure compared to nonsurgical endodontic therapy.[142] However, endodontic surgery is more invasive. The distinctive difference between nonsurgical and surgical endodontic therapy is that the goal of nonsurgical endodontic therapy is to remove primary microbial etiology from the root canal system, and the goal of surgical endodontic therapy is often to seal microbial etiology within the root canal system by root-end filling in most cases (see also Chapter 22).

Can radicular cysts in apical periodontitis lesions regress after nonsurgical endodontic therapy?

Based on histology and cell biology, no studies have ever shown that apical true cysts are different from apical pocket cysts. It was suggested that pocket cysts in apical periodontitis lesions might regress after nonsurgical root canal therapy by the mechanism of apoptosis or programmed cell death, based on molecular cell biology.[163] In contrast, apical true cysts may be less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature.[191] Histologically, inflammatory cell infiltration is always present in the lining epithelium or fibrous connective tissue capsule of apical true cysts.[191,223,224] This indicates there is continued presentation of irritants (such as bacteria)—present in the root canal system, the periapical tissues, or the lumen of cysts—to attract inflammatory cells to the cystic lining epithelium or fibrous connective tissue capsule.[223,224] It is not known if epithelial cells of apical true cysts alone are capable of acting as autocrine cells and secreting growth factors to sustain their own survival. It is important to realize that the apical true cyst is completely different from the odontogenic keratocyst, which is self-sustaining because it is a neoplastic lesion. Biologically, it is unlikely that the hyperplastic epithelial cells of inflammatory apical true cysts

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would suddenly transform into cells that behave like self-sustaining neoplasms. Any disease caused by infection should be able to regress after removal of its causative irritant(s), unless the irritants themselves are neoplasm-inducing agents or carcinogens, such as some viruses and human malignant tumors.[171,305] From pathogenesis, histology, and molecular cell biology, apical true cysts are similar to pocket cysts. Accordingly, apical true cysts, similar to pocket cysts, may be able to regress after nonsurgical root canal therapy by the mechanism of apoptosis if root canal infection is effectively under control.[163] This prediction is consistent with the high level of healing observed after nonsurgical root canal treatment.

In apical periodontitis lesions with cyst formation, the cysts have to regress before the periapical tissues can be restored to their original architecture. It is not known what matrix serves as a scaffold for endothelial cells, fibroblasts, and osteoblasts to migrate into the lumen of regressing cysts after nonsurgical root canal therapy. Complete regression of radicular cysts after nonsurgical root canal therapy could be due to any of several possible scenarios. Regression of radicular cysts and regeneration of bone may occur concurrently, or during regression of radicular cysts, part of the cystic lining epithelium could disintegrate due to apoptosis of local epithelial cells. Together with degradation of the basal lamina by MMPs, this could allow a fibrous connective tissue capsule to grow into the lumen of radicular cysts. Eventually, the cystic lining epithelium will completely regress or become remnants of epithelial cell rests remaining in the periodontal ligament.

Taken together, our knowledge of apical cyst formation and healing offers tantalizing clinical implications and clearly supports the conclusion that more studies are required to understand the complex mechanisms of regression of inflammatory apical cysts, both pocket and true.

Factors influencing periapical wound healing after endodontic therapy

Local and systemic factors may affect periapical wound healing. Infection will complicate and prevent wound healing, foreign bodies can impair wound healing,[193,324] and nutrition can also affect wound healing.[316] Diabetes was reported to reduce the likelihood of healing of apical periodontitis lesions

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after nonsurgical root canal therapy.[77] Impaired nonspecific immune response and disorders of the vascular system appeared to have a significant influence on the success rate of nonsurgical root canal therapy on teeth with apical periodontitis.[172] However, immunocompromised patients, such as HIV patients, responded as well as counterpart patients after nonsurgical endodontic therapy.[52,85,214] Although smoking has not been shown to be associated with increased incidence of apical periodontitis and prognosis of nonsurgical root canal therapy,[26,66] smoking may increase complications of periapical surgery, such as pain and swelling.[83]

Patients receiving radiotherapy of jaws and bisphosphonate therapy have a risk of developing osteonecrosis of jawbones,[220,226,317] so nonsurgical root canal therapy is recommended for these patients.[124,157] However, for endodontic surgery, the guidelines of the American Association of Oral and Maxillofacial Surgeons position paper on bisphosphonate-related osteonecrosis of the jaws are highly recommended.[9] For asymptomatic patients who have taken an oral bisphosphonate for less than 3 years and have no clinical risk factors, endodontic surgery is not contraindicated.[9] Nevertheless, the patient's physician should be consulted. Any kind of surgical procedure should be avoided for patients receiving intravenous bisphosphonate.[9] To prevent the complication of osteonecrosis, if patients are going to receive radiation of jaws or bisphosphonate therapy, nonsurgical or surgical endodontic therapy should be completed before the initiation of radiation or bisphosphonate therapy.[9]

Telegram: @dental_k

www.konkur.in

References

1. Abbas AK, Lichtman AH, Pober JS. Cellular and molecular immunology 2007; Saunders Philadelphia.
2. Abous-Rass M, Bogen G. *Microorganisms in closed periapical lesions*, Int Endod J 1998;31: 39.
3. Aggarwal S, Pittenger MF. *Human mesenchymal stem cells modulate allogeneic immune Cell responses*, Blood 2005;105: 1815.
4. Akamine A, Hashiguchi I, Toriya Y, et al: *Immunohistochemical examination of the localization of macrophages and plasma cells in induced rat's periapical lesions*, Endod Dent Traumatol 1994;10: 121.
5. Akira S, Takeda K, Kaisho T. *Toll-like receptors: critical proteins linking innate and acquired immunity*, Nature Immunol 2001;2: 675.
6. Akira S, Uematsu S, Takeuchi O. *Pathogen recognition and innate immunity*, Cell 2006;124: 783.
7. Al-Aql ZS, Alagl AS, Graves DT, et al: *Molecular mechanisms controlling bone formation during fracture healing and distraction osteogenesis*, J Dent Res 2008;87: 107.
8. Amaya MP, Criado L, Blanco B, et al: *Polymorphisms of pro-inflammatory cytokine genes and the risk for acute suppurative or chronic nonsuppurative apical periodontitis in a Colombian population*, Int Endod J 2013;46: 71.
9. American Association of Oral and Maxillofacial Surgeons: *Medication-Related Osteonecrosis of the Jaw-2014 Update*.
10. American Dental Association. 2005-2006 survey of dental

Telegram: @dental_k

www.konkur.in

services rendered 2006; American Dental Association Chicago.
11. Andreasen FM. *Transient apical breakdown and its relation to color and sensitivity changes after luxation injuries to teeth*, Endod Dent Traumatol 1986;2: 9.
12. Andreasen JO, Andreasen FM. Textbook and color atlas of traumatic injuries to the teeth ed 3 1994; Munksgaard Copenhagen.
13. Andreasen JO, Rud J. *Modes of healing histologically after endodontic surgery in 70 cases*, Int Oral Surg 1972;1: 148.
14. Andrews CH, England MC, Kemp WB. *Sickle cell anemia: an etiological factor in pulp necrosis*, J Endod 1986;9: 249.
15. Araujo-Pires AC, Biguetti, Repeke CE, et al: *Mesenchymal stem cells as active prohealing and immunosuppressive agents in periapical environment: evidence from human and experimental periapical lesions*, J Endod 2014;40: 1560.
16. Armitage GC. *Periodontal diagnoses and classification of periodontal disease*, Periodontology 2000 2004;34: 9.
17. Babior BM. *The respiratory burst of phagocytes*, J Clin Invest 1984;73: 599.
18. Baron R. *Molecular mechanisms of bone resorption by the osteoclasts*, Anat Rec 1989;224: 317.
19. Barthel CR, Zimmer S, Trope M. *Relationship of radiologic and histologic signs of inflammation in human root-filled teeth*, J Endod 2004;30: 75.
20. Bartkiewicz M, Hernando N, Reddy SV, et al: *Characterization of the osteoclast vacuolar H 1 -ATPase B-subunit*, Gene 1995;160: 157.
21. Basbaum AL, Bautista DM, Scherrer G, et al: *Cellular*

Telegram: @dental_k

www.konkur.in

and molecular mechanisms of pain, Cell 2009;139: 267.
22. Baumgartner JC, Heggers JP, Harrison JW. *The incidence of bacteremias related to endodontic procedures. I. Non-surgical endodontics*, J Endod 1976;2: 135.
23. Bender IB. *Factors influencing the radiographic appearance of bone lesions*, J Endod 1982;8: 161.
24. Bender IB, Bender AB. *Diabetes mellitus and the dental pulp*, J Endod 2003;29: 383.
25. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone: 1*, J Am Dent Assoc 1961;62: 152.
26. Bergstrom J, Babcan J, Eliasson S. *Tobacco smoking and dental periapical condition*, Eur J Oral Sci 2004;112: 115.
27. Bezerra MC, Carvalho JF, Prokopowitsch AS, Pereira RM. *RANK, RANKL and osteoprotegerin in arthritic bone loss*, Braz J Med Biol Res 2005;38: 161.
28. Birek C, Heersche JN, Jez D, et al: *Secretion of a bone resorbing factor by epithelial cells cultured from porcine rests of Malassez*, J Periodontal Res 1983;18: 75.
29. Birklein F, Schmelz M. *Neuropeptides, neurogenic inflammation and complex regional pain syndrome (CRPS)*, Neurosci Lett 2008;437: 199.
30. Block RM, Bushell A, Rodrigues H, et al: *A histopathologic, histobacteriologic, and radiographic study of periapical endodontic surgical specimens*, Oral Surg Oral Med Oral Pathol 1976;42: 656.
31. Boyle WJ, Simonet WS, Lacey DL. *Osteoclast differentiation and activation*, Nature 2003;423: 337.
32. Brian SD. *Sensory peptides: their role in inflammation and wound healing*, Immunopharmacology 1997;37: 133.
33. Brinkmann V, Zychlinsky A. *Beneficial suicide: why neutrophils die to make NETs*, Nat Rev Microbiol 2007;5:

Telegram: @dental_k

www.konkur.in

577.
34. Britto LR, Katz J, Guelmann M, Heft M. *Periradicular radiographic assessment in diabetic and control individuals*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 449.
35. Brook I, Fraizier EH, Gher ME. *Aerobic and anaerobic microbiology of periapical abscess*, Oral Microbiol Immunol 1991;6: 123.
36. Browne RM. *The origin of cholesterol in odontogenic cysts in man*, Arch Oral Biol 1971;16: 107.
37. Brownless M. *The pathobiology of diabetes complications: a unifying mechanism*, Diabetes 2005;54: 1615.
38. Brunette DM. *Cholera toxin and dibutyl cyclic-AMP stimulate the growth of epithelial cells derived from epithelial cell rests from porcine periodontal ligament*, Arch Oral Biol 1984;29: 303.
39. Brynolf I. *A histological and roentgenological study of the periapical region of human upper incisors*, Odontol Rev Suppl 11, 1967;18: 1.
40. Burd PR, Rogers HW, Gordon JR, et al: *Interleukin 3-dependent and -independent mast cells stimulated with IgE and antigen express multiple cytokines*, J Exp Med 1989;170: 245.
41. Byers MR, Narhi MVO. *Dental injury models: experimental tools for understanding neuroinflammatory nociceptor functions*, Crit Rev Oral Biol 1999;10: 4.
42. Byers MR, Taylor PE, Khayat BG, et al: *Effects of injury and inflammation on the pulp and periapical nerves*, J Endod 1990;16: 78.
43. Canalis E, McCarthy TL, Centrella M. *Growth factors and cytokines in bone cell metabolism*, Annu Rev Med

Telegram: @dental_k

www.konkur.in

1991;42: 17.
44. Caplan DJ, Chasen JB, Krall EA, et al: *Lesions of endodontic origin and risk of coronary heart disease*, J Dent Res 2006;85: 996.
45. Cargill M, Altshuler D, Ireland J, et al: *Characterization of single-nucleotide polymorphisms in coding regions of human genes*, Nat Genet 1999;22: 231.
46. Caviedes-Bucheli J, Muñoz HR, Azuero-Holguín MM, et al: *Neuropeptides in dental pulp: the silent protagonists*, J Endodon 2008;34: 773.
47. Cawson RA, Everson JW. Oral pathology and diagnosis 1987; Saunders Philadelphia.
48. Chai L, Song YQ, Zee KY, et al: *SNPs of Fc-gamma receptor genes and chronic periodontitis*, J Dent Res 2010;89: 705.
49. Chedid M, Rubin JS, Csaky KG, Aaronson SA. *Regulation of keratinocyte growth factor gene expression by interleukin 1*, J Biol Chem 1994;269: 10753.
50. Cochran DL, Wozney JM. *Biological mediators for periodontal regeneration*, Periodontol 2000 1999;19: 40.
51. Cook DN, Pisetsky DS, Schwartz DA. *Toll-like receptors in the pathogenesis of human disease*, Nat Immunol 2004;5: 975.
52. Cooper H. *Root canal treatment in patients with HIV infection*, Int Endod J 1993;26: 369.
53. Cortesini R, LeMaoult J, Ciubotariu R, et al: *CD81CD28- T suppressor cells and the induction of antigen-specific, antigen-presenting cell-mediated suppression of T H reactivity*, Immunol Rev 2001;182: 201.
54. Costerton JW, Geesey GG, Chen KJ. *How bacteria stick*, Sci Am 1978;238: 86.
55. Cymerman JJ, Cymerman DH, Walters I, et al: *Human T*

Telegram: @dental_k

www.konkur.in

lymphocyte subpopulations in chronic periapical lesions, J Endod 1984;10: 9.

56. de Sa AR, Moreira PR, Xavier GM, et al: *Association of CD14, IL1B, IL6, IL10 and TNFA functional gene polymorphisms with symptomatic dental abscesses*, Int Endod J 2007;40: 563.
57. Dahlen G, Magnusson BC, Moller A. *Histological and histochemical study of the influence of lipopolysaccharide extracted from Fusobacterium nucleatum on the periapical tissues in the monkey Macaca fascicularis*, Arch Oral Biol 1981;26: 591.
58. Dahlin C, Gottlow J, Linde A, et al: *Healing of maxillary and mandibular bone defects using a membrane technique: an experimental study in monkeys*, Scand J Plast Reconstr Hand Surg 1990;24: 13.
59. Dahlin C, Linde A, Gottlow J, et al: *Healing of bone defects by guided tissue regeneration*, Plast Reconstr Surg 1988;81: 672.
60. Damoiseaux J. *Regulatory T cells: back to the future*, Neth J Med 2006;64: 4.
61. Debelian GJ, Olsen I, Transtad L. *Systemic diseases caused by oral microorganisms*, Endod Dent Traumatol 1994;10: 5.
62. Debelian GJ, Olsen I, Transtad L. *Bacteremia in conjunction with endodontic therapy*, Endod Dent Traumatol 1995;11: 142.
63. Desmouliere A, Redard M, Darby I, et al: *Apoptosis mediates the decrease in cellularity during the transition between granulation tissue and scar*, Am J Pathol 1995;146: 56.
64. Dickerson C, Undem B, Bullock B, et al: *Neuropeptides regulation of proinflammatory cytokine responses*, J

Telegram: @dental_k

www.konkur.in

Leukocyte Biol 1998;63: 602.
65. Diehl SR, Wang Y, Brooks CN, et al: *Linkage disequilibrium of interleukin-1 genetic polymorphisms with early-onset periodontitis*, J Periodontol 1999;70: 418.
66. Duncan HF, Pitt Ford TR. *The potential association between smoking and endodontic disease*, Int Endod J 2006;39: 843.
67. Dwyer TG, Torabinejad M. *Radiographic and histologic evaluation of the effect of endotoxin on the periapical tissues of the cat*, J Endod 1980;7: 31.
68. Dziak R. *Biochemical and molecular mediators of bone metabolism*, J Periodontol 1993;64: 407.
69. Echchannaoui H, Frei K, Schnell C, et al: *Toll-like receptor 2-deficient mice are highly susceptible to Streptococcus pneumoniae meningitis because of reduced bacterial clearing and enhanced inflammation*, J Infect Dis 2002;186: 798.
70. Egelberg J. *Regeneration and repair of periodontal tissues*, J Periodontal Res 1987;22: 233.
71. Ehrmann EH. *Focal infection: the endodontic point of view*, Oral Surg Oral Med Oral Pathol 1977;44: 628.
72. Eliasson S, Halvarson C, Ljunheimer C. *Periapical condensing osteitis and endodontic treatment*, Oral Surg Oral Med Oral Pathol 1984;57: 195.
73. Eriksen HM, Bjertness E. *Prevalence of apical periodontitis and results of endodontic treatment in middle-aged adults in Norway*, Dent Traumatol 1991;7: 1.
74. Figdor D. *Apical periodontitis: a very prevalent problem*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 65.
75. Formigli L, Orlandini SZ, Tonelli P, et al: *Osteolytic processes in human radicular cysts: morphological and*

Telegram: @dental_k

www.konkur.in

biochemical results, J Oral Pathol Med 1995;24: 216.
76. Fouad AF, Barry J, Russo J, et al: *Periapical lesion progression with controlled microbial inoculation in a type I diabetic mouse model*, J Endod 2002;28: 8.
77. Fouad AF, Burleson J. *The effect of diabetes mellitus on endodontic treatment outcome: data from an electronic patient record*, J Am Dent Assoc 2003;134: 43.
78. Fouad AF, Walton RE, Rittman BR. *Induced periapical lesions in ferret canines: histological and radiographic evaluation*, Dent Traumatol 1992;8: 56.
79. Friedman S. *Considerations and concepts of case selection in the management of post-treatment endodontic disease (treatment failure)*, Endod Topics 2002 2002;1: 54.
80. Fuchs TA, Abed U, Goosmann C, et al: *Novel cell death program leads to neutrophil extracellular traps*, J Cell Biol 2007;176: 231.
81. Gao Z, Falitz CM, Mackenzie IC. *Expression of keratinocyte growth factor in periapical lesions*, J Dent Res 1996;75: 1658.
82. Gao Z, Mackenzie IC, Rittman BR, et al: *Immunocytochemical examination of immune cells in periapical granulomas and odontogenic cysts*, J Oral Pathol 1988;17: 84.
83. Garcia B, Penerrocha M, Marti E, et al: *Pain and swelling after periapical surgery related to oral hygiene and smoking*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 271.
84. Giachelli CM, Steitz S. *Osteopontin: a versatile regulator of inflammation and biomineralization*, Matrix Biol 2000;19: 615.
85. Glick M, Abel SN, Muzyka BC, et al: *Dental*

Telegram: @dental_k

www.konkur.in

complications after treating patients with AIDS, J Am Dent Assoc 1994;125: 296.

86. Gonzalez-Rey E, Chorny A, Delgado M. *Regulation of immune tolerance by anti-inflammatory neuropeptides*, Nat Rev Immunol 2007;7: 52.
87. Gore EA, Sanders JJ, Pandey JP, et al: *Interleukin-1beta13953 allele 2: association with disease status in adult periodontitis*, J Clin Periodontol 1998;25: 781.
88. Green TL, Walton RE, Taylor JK, et al: *Radiographic and histologic findings of root canal treated teeth in cadaver*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 707.
89. Greenhalgh DG. *The role of apoptosis in wound healing*, Int J Biochem Cell Biol 1998;30: 1019.
90. Grossman RM, Kreuger J, Yourish D, et al: *Interleukin-6 is expressed in high levels in psoriatic skin and stimulates proliferation of cultured human keratinocyte*, Proc Nat Acad Sci U S A 1989;86: 6367.
91. Grzesik WJ, Narayanan AS. *Cementum and periodontal wound healing and regeneration*, Crit Rev Oral Biol Med 2002;13: 474.
92. Haanen C, Vermes IV. *Apoptosis and inflammation*, Mediators Inflamm 1995;4: 5.
93. Haapasalo M, Ranta K, Ranta H. *Mixed anaerobic periapical infection with sinus tract*, Endod Dent Traumatol 1987;3: 83.
94. Hahn C-L, Liewehr FR. *Innate immune responses of the dental pulp to caries*, J Endod 2007;33: 643.
95. Hammarstrom L, Lindskog S. *General morphological aspects of resorption of teeth and alveolar bone*, Int Endod J 1985;18: 93.
96. Hanada T, Yoshimura A. *Regulation of cytokine signaling*

Telegram: @dental_k

www.konkur.in

and inflammation, Cytokine Growth Factor Rev 2002;13: 413.

97. Hancock HH III, Sigurdsson A, Trope M, et al: *Bacteria isolated after unsuccessful endodontic treatment in a North American population*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 579.
98. Happonen RP. *Periapical actinomycosis: a follow-up study of 16 surgically treated cases*, Endod Dent Traumatol 1986;2: 205.
99. Hargreaves KM, Goodis HE, Tay FR. Dental pulp 2012; Quintessence Chicago.
100. Hargreaves KM, Swift JQ, Roszkowski MT, et al: *Pharmacology of peripheral neuropeptide and inflammatory mediator release*, Oral Surg Oral Med Oral Pathol 1994;78: 503.
101. Hart TC, Kornman KS. *Genetic factors in the pathogenesis of periodontitis*, Periodontol 2000 1997;14: 202.
102. Hauman CHJ, Love RM. *Biocompatibility of dental materials used in contemporary endodontic therapy: a review. Part 2. Root-canal filling materials*, Int Endod J 2003;36: 147.
103. Hebble PR, Vercellotti GM, Nath KA. *A systems biology consideration of the vasculopathy of the sickle cell anemia: the need for multi-modality chemo-prophylaxis*, Cardiovasc Hematol Disord Drug Targets 2009;9: 271.
104. Hirsh BC, Johnson WC. *Concepts of granulomatous inflammation*, Int J Dermatol 1984;23: 90.
105. Hirvonen T, Hippi P, Narhi M. *The effect of an opioid antagonist and somatostatin antagonist on the nerve function in normal and inflamed dental pulps*, J Dent Res 1998;77: 1329.

Telegram: @dental_k

www.konkur.in

106. Hommez GG, De Meerkeer GO, De Neve WJ, et al: *Effect of radiation dose on the prevalence of apical periodontitis: a dosimetric analysis*, Clin Oral Invest 2012;16: 1543.
107. Hong CHL, Napenas JJ, Hodgson BD, et al: *A systematic review of dental disease in patients undergoing cancer therapy*, Support Care Cancer 2010;18: 1007.
108. Hou L, Sasaki H, Stashenko P. *Toll-like receptor 4-deficient mice have reduced bone destruction following mixed anaerobic infection*, Infect Immun 2000;68: 4681.
109. Hou L, Sasakj H, Stashenko P. *B-Cell deficiency predisposes mice to disseminating anaerobic infections: protection by passive antibody transfer*, Infect Immun 2000;68: 5645.
110. Hou L, Wang X. *PKC and PKA, but not PKG mediate LPS-induced CGRP release and (Ca 2 1) I elevation in DRG neurons of neonatal rats*, J Neurosci Res 2001;66: 592.
111. Hunter W. *Oral sepsis as a cause of disease*, Br Med J 1900;2: 215.
112. Huumonen S, Orstavik D. *Radiological aspects of apical periodontitis*, Endod Topics 2002;1: 3.
113. Ingle J, Bakland L, Baugartner C. Ingle's endodontics ed 6 2008; BC Decker Hamilton.
114. Irwin CR, Schor SL, Ferguson NW. *Expression of EGF-receptor on epithelial and stromal cells of normal and inflamed gingiva*, J Periodont Res 1991;26: 388.
115. Isaka J, Ohazama A, Kobayashi M, et al: *Participation of periodontal ligament cells with regeneration of alveolar bone*, J Periodontol 2001;72: 314.
116. Iwama A, Nishigaki N, Nakamura K, et al: *The effect of high sugar intake on the development of periradicular*

Telegram: @dental_k

www.konkur.in

lesions in rats with type 2 diabetes, J Dent Res 2003;82: 322.

117. Iwu C, MacFarlane TW, MacKenzie D, et al: *The microbiology of periapical granulomas*, Oral Surg Oral Med Oral Pathol 1990;69: 502.
118. Janeway CA, Medzhitov R. *Innate immune recognition*, Annu Rev Immunol 2002;20: 197.
119. Janeway CA, Travers P, Walport M, et al: Immunobiology: the immune system in health and disease ed 6 2005; Garland Science New York.
120. Jimenez-Pinzon A, Segura-Egea JJ, Poyato-Ferrera M, et al: *Prevalence of apical periodontitis and frequency of root-filled teeth in an adult Spanish population*, Int Endod J 2004;37: 167.
121. Johnson GM, Lee DA, Regelmann WE, et al: *Interference with granulocyte function by Staphylococcus epidermidis slime*, Infect Immun 1986;54: 13.
122. Kakehashi S, Stanley H, Fitzgerald R. *The effect of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral Pathol 1965;20: 340.
123. Kaneko T, Okiji T, Kan L, et al: *Ultrastructural analysis of MHC class II molecule-expressing cells in experimentally induced periapical lesions in the rat*, J Endod 2001;27: 337.
124. Katz H. *Endodontic implications of bisphosphonate-associated osteonecrosis of the jaws: a report of three cases*, J Endod 2005;31: 831.
125. Kawashima N, Okiji T, Kosaka T, et al: *Kinetics of macrophages and lymphoid cells during the development of experimentally induced periapical lesions in rat molars*, J Endod 1996;22: 311.

Telegram: @dental_k

www.konkur.in

126. Kaya AD, Aktener BO, Unsal P. *Pulp necrosis with sickle cell anemia*, Int Endod J 2004;37: 602.
127. Kayaoglu G, Orstavik D. *Virulence factors of Enterococcus faecalis: relationship to endodontic disease*, Crit Rev Oral Biol Med 2004;15: 308.
128. Khan AA, Hargreaves KM. *Dental Pain*, Trup JC Sommer C Hugger A The Puzzle of orofacial pain 2007; Karger Basel.
129. Kierszenbaum AL. Histology and cell biology 2002; Mosby St. Louis.
130. Kim J, Amar S. *Periodontal disease and systematic conditions: a bidirectional relationship*, Odontology 2006;94: 10.
131. Kimberly CL, Byers MR. *Inflammation of rat molar pulp and periodontium causes increased calcitonin gene-related peptide and axonal sprouting*, Ant Rec 1988;222: 289.
132. Kobayashi T, Westerdaal NA, Miyazaki A, et al: *Relevance of immunoglobulin G Fc receptor polymorphism to recurrence of adult periodontitis in Japanese patients*, Infect Immun 1997;65: 3556.
133. Kobayashi T, Yamamoto K, Sugita N, et al: *The Fc gamma receptor genotype as a severity factor for chronic periodontitis in Japanese patients*, J Periodontol 2001;72: 1324.
134. Kohsaka T, Kumazawa M, Yamasaki M, et al: *Periapical lesions in rats with streptozotocin-induced diabetes*, J Endod 1996;22: 418.
135. Kopp W, Schwarting R. *Differentiation of T lymphocyte subpopulations, macrophages, and HLA-DR-restricted cells of apical granulation tissue*, J Endod 1989;15: 72.
136. Koppang HS, Koppang R, Solheim T, et al: *Cellulose*

Telegram: @dental_k

www.konkur.in

fibers from endodontic paper points as an etiological factor in postendodontic periapical granulomas and cysts, J Endod 1989;15: 369.

137. Kornman KS, Crane A, Wang HY, et al: *The interleukin-1 genotype as a severity factor in adult periodontal disease*, J Clin Periodontol 1997;24: 72.
138. Kovacevic M, Tamarut T, Jonjic N, et al: *The transition from pulpitis to periapical periodontitis in dog's teeth*, Aust Endod J 2008;34: 12.
139. Kramer IR, Pindberg JJ, Shear M. WHO histological typing of odontogenic tumors ed 2 1992; Springer Verlag Geneva.
140. Kuchler EC, Mazzi-Chaves JF, Antunes LS, et al: *Current trends of genetics in apical periodontitis research*, Braz Oral Res 2018;32: 1807.
141. Kumar V, Abbas AK, Fausto N, et al: Robbins and cotran pathologic basis of disease ed 8 2010; Saunders Philadelphia.
142. Kvist T, Reit C. *Results of endodontic retreatment: a randomized clinical study comparing surgical and nonsurgical procedures*, J Endod 1999;25: 814.
143. Lacey DC, Simmons PJ, Graves SE, et al: *Proinflammatory cytokines inhibit osteogenic differentiation from stem cells: implications for bone repair during inflammation*, Osteoarthritis Cartilage 2009;17: 735.
144. Lalonde ER. *A new rationale for the management of periapical granulomas and cysts: an evaluation of histopathological and radiographic findings*, J Am Dent Assoc 1970;80: 1056.
145. Langeland K, Block RM, Grossman LI. *A histopathological and histobacteriologic study of 35*

Telegram: @dental_k

www.konkur.in

periapical endodontic surgical specimens, J Endod 1977;3: 8.

146. Langeland K, Rodrigues H, Dowden W. *Periodontal disease, bacteria and pulpal histopathology*, Oral Surg Oral Med Oral Pathol 1974;37: 257.
147. Laskin DM. *Anatomic considerations in diagnosis and treatment of odontogenic infections*, J Am Dent Assoc 1964;69: 308.
148. Laux M, Abbott PV, Pajarola G, et al: *Apical inflammatory root resorption: a correlative radiographic and histological assessment*, Int Endod J 2000;33: 483.
149. Lekic P, Rojas J, Birek C, et al: *Phenotypic comparison of periodontal ligament cells in vivo and in vitro*, J Periodontal Res 2001;36: 71.
150. Leonardo MR, Hemandez MEFT, Silva LAB, et al: *Effect of a calcium hydroxide-based root canal dressing on periapical repair in dogs: a histological study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: 680.
151. Leonardo MR, Silva LAB, Utrilla LS, et al: *Calcium hydroxide root canal sealers—histologic evaluation of apical and periapical repair after endodontic treatment*, J Endod 1997;23: 232.
152. Lerner UH. *Inflammation-induced bone remodeling in periodontal disease and the influence of post-menopausal osteoporosis*, J Dent Res 2006;85: 596.
153. Ley K. *Molecular mechanisms of leukocyte recruitment in the inflammatory process*, Cardiovasc Res 1996;32: 733.
154. Li H, Chen V, Chen Y, et al: *Herpesviruses in endodontic pathosis: association of Epstein-Barr virus with irreversible pulpitis and apical periodontitis*, J Endod 2009;35: 23.

Telegram: @dental_k

www.konkur.in

155. Li QY, Zhao HS, Meng HX, et al: *Association analysis between interleukin-1 family polymorphisms and generalized aggressive periodontitis in a Chinese population*, J Periodontol 2004;75: 1627.
156. Liapatas S, Nakou M, Rontogianni D. *Inflammatory infiltrate of chronic periradicular lesions: an immunohistochemical study*, Int Endod J 2003;36: 464.
157. Lilly JP, Cox D, Arcuri M, Krell KV. *An evaluation of root canal treatment in patients who have received irradiation to the mandible and maxilla*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86: 224.
158. Lin L, Langeland K. *Innervation of inflammatory periapical lesions*, Oral Surg Oral Med Oral Pathol 1981;51: 535.
159. Lin L, Langeland K. *Light and electron microscopic study of teeth with carious pulp exposures*, Oral Surg Oral Med Oral Pathol 1981;51: 292.
160. Lin L, Shovlin F, Skribner J, et al: *Pulp biopsies from the teeth associated with periapical radiolucency*, J Endod 1984;10: 436.
161. Lin LM, Di Fiore PM, Lin JL, et al: *Histological study of periradicular tissue responses to uninfected and infected devitalized pulps in dogs*, J Endod 2006;32: 34.
162. Lin LM, Gaengler P, Langeland K. *Periapical curettage*, Int Endod J 1996;29: 220.
163. Lin LM, Huang G T-J, Rosenberg P. *Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after periapical wound healing*, J Endod 2007;33: 908.
164. Lin LM, Pascon EA, Skribner J, et al: *Clinical, radiographic, and histological study of endodontic treatment failures*, Oral Surg Oral Med Oral Pathol

Telegram: @dental_k

www.konkur.in

1991;11: 603.
165. Lin LM, Wang SL, Wu-Wang C, et al: *Detection of epidermal growth factor receptor in inflammatory periapical lesions*, Int Endod J 1996;29: 179.
166. Linkhart TA, Mohan S, Baylink DJ. *Growth factors for bone growth and repair*, Bone 1996;19: 1S.
167. Lopez NJ, Jara L, Valenzuela CY. *Association of interleukin-1 polymorphisms with periodontal disease*, J Periodontol 2005;76: 234.
168. Lopez-Lopez J, Jane-Salas E, Estrugo-Devesa A, et al: *Periapical and endodontic status of type 2 diabetic patients in Catalonia, Spain: a cross-sectional study*, J Endod 2011;37: 598.
169. Lyer SS, Rojas M. *Anti-inflammatory effects of mesenchymal stem cells: novel concept for future therapies*, Expert Opin Biol Ther 2008;8: 569.
170. MacNeil RL, Somerman MJ. *Development and regeneration of the periodontium: parallels and contrasts*, Periodontol 1999;19: 8.
171. Majno G, Joris I. Cells, tissues, and disease ed 2 2004; Oxford University Press Oxford.
172. Marending M, Peters OA, Zehnder M. *Factors affecting the outcome of orthograde root canal therapy in a general dentistry hospital practice*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 119.
173. Marotta PS, Fontes TV, Armada L, et al: *Type 2 diabetes mellitus and the prevalence of apical periodontitis and endodontic treatment in an adult Brazilian population*, J Endod 2012;38: 297.
174. Martinelli C, Rulli MA. *The innervation of chronic inflammatory human periapical lesions*, Arch Oral Biol 1967;12: 593.

Telegram: @dental_k

www.konkur.in

175. Marton IJ, Kiss C. *Characterization of inflammatory cell infiltrate in dental periapical lesions*, Int Endod J 1993;26: 131.
176. Marton IJ, Kiss C. *Overlapping protective and destructive regulatory pathways in apical periodontitis*, J Endod 2014;40: 144.
177. Medzhitov R, Janeway C Jr. *Innate immunity*, N Engl J Med 2000;343: 338.
178. Medzhitove R. *Toll-like receptors and innate immunity*, Nat Rev Immunol 2001;1: 135.
179. Meghji S, Qureshi W, Henderson B, et al: *The role of endotoxin and cytokines in the pathogenesis of odontogenic cysts*, Arch Oral Biol 1996;41: 523.
180. Meisel P, Carlsson LE, Sawaf H, et al: *Polymorphisms of Fc gamma-receptors RIIa, RIIIa, and RIIIb in patients with adult periodontal diseases*, Genes Immun 2001;2: 258.
181. Menezes-Silva R, Khaliq S, Deeley K, et al: *Genetic susceptibility to periapical disease: conditional contribution of MMP2 and MMP3 genes to the development of periapical lesions and healing response*, J Endod 2012;38: 604.
182. Molander A, Reit C, Dahlen G, et al: *Microbiological status of root-filled teeth with apical periodontitis*, Int Endod J 1998;31: 1.
183. Moldauer I, Velez I, Kuttler S. *Upregulation of basic fibroblast growth factor in human periapical lesions*, J Endod 2006;32: 408.
184. Moller AJR, Fabricius L, Dahlen G, et al: *Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys*, Scand J Dent Res 1981;89: 29.
185. Morsani JM, Aminoshariae A, Han YW, et al: *Genetic*

Telegram: @dental_k

www.konkur.in

predisposition to persistent apical periodontitis, J Endod 2011;37: 455.

186. Mundy GR. *Inflammatory mediators and the destruction of bone*, J Periodont Res 1991;26: 213.
187. Murray CA, Saunders WP. *Root canal treatment and general health: a review of the literature*, Int Endod J 2000;33: 1.
188. Nagatomo K, Komaki M, Sekiya Y, et al: *Stem cell properties of human periodontal ligament cells*, J Periodontal Res 2006;41: 303.
189. Nair PNR. *Light and electron microscopic studies on root canal flora and periapical lesions*, J Endod 1987;13: 29.
190. Nair PNR. *Apical periodontitis: a dynamic encounter between root canal infection and host response*, Periodontol 2000 1997;13: 121.
191. Nair PNR. *New perspectives on radicular cysts: do they heal?*, Int Endod J 1998;31: 155.
192. Nair PNR, Pajarola G, Schroeder HE. *Types and incidence of human periapical lesions obtained with extracted teeth*, Oral Surg Oral Med Oral Pathol 1996;81: 93.
193. Nair PNR, Sjogren U, Krey G, et al: *Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth*, J Endod 1990;26: 225.
194. Nair PNR, Sjogren U, Sundqvist G. *Cholesterol crystals as an etiological factor in non-resolving chronic inflammation: an experimental study in guinea pigs*, Eur J Oral Sci 1998;106: 644.
195. Nair PNR, Sundqvist G, Sjogren U. *Experimental evidence supports the abscess theory of development of radicular cysts*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 294.

Telegram: @dental_k

www.konkur.in

196. Nakamura I, Takahashi N, Sasaki T, et al: *Chemical and physical properties of extracellular matrix are required for the actin ring formation in osteoclasts*, J Bone Miner Res 1996;11: 1873.
197. Nakashima M. *Induction of dentin formation on canine amputated pulp by recombinant human bone morphogenic proteins (BMP)-2 and -4*, J Dent Res 1994;73: 1515.
198. Nanci A. Ten Cate's oral histology: development, structure, and function ed 7 2008; Mosby St. Louis.
199. Nathan C. *Points of control in inflammation*, Nature 2002;420: 846.
200. Nauseef WM. *How human neutrophils kill and degrade microbes: an integrated view*, Immunol Rev 2007;219: 88.
201. Neville BW, Damm DD, Allen CM, et al: Oral and maxillofacial pathology ed 3 2009; Saunders St. Louis.
202. Niederman R, Westernoff T, Lee C, et al: *Infection-mediated early-onset periodontal disease in P/E-selectin-deficient mice*, J Clin Periodontol 2001;28: 569.
203. Noiri Y, Ehara A, Kawahara T, et al: *Participation of bacterial biofilms in refractory and chronic periapical periodontitis*, J Endod 2002;28: 679.
204. Nordlund L, Hormia M, Saxen L, et al: *Immunohistochemical localization of epidermal growth factor receptors in human gingival epithelia*, J Periodont Res 1991;26: 333.
205. Nualart Grosllmus ZC, Morales Chavez MC, Slvestre Donat FJ. *Periodontal disease associated to systemic genetic disorders*, Med Oral Patol Oral Cir Bucal 2007;12: E211.
206. Oehlers FAC. *Periapical lesions and residual dental cysts*, Br J Oral Surg 1970;8: 103.
207. Oguntebi B, Slee AM, Tanzer JM, et al: *Predominant*

Telegram: @dental_k

www.konkur.in

microflora associated with human dental periradicular abscesses, J Clin Microbiol 1982;15: 964.

208. Okiji T, Kawashima N, Kosaka T, et al: *Distribution of la antigen-expressing nonlymphoid cells in various stages of induced periapical lesions in rat molars*, J Endod 1994;20: 27.
209. Orstavik D, Pitt Ford TR. Essential endodontology: prevention and treatment of apical periodontitis ed 2 2008; Wiley-Blackwell Philadelphia.
210. Pallasch TJ, Wahl MJ. *Focal infection: new age or ancient history?*, Endod Topics 2003;4: 32.
211. Pascon EA, Leonardo MR, Safavi K, et al: *Tissue reaction to endodontic materials: methods, criteria, assessment, and observations*, Oral Surg Oral Med Oral Pathol 1991;2: 222.
212. Patel S, Wilson A, Dawood A, et al: *The detection of periapical pathosis using periapical radiography and cone computed tomography-Part 1: pre-operative status*, Int Endod J 2012;45: 702.
213. Peciuliene V, Reynaud AH, Balciuniene I, et al: *Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis*, Int Endod 2001;34: 429.
214. Quesnell BT, Alves M, Hawkinson RW, et al: *The effect of human immunodeficiency virus on endodontic treatment outcome*, J Endod 2005;31: 633.
215. Raggatt LJ, Partirdge NC. *Cellular and molecular mechanism of bone remodeling*, J Biol Chem 2010;285: 25103.
216. Reddy SV, Roodman GD. *Control of osteoclast differentiation*, Crit Rev Eukaryotic Gene Expression 1998;8: 1.
217. Reeve CM, Wentz FM. *The prevalence, morphology, and*

Telegram: @dental_k

www.konkur.in

distribution of epithelial cell rests in the human periodontal ligament, Oral Surg 1962;15: 785.

218. Regezi JA, Scubba JJ, Jordan RCK. Oral pathology: clinical pathologic correlations ed 5 2008; Saunders St. Louis.
219. Reinke E, Fabry Z. *Breaking or making immunological privilege in the central nervous system: the regulation of immunity by neuropeptides*, Immunol Lett 2006;104: 102.
220. Reuther T, Schuster T, Mende U, Kubler A. *Osteoradionecrosis of the jaws as a side effect of radiotherapy of head and neck tumor patients—a report of a thirty year retrospective review*, Int J Oral Maxillofacial Surg 2003;32: 289.
221. Richardson JD, Vasko MR. *Cellular mechanisms of neurogenic inflammation*, J Pharmacol Exp Ther 2002;302: 839.
222. Ricucci D, Bergenholtz G. *Histologic features of apical periodontitis in human biopsies*, Endod Topics 2004 2004;8: 68.
223. Ricucci D, Pascon EA, Pitt Ford TR, et al: *Epithelium and bacteria in periapical lesions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 241.
224. Ricucci D, Siqueira JF Jr. Endodontology 2013; Quintessence London.
225. Rosenberg PA, Frisbie J, Lee J, et al: *Evaluation of pathologists (histopathology) and radiologists (cone bean computed tomography) differentiating radicular cysts from granulomas*, J Endod 2010;36: 423-428.
226. Ruggiero SL, Drew SJ. *Osteonecrosis of the jaws and bisphosphonate therapy*, J Dent Res 2007;86: 1013.
227. Rutherford RB, Spangberg L, Tucker M, et al: *The time-course of the induction of reparative dentin formation in*

Telegram: @dental_k

www.konkur.in

monkeys by recombinant human osteogenic protein-1, Arch Oral Biol 1994;39: 833.

228. Sabeti M, Simon JH, Howzari H, et al: *Cytomegalovirus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns*, J Endod 2003;29: 321.
229. Sahara N, Toyoki A, Ashizawa Y, et al: *Cytodifferentiation of the odontoclast prior to the shedding of human deciduous teeth: an ultrastructural and cytochemical study*, Anat Rec 1996;244: 33.
230. Sasaki T. *Differentiation and functions of osteoclasts and odontoclasts in mineralized tissue resorption*, Microscopy Res Tech 2003;61: 483.
231. Sasaki T, Sasaki T, Motegi N, et al: *Dentin resorption mediated by odontoclasts in physiological root resorption of human deciduous teeth*, Am J Anat 1988;183: 303.
232. Sathorn C, Parashos P, Messer HH. *How useful is root canal culturing in predicting treatment outcome?*, J Endod 2007;33: 220.
233. Saunder DN: Interleukin-1 in dermatological disease. In Bomford R, Henderson B, editors: *Interleukin-1, inflammation and disease*, North Holland, 1989, Elsevier.
234. Scapoli C, Mamolini E, Carrieri A, et al: *Gene-gene interaction among cytokine polymorphisms influence susceptibility to aggressive periodontitis*, Genes Immun 2011;12: 473.
235. Schaffer M, Beiter T, Becker HD, et al: *Neuropeptides: mediators of inflammation and tissue repair?*, Arch Surg 1998;133: 1107.
236. Schoenfeld SE, Greening AB, Glick DH, et al: *Endotoxic activity in periapical lesions*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1982;53: 82.
237. Schork NJ, Fallin D, Lanchbury JS. *Single nucleotide*

Telegram: @dental_k

www.konkur.in

polymorphisms and the future of genetic epidemiology, Clin Genet 2000;58: 250.

238. Segura-Egea JJ, Castellanos-Cosano L, Machuca G, et al: *Diabetes mellitus, periapical inflammation and endodontic treatment outcome*, Med Oral Pathol Cir Bucal 2012;17: e356.
239. Segura-Egea JJ, Jimenez A, Rios-Santos JV, et al: *High prevalence of apical periodontitis amongst type 2 diabetic patients*, Int Endod J 2005;38: 564.
240. Seltzer S. Endodontology ed 2 1988; Lea & Febiger Philadelphia.
241. Seltzer S, Bender IB, Zionitz M. *The dynamics of pulp inflammation: correlation between diagnostic data and actual histological finding in the pulp. Part 1 and Part 2*, Oral Surg Oral Med Oral Pathol 1963;16: 846.
242. Seltzer S, Naidorf IJ. *Flare-ups in endodontics: 1. Etiological factors*, J Endod 1985;11: 472.
243. Seo B-M, Miura M, Gronthos S, et al: *Investigation of multipotent postnatal stem cells from human periodontal ligament*, Lancet 2004;364: 149.
244. Serbina NV, Jia T, Hohl TM, Pamer EG. *Monocyte-mediated defense against microbial pathogens*, Ann Rev Immunol 2008;26: 421.
245. Shear M. *The histogenesis of dental cysts*, Dent Pract 1963;13: 238.
246. Shimono M, Suda H, Maeda T, et al: Dentin/pulp complex 1996; Quintessence New York.
247. Sidaravicius B, Aleksejuniene J, Eriksen HM. *Endodontic treatment and prevalence of apical periodontitis in an adult population of Vilnius, Lithuania*, Dent Traumatol 2000;15: 210.
248. Sigurdsson A. *Pulpal diagnosis*, Endod Topics 2003;5:

Telegram: @dental_k

www.konkur.in

12.
249. Silva TA, Garlet GP, Fukada SY, et al: *Chemokines in oral inflammatory diseases: apical periodontitis and periodontal disease*, J Dent Res 2007;86: 306.
250. Silva Costa CP, Abreu Fonseca EB, Carvalho Souza S de F. *Association between sickle cell anemia and pulp necrosis*, J Endod 2013;39: 177.
251. Simon JHS. *Incidence of periapical cysts in relation to root canal*, J Endod 1980;6: 116.
252. Simon JHS, Yonemoto GS, Bakland LK. *Comparison of cellular cementum in normal and diseased teeth—a scanning electron microscope study*, J Endod 1981;7: 370.
253. Singer NG, Caplan AI. *Mesenchymal stem cells: mechanisms of inflammation*, Ann Rev Pathol Mech Dis 2011;6: 457.
254. Siqueira JF Jr, Rocas IN. *Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 85.
255. Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Polymorphism of the FcgammaRIIIa gene and post-treatment apical periodontitis*, J Endod 2011;37: 1345.
256. Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Relationship between Fcgamma receptor and interleukin-1 gene polymorphisms and post-treatment apical periodontitis*, J Endod 2009;35: 1186.
257. Siqueira JF, Lopes HP. *Bacteria on the apical root surfaces of untreated teeth with periradicular lesions: a scanning electron microscopy study*, Int Endod J 2001;34: 216.
258. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J

Telegram: @dental_k

www.konkur.in

Endod 1990;16: 498.
259. Slauson DO, Cooper BJ. Mechanisms of disease ed 3 2002; Mosby St. Louis.
260. Slots J, Nowzari H, Sabeti M. *Cytomegalovirus infection in symptomatic periapical pathosis*, Int Endod J 2004;37: 519.
261. Solheim E. *Growth factors in bone*, Int Orthop 1998;22: 410.
262. Sonoyama W, Seo B-M, Yamaza T, et al: *Human Hertwig's epithelial root sheath cells play crucial roles in cementum formation*, J Dent Res 2007;86: 594.
263. Stashenko F. *The role of immune cytokines in the pathogenesis of periapical lesions*, Endod Dent Traumatol 1990;6: 89.
264. Stashenko P, Teles R, D'Souza R. *Periapical inflammatory responses and their modulation*, Crit Rev Oral Biol Med 1998;9: 498.
265. Stashenko P, Van Dyke T, Tully P, et al: *Inflammation and genetic risk indicators for early periodontitis in adults*, J Periodontol 2011;82: 588.
266. Stashenko P, Wang CY, Riley E, et al: *Reduction of infection-stimulated bone periapical bone resorption by the biological response modifier PGG glucan*, J Dent Res 1995;74: 323.
267. Stashenko P, Yu SM. *T helper and T suppressor cell reversal during the development of induced rat periapical lesions*, J Dent Res 1989;68: 830.
268. Steed DL. *The role of growth factors in wound healing*, Surg Clin North Am 1997;77: 575.
269. Stern MH, Dreizen S, Mackler BF, et al: *Quantitative analysis of cellular composition of human periapical granulomas*, J Endod 1981;7: 117.

Telegram: @dental_k

www.konkur.in

270. Stern MH, Dreizen S, Mackler BF, et al: *Isolation and characterization of inflammatory cells from the human periapical granuloma*, J Dent Res 1982;61: 1408.
271. Sunde P, Olsen I, Debelian G, et al: *Microbiota of periapical lesions refractory to endodontic therapy*, J Endod 2002;28: 304.
272. Sundqvist G. Bacteriological studies of necrotic dental pulps 1976; Dissertation Umea, Sweden.
273. Sundqvist G. *Taxonomy, ecology, and pathogenicity of the root canal flora*, Oral Surg Oral Med Oral Pathol 1994;78: 522.
274. Sundqvist G, Eckerbom MI, Larsson AP, et al: *Capacity of anaerobic bacteria from necrotic pulp to induce purulent infection*, Infect Immun 1979;25: 685.
275. Sundqvist G, Figdor D. *Life as an endodontic pathogen*, Endod Topics 2003 2003;6: 3.
276. Sundqvist G, Figdor D, Persson S, et al: *Microbiological analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 86.
277. Sundqvist G, Reuterving CO. *Isolation of Actinomyces israelii from periapical lesion*, J Endod 1980;6: 60.
278. Syrjanen S, Tammisalo E, Lilja R, et al: *Radiographical interpretation of the periapical cysts and granulomas*, Dentomaxillofac Radiol 1982;11: 89.
279. Tagger M, Massler M. *Periapical tissue reactions after pulp exposure in rat molars*, Oral Surg Oral Med Oral Pathol 1975;39: 304.
280. Takahashi K. *Microbiological, pathological, inflammatory, immunological and molecular biological aspects of periradicular disease*, Int Endod J 1998;31: 311.

Telegram: @dental_k

www.konkur.in

281. Takeuchi O, Hoshino K, Akira S. *Cutting edge: TLR2-deficient and MyD88-deficient mice are highly susceptible to Staphylococcus aureus infection*, J Immunol 2000;165: 5392.
282. Tani N, Kuchiba K, Osada T, et al: *Effect of T-cell deficiency on the formation of periapical lesions in mice: histological comparison between periapical lesion formation in BALB/c and BALB/c nu/nu mice*, J Endod 1995;21: 195.
283. Teitelbaum SL, Tondravi MM, Ross FP. *Osteoclasts, macrophages, and the molecular mechanisms of bone resorption*, J Leukocyte Biol 1997;61: 381.
284. Teles R, Wang CY, Stashenko P. *Increased susceptibility of RAG-2 SCID mice to dissemination of endodontic infections*, Infect Immun 1997;65: 3781.
285. Ten Cate AR. *The epithelial cell rests of Malassez and the genesis of the dental cyst*, Oral Surg Oral Med Oral Pathol 1972;34: 56.
286. Teronen O, Salo T, Laitinen J, et al: *Characterization of interstitial collagenases in jaw cyst wall*, Eur J Oral Sci 1995;103: 141.
287. Thesleff I. *Epithelial cell rests of Malassez bind epidermal growth factor intensely*, J Periodont Res 1987;22: 419.
288. Toller PA. *The osmolality of fluids from cysts of the jaws*, Br Dent J 1970;129: 275.
289. Torabinejad M. *Mediators of acute and chronic periradicular lesions*, Oral Surg Oral Med Oral Pathol 1994;78: 511.
290. Torabinejad M, Eby WC, Naidorf IJ. *Inflammatory and immunological aspects of the pathogenesis of human periapical lesions*, J Endod 1985;11: 479.

Telegram: @dental_k

www.konkur.in

291. Torabinejad M, Kettering J. *Identification and relative concentration of B and T lymphocytes in human chronic periapical lesions*, J Endod 1985;11: 122.
292. Torabinejad M, Kiger RD. *Experimentally induced alterations in periapical tissues of the cat*, J Dent Res 1980;59: 87.
293. Torabinejad M, Walton RE. Endodontics: principles and practice ed 4 2009; Saunders St. Louis.
294. Trombelli L, Lee MB, Promsudthi A, et al: *Periodontal repair in dogs: histologic observations of guided tissue regeneration with a prostaglandin E1 analog/methacrylate composite*, Clin Periodontol 1999;26: 381.
295. Tronstad L, Barnett F, Cervone F. *Periapical bacterial plaque in teeth refractory to endodontic treatment*, Dent Traumatol 1990;6: 73.
296. Tronstad L, Barnett F, Riso K, Slots J. *Extraradicular endodontic infection*, Dent Traumatol 1987;3: 86.
297. Trott JR, Chebeb F, Galindo Y. *Factors related to cholesterol formation in cysts and granulomas*, J Cand Dent Assoc 1973;38: 76.
298. Trowbridge H, Emling RC. Inflammation: a review of the process ed 5 1997; Quintessence Chicago.
299. Tuan T-L, Nichter LS. *The molecular basis of keloid and hypertrophic scar formation*, Mol Med Today 1998;4: 19.
300. Tzaifas D, Alvanou A, Papadimitrious S, et al: *Effects of recombinant basic fibroblast growth factor, insulin-like growth factor-II and transforming growth factor-b 1 on dog dental pulp cells in vivo*, Arch Oral Biol 1998;43: 431.
301. van Oosterhout AJ, Bloksma N. *Regulatory T-lymphocytes in asthma*, Eur Respir J 2005;26: 918.
302. van Winkelhoff AJ, Carless AW, de Graaff J. *Bacteroides endodontalis and other black-pigmented Bacteroides*

Telegram: @dental_k

www.konkur.in

species in odontogenic abscesses, Infect Immun 1985;49: 494.

303. Vier FV, Figueiredo JA. *Prevalence of different periapical lesions associated with human teeth and their correlation with the presence and extension of apical external root resorption*, Int Endod J 2002;35: 710.
304. Wadachi R, Hargreaves KW. *Trigeminal nociceptors express TLR-4 and CD14: a mechanism for pain due to infection*, J Dent Res 2006;85: 49.
305. Wakelin D, Roitt I, Mims C, et al: Mims' medical microbiology ed 4 2008; Mosby Philadelphia.
306. Wallstrom JB, Torabinejad M, Kettering J, et al: *Role of T cells in the pathogenesis of periapical lesions: a preliminary report*, Oral Surg Oral Med Oral Pathol 1993;76: 213.
307. Walton RE, Ardjmand K. *Histological evaluation of the presence of bacteria in induced periapical lesions in monkeys*, J Endod 1992;18: 216.
308. Wang CY, Stashenko P. *Kinetics of bone-resorbing activity in developing periapical lesions*, J Dent Res 1991;70: 1362.
309. Wang CY, Tani-Ishii N, Stashenko P. *Bone resorptive cytokine gene expression in developing rat periapical lesions*, Oral Microbiol Immunol 1997;12: 65.
310. Warren JR, Scarpelli DG, Reddy JK, et al: Essentials of general pathology 1987; Macmillan Publishing New York.
311. Wayman BE, Murata SM, Almeida RJ, Fowler CB. *A bacteriological and histological evaluation of 58 periradicular lesions*, J Endod 1992;18: 152.
312. Weiger R, Manncke B, Werner H, Lost C. *Microbial flora of sinus tracts and root canals of non-vital teeth*, Endod Dent Traumatol 1995;11: 15.

Telegram: @dental_k

www.konkur.in

313. Werner S, Grose R. *Regulation of wound healing by growth factors and cytokines*, Physiol Rev 2003;83: 835.
314. Williams BL, McCann GF, Schoenknecht FD. *Bacteriology of dental abscesses of endodontic origin*, J Clin Microbiol 1983;18: 770.
315. Williams GT, Williams WJ. *Granulomatous inflammation—a review*, J Clin Pathol 1983;36: 723.
316. Williams JZ, Barbul A. *Nutrition and wound healing*, Surg Clin North Am 2003;83: 571.
317. Woo S-B, Hellstein JW, Kalmar JR. *Systematic review: bisphosphonates and osteonecrosis of the jaws*, Ann Int Med 2006;144: 753.
318. World Health Organization. Application of the international classification of disease to dentistry and stomatology ed 3 1995; WHO Geneva.
319. Wu JR, Zeng R. *Molecular basis for population variation: from SNPs to SAPs*, FEBS Lett 2012;586: 2841.
320. Yamamoto K, Kobayashi T, Grossi S, et al: *Association of Fcgamma receptor IIa genotype with chronic periodontitis in Caucasians*, J Periodontol 2004;75: 517.
321. Yamasaki M, Kumazawa M, Kohsaka T, et al: *Pulp and periapical tissue reactions after experimental pulpal exposure in rats*, J Endod 1994;20: 13.
322. Yamasaki M, Nakane A, Kumazawa M, et al: *Endotoxin and gram-negative bacteria in the rat periapical lesion*, J Endod 1992;18: 501.
323. Yu SM, Stashenko P. *Phenotypic characterization of inflammatory cells in induced rat periapical lesions*, J Endod 1987;13: 535.
324. Yusulf H. *The significance of the presence of foreign material periapically as a cause of failure of root treatment*, Oral Surg Oral Med Oral Pathol 1982;54: 566.

Telegram: @dental_k

www.konkur.in

325. Zimmerli W, Lew PD, Walvogel FA. *Pathogenesis of foreign body infection: evidence for a local granulocyte defect*, J Clin Invest 1984;73: 1191.
326. Zimmerli W, Waldvogel FA, Vaudaux P, et al: *Pathogenesis of foreign body infection: description and characteristics of an animal model*, J Infect Dis 1982;146: 487.
327. Zimring JC, Kapp JA. *Identification and characterization of CD81 suppressor T cells*, Immunol Res 2004;29: 303.

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PART III
Advanced Clinical Topics

OUTLINE

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17: Evaluation of outcomes

Yuan-Ling Ng, Kishor Gulabivala

CHAPTER OUTLINE

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Context of evaluating endodontic outcomes

The development of medical and dental practices has been guided by prevailing philosophies and consensus of expert opinions. The foundations of modern endodontics were shaken when Billings[97] brought the apparent relationship between oral sepsis and bacterial endocarditis to the attention of dentistry and medicine. He had strengthened the concept further from Miller,[134] who had coined the term *focus of infection* to highlight a possible link between "mouth germs" and systemic disease. The disastrous consequences of the focal infection era were sealed when Hunter[96] delivered his famous address at McGill University. Fears of fatal oral sepsis from deficient root canal treatments led to widespread extraction of pulpless teeth. Endodontics virtually disappeared from many dental schools, whereas in some areas, treatment was restricted to anterior teeth. The focal infection theory reigned for about 50 years[83] until around 1940.

The discipline of endodontics was rescued by individual, diligent practitioners in Europe and the United States who meticulously recorded their treatments, as well as their outcomes, to demonstrate the effectiveness of the procedures in controlling root canal infection. It was through these individual endeavors of merit that the reputation of endodontic procedures was restored and the discipline granted its specialist status in 1952 in the United States.[59]

Toward the 1990s, the improvements in general health and longer survival of Western populations as well as their teeth, coupled with the rising costs of health care for longer-living individuals, prompted a host of reevaluations of the altered economic burdens on society. Among them, the *cost-effectiveness of treatment procedures* for management of diseases loomed large. Thus began the era of evidence-based medicine and dentistry, with its emphasis on costs, benefits, and outcomes of treatment. Attempts to pool outcome data for greater power brought with it the realization that studies originating from

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different centers varied in diverse ways, leading to very heterogeneous data that challenged formal attempts at drawing definitive conclusions. Characterization of data types and its quality prompted calls for standardization of approaches in measuring outcomes to enable more meaningful pooling.

Because of the better established science of evidence assessment, a modern revisitation of the "focal infection era"[94] did not lead to the same threat to the endodontic discipline that was evident at the beginning of the 20th century. In fact, it was suggested as an opportunity to secure research funding for assessing and managing the importance of dental care on systemic health.

Interestingly, the modern threat to endodontics was posed by the cost-economic pressures exerted on treatment planning decisions centering on the question of whether to "save the tooth" or extract it with replacement by an implant-supported crown.[238] Once again, the science of evidence-based practice has helped to avert irrational treatment recommendations leaning toward extraction of savable teeth.[54,100,103]

Types of disease and their treatment

The statement, "Endodontists provide endodontic treatment to manage endodontic disease" is a gross oversimplification that masks important subtleties in recognition of the nature of the diseases and how best to address them. Endodontists manage inflammation of the specialized connective tissues within and surrounding the teeth; more specifically, they deal with inflammation that generally commences in the pulp tissue and progresses to the periradicular tissues via portals of communication that convey the neurovascular supply. Incipient or established pulpitis may be managed by vital pulp therapy when judged reversible, largely because a sufficient body of pulp tissue remains healthy and unaffected. More advanced pulpitis that approaches the foramina into the periodontal ligament may require root canal treatment. When pulpal inflammation, necrosis, or infection encroaches on the apical periodontal ligament, root canal treatment is required. Apical periodontitis that persists despite technically adequate root canal treatment may require root canal retreatment, periradicular surgery, or extraction to manage the source of the persistent disease. *Endodontic treatment* is therefore a collective and nonspecific term for a range of procedures directed at

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managing the spread of pulpal inflammation or infection. Endodontic treatment encompasses the following procedures:

1. Vital pulp therapy (indirect pulp therapy, direct pulp capping, pulpotomy, regenerative pulp therapy)
2. Nonsurgical root canal treatment
3. Nonsurgical root canal retreatment
4. Surgical retreatment

The ideal outcome for endodontic treatment consists of the controlled reduction of inflammation, accompanied by healing through regeneration, although sometimes repair may follow instead. Unfortunately, none of the involved tissues are within the direct sight of the clinician, hidden as they are by their housings of hard tooth structure or alveolar bone and their gingival/mucosal coverings. Consequently, surrogate measures must be employed to evaluate the presence or absence of the disease process and its resolution. The evaluation process is further complicated by the lack of direct correlation between measures of the disease process and its clinical manifestation.

What are surrogate outcome measures?

Signs of acute inflammation are classically described in the “triple response” exhibited by mechanically injured skin, which includes altered color (redness), texture/contour (swelling), and sensation (pain), which are directly accessible and viewable. These changes have a direct correlation with histopathologic and molecular alterations. Chronic inflammation does not necessarily exhibit the same highly visible signs and symptoms of its histopathologic character. When hidden from view, as are pulpal and periradicular tissues, the task of recognizing the presence or absence of chronic inflammation is even more challenging. The clinician is therefore left with indirect or associated (proxy) changes by which to judge the presence or absence of disease; these are called “surrogate” measures. Some of these may have to be inferred through associations, some may be directly visualized, and others are only indirectly “viewable” through various imaging techniques (such as radiography). The process therefore calls upon the clinician to

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assimilate various sources of information to form a judgment about the presence or absence of disease.

Types of outcome measures

In its broadest sense, an outcome measure for a treatment intervention may constitute any consistently anticipated and measurable consequence of the treatment. The prepared shape of the root canal system, bacterial load reduction, and technical quality of the root filling may all be regarded as outcome measures by this definition. But the ultimate *clinical* measure of a treatment outcome is assessing the prevention and resolution of disease.

The outcome of endodontic treatment may be assessed in four dimensions as in other medical disciplines.[9] The first dimension is physical/physiologic and related to presence or absence of pulpal/periapical health/disease, pain, and function. The second dimension assesses longevity or tooth survival. The third dimension relates to economics and assesses direct and indirect costs. Finally, the fourth dimension examines psychologic aspects involving perceptions of oral health–related quality of life (OHRQoL) and aesthetics.

What is the purpose of evaluating outcomes?

Apart from the importance of developing a solid foundation of evidence-based practice, it is important to evaluate treatment outcomes for a number of reasons.

Effectiveness of procedures

First, treatment procedures must be effective. Otherwise, there is no reason to recommend them to patients as a treatment option. The patient must be properly informed as to the risks, benefits, and potential outcomes of the offered treatment. The availability of pooled outcome data and consensus guidelines offers both patients and endodontists reassurance and confidence in the validity and predictability of the offered procedure. However, the pooled average outcome data may not pertain if the clinician does not have the requisite experience, skill, and personal outcomes matching at least these average figures of performance. Personal outcome data offer the patient more precise measures for comparison and expectation. It also aids practitioners in

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refining their technique and knowledge to further improve their outcomes and, ultimately, to help improve the overall pooled data.[34] The treating clinician must assess his or her own personal outcome data and personal expectation of success. If the clinician is uncertain of a result or feels that the outcome could be enhanced by treatment from another clinician, then referral to someone else more qualified is paramount.

Factors affecting outcomes

Pooling data (that is preferably homogeneous) offers the potential to evaluate and prioritize the factors that exert a dominant influence on outcomes. In this way, protocols for treatment may be improved in a progressive fashion, bringing about the perfect meld of technical, clinical, and biologic insight necessary for the highest performance and most predictable outcome. Evaluation of factors affecting treatment outcomes arguably provides the strongest influence for making changes that will improve the clinical outcomes. Weighing the relative importance of individual factors should help to identify the key biologic factors at play and how best to manage them from a clinical or technical perspective.

Value for prognostication

Prognostication, which could be defined as the prediction, projection, prophesizing, or foretelling the likely outcome of treatment, is not often well defined in endodontics. The overall prognosis of a tooth depends on the interaction among three individual and often independent variables, including endodontic, periodontal, and restorative prognoses. Each has a set of subsidiary factors that must be considered to derive an overall prognosis. Finally, the tooth in question must be considered from a strategic perspective, relative to its position in the dental arch and the contribution it makes in the dynamic occlusion.

A deeper analysis of the factors affecting outcomes as prognostic indicators is merited in order to clarify the degree of complexity inherent to the problem. A comparison may be made with the randomized controlled trial of a drug as a treatment intervention. The drug therapy is delivered as a clearly prescribed and standardized dose regimen delivered by specified timings to effect an anticipated blood or target tissue concentration for a

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specified duration. Data recording is confined to compliance with the prescription and possibly to the checking of actual blood levels achieved as well as the final outcome effect.

In stark contrast, a surgical intervention, regardless of any standardization of the described procedural protocol, is subject to enormous variation in its delivery dependent on interpretation and execution of the protocol by each operator. To add to the levels of complexity and variation, surgical protocols are often multistep, sequential procedures, each prospective step dependent on the previous for its efficacy. Even characterizing and accurately recording any variations in treatment protocols becomes a challenge because many aspects and steps must be documented. This leads to the next challenge, that of demonstrating compliance with the accuracy of such complex data gathering. Furthermore, it becomes important from an analytic perspective to not only consider the effect of individual steps (factors) alone but also any interactive effect between the multiple steps inherent in the procedure. Even when such comprehensive prospective outcome data are available, utilizing these data to individually calculate the prognosis in a particular scenario may be challenging. Clinically, there may be two distinct ways to estimate prognosis: (1) to apply heuristic principles and intuitively gauge the effect of dominant factors or, at the other end of the spectrum, (2) to mathematically input comprehensive data into an algorithmic model to calculate the estimated outcome. An even more sophisticated approach may be to use mathematical modeling to calculate iterations on variations within the system. At present, the evidence base is insufficient to allow such a sophisticated approach.

It therefore follows that evaluating outcomes and the factors that affect them are fundamental to the creation of a suitable data foundation for future application to endodontic prognostication. The endodontic treatment option is in competition with other alternative therapies and therefore for the discipline to survive and thrive, a suitable blend of efficacy, efficiency, utility, predictability, and cost-effectiveness data must be generated for its principal therapies. Achievement of such a goal will require a diligent and conscientious collection of data from many sources. Analyses of such powerful datasets will ultimately yield proper insight into outcomes of the procedures we use. Coupled with biologic insight of the problem, it will become possible to derive new therapeutic solutions necessary to deliver

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them.

Outcome measures for endodontic treatment

Before determining the success or failure of an endodontic procedure, the parameters as to what is considered a success or a failure have to be discussed. These are termed the *outcome measures* of the procedures.

Outcome measures for vital pulp therapy procedures

The techniques used to maintain the vitality and health of pulps in teeth with extensive caries or those with traumatic/mechanical exposures include (1) indirect pulp capping with one-step or stepwise caries excavation, (2) direct pulp capping of exposed pulps, and (3) partial/full pulpotomy procedures for more extensively involved pulps. The surrogate outcome measures adopted in studies include (1) clinical success (pulp sensitivity to cold test and absence of pain, soft-tissue swelling, sinus tract, periradicular radiolucency, or pathologic root resorption), (2) patient satisfaction, (3) adverse events (pain, swelling, tooth fracture), and (4) tooth extraction.[89,136] Although not providing a specific follow-up strategy, the quality guidelines of the European Society of Endodontology (ESE)[61] suggests “initial review at no longer than 6 months and thereafter at further regular intervals.” They also provide the criteria for judging the favorable outcome of vital pulp therapy (Table 17.1). There are substantial differences in the criteria adopted in published clinical studies on outcomes of vital pulp therapy[89,136] with few studies adopting all of the criteria. The frequency of radiologic review adopted in published studies also varies substantially, with some recommending the first review at 1 month followed by subsequent 3 monthly reviews.[172,242] The relative benefit of acquired information versus unnecessary radiation to patients has been questioned.[66] An initial assessment at 6 to 12 weeks, followed by a review 6 and 12 months after treatment, seems to have been accepted and is recommended. Some studies reported up to 10 years follow-up on case series demonstrating lower success rates, inferring the possibility of slow spread of pulpal inflammation and late failures. The conclusion is that longer follow-up periods are merited.

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Table 17.1

Criteria for Assessing the Outcome of Pulp Therapy

Quality Guidelines for Endodontic Treatment: Consensus Report of the European Society of Endodontology (2006)	Guidelines on Pulp Therapy for Primary and Immature Permanent Teeth (American Academy of Pediatric Dentistry, 2014)
1. Normal response to pulp sensitivity tests (when feasible) 2. Absence of pain and other symptoms 3. Radiologic evidence of dentinal bridge formation 4. Radiologic evidence of continued root formation in immature teeth 5. Absence of clinical and radiographic signs of internal root resorption and apical periodontitis	1. Tooth vitality maintained 2. Absence of posttreatment signs or symptoms such as sensitivity, pain, or swelling 3. Occurrence of pulp healing and reparative dentin formation 4. Absence of radiographic evidence of internal or external root resorption, periapical radiolucency, abnormal calcification, or other pathologic changes 5. Teeth with immature roots should show continued root development and apexogenesis

The process at each review consists of obtaining a history of symptoms, coupled with an examination to determine the presence or absence of tenderness to palpation of adjacent soft tissues, tenderness to pressure and percussion of the tooth, signs of radiographic pulpal, periapical changes, and responses to pulp tests. The accuracy of pulp tests may be limited in pulpotomized teeth because of the distance of the remaining pulp tissue from the tooth's surface. In the case of pulp capping and pulpotomy, additional tests include radiographic verification of the presence of the calcific barrier (Fig. 17.1) and its integrity by removal of the dressing and direct probing. Although an initial examination at 6 weeks has been suggested, this can be modified for the radiographic assessment. If there is no evidence of complete bridge formation, the treatment is considered failed and root canal treatment should be considered. In addition, in the case of incompletely formed roots there should be radiographic evidence of continued root development (Fig. 17.2).

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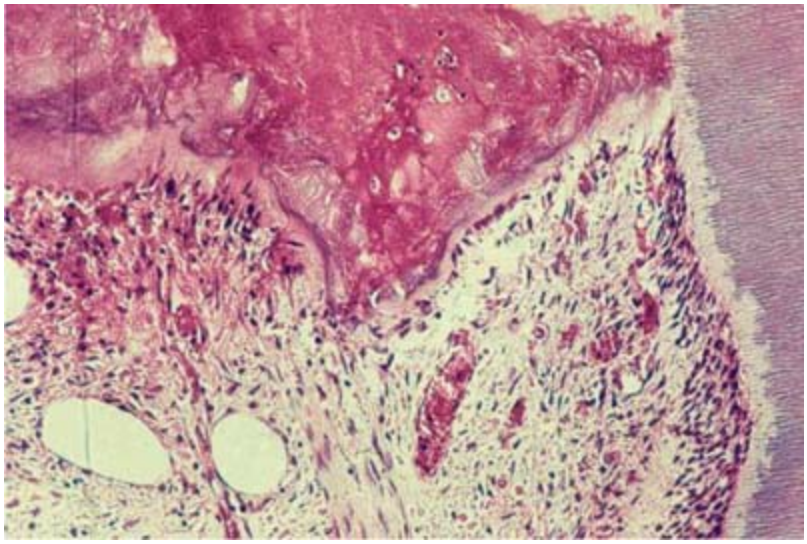

FIG. 17.1 Histologic view of intact calcific bridge formation following pulpotomy.

Stained micrograph of tooth shows a discolored oval and a circular structure surrounded by connective tissues in the pulp.

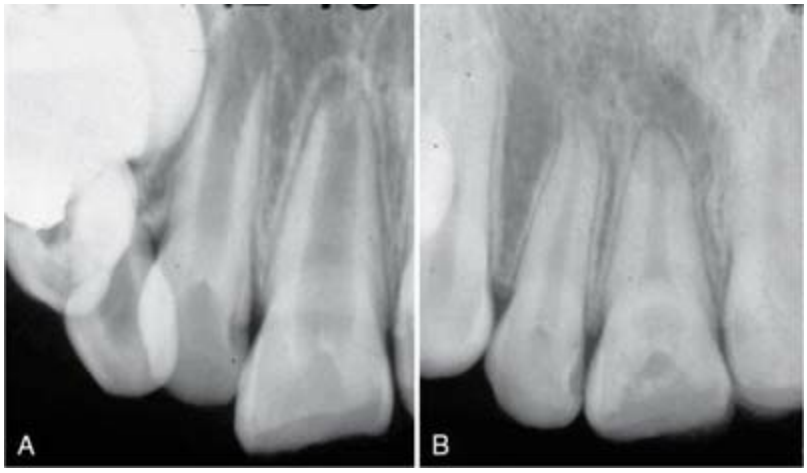

FIG. 17.2 Immature maxillary right lateral and central incisors **(A)** showing continued root development following pulpotomies **(B)**.

A) Radiograph of teeth shows radiolucent pulp extending below the root apices.

B) Radiograph shows teeth surrounded by less-density tissue below gum line and a radiolucent patch at one of the tooth.

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Outcome measures for nonsurgical root canal treatment and retreatment

Root canal treatment may be employed either to prevent or resolve periapical disease. Given that periapical lesions develop as a result of the *interaction* between bacteria (and their by-products) and the host defenses, it is clear that prevention or resolution of the disease process depends on preventing or terminating this interaction.

Prevention of apical periodontitis applies to the clinical situation where it is judged that the pulp is irreversibly inflamed, necrotic, or infected to the extent that vital pulp therapy would not resolve the problem, which therefore requires pulpectomy. The implication is that the procedure demands asepsis as a prime requirement. The precise technical details of the protocol may not be so important, as long as they are focused on effective and aseptic removal of the pulp tissue. This is validated by the high chance of retaining periapical health (judged by conventional radiography), regardless of the clinical protocol used.[153]

Once the periapical lesion has become established, the challenge is a different one because the purpose now is to remove the bacterial biofilm and to effect switching off of the periapical host response. The challenge seems to be greater still if the periapical lesion is larger as it is associated with a more diverse infection. A number of approaches (protocols) have been used to achieve this general aim. The periapical healing process that occurs after root canal treatment is less clear. Nevertheless, ideal healing would eventually result in regeneration and the formation of cementum over the apical termini, isolating the root canal system from the periapex (Fig. 17.3), but this is not an inevitable result. Incomplete removal of the infection will reduce but not eliminate the periradicular inflammatory reaction, and in fact this is generally what happens.[147] This implies that residual infection in the apical anatomy is typical following completion of root canal treatment and that an ongoing interaction continues between the residual infection, root filling material, and host defenses, which plays a definitive role in determining the final healing outcome.

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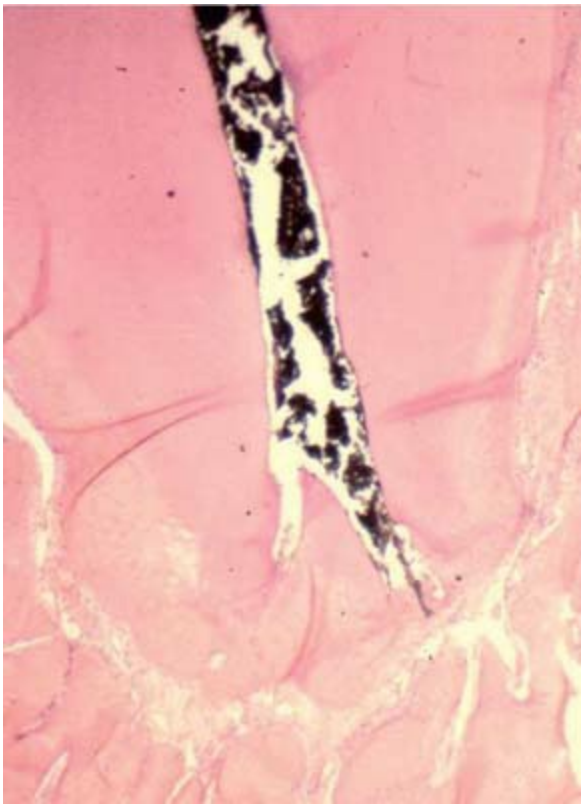

FIG. 17.3 Low-power view of healing by cementum formation when Sealpex is used (black particles are residual Sealapex and root filling material).

Microscopic image shows black residues extending to one end in the root.

Source: (Courtesy Prof. M. Tagger.)

The culture test has been used during root canal treatment as an interim measure of the efficacy of the chemomechanical procedure; however, it has fallen out of favor in contemporary practice for a variety of reasons.[139] The outcome measures that quantitate healing subsequent to root canal treatment are the absence of clinical signs and symptoms of persistent periapical disease.[17,18] The definitive outcome measure (in conjunction with the absence of signs and symptoms), however, is periapical healing, because the treatment is aimed at resolution of periapical disease (Fig. 17.4).[152] Clinical judgment of the outcome of treatment is based on the absence of signs of infection and inflammation, such as pain, tenderness to pressure/percussion of the tooth, tenderness to palpation of the related soft tissues, absence of swelling and sinus tract, and radiographic demonstration of reduction in the

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size of the periapical lesion (if sufficient time has lapsed), with a completely normal development of the periodontal ligament space. Although the majority of periapical lesions heal within 1 year, healing may continue for up to 4 years or longer.[229]

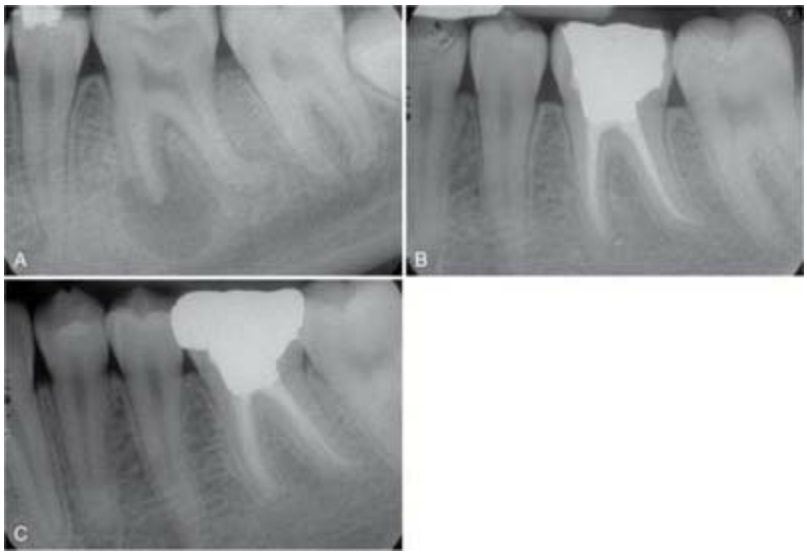

FIG. 17.4 Resolution of periapical disease after root canal treatment of the mandibular left molar with preoperative periapical radiolucency associated with mesial and distal roots **(A)**, delayed healing caused by extruded filling material from the distal canal **(B)** and nearing complete healing as extruded material is resorbed **(C)**.

Three radiographs marked A through C as follows:

A) A less-density patch at the root tip of canal in first molar.

B) The first molar shows filled crown and root canals. The tissue is less-dense below the gum line and inside the roots.

C) The first molar has less-density tissue below the gum line on one side of the molar tooth.

Absence of signs and symptoms of periapical disease with radiographic evidence of a persistent periapical radiolucency may indicate either fibrous repair (Fig. 17.5) or persistent chronic inflammation or infection. Only time and acute exacerbation would identify the latter, whereas the former should remain asymptomatic.

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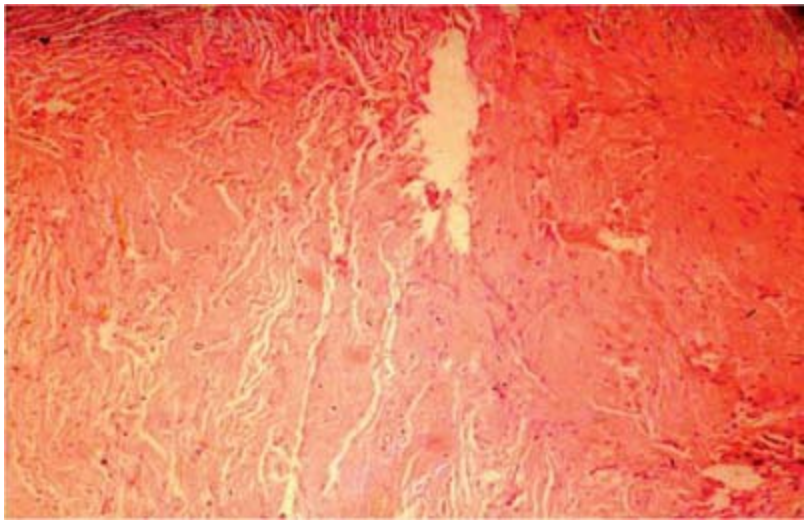

FIG. 17.5 Histologic view of fibrous periapical healing.

A large light scar with fibers is shown in the dark background.

Longevity measures include survival of the root canal fillings or treatment[118,228,236] and tooth retention or survival.[111,149,190] The term *functional retention* was coined by Friedman in 2004[69] to mean retention of the tooth in the absence of signs and symptoms regardless of the radiographic presence of a lesion. The term *functionality* should further and more specifically cover the functional utility of the tooth—that is, some patients may complain that despite the absence of specific signs and symptoms of infection or inflammation, they find it impossible to use the tooth because it "feels" weak.

Modern definitions of health embrace a broader spectrum of measures including psychological well-being.[116] Instruments to assess these aspects have been developed in general medicine and adapted for application to dentistry, such as the Oral Health Related Quality of Life instrument.[55,87,115] There is no definitive instrument for measuring these aspects in endodontics as yet. Studies published so far have mainly adapted versions of the Oral Health Impact Profile (OHIP).

The periapical status of root-treated teeth has traditionally been assessed using two-dimensional conventional radiographic imaging. The use and limitations of two-dimensional radiographic images for assessing treatment outcome has been reviewed thoroughly and is reported to suffer from low sensitivity, particularly in the posterior parts of the mouth.[98] The

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development of digital imaging technology[80] brought the possibility of image manipulation, including digital subtraction,[50,158,243,259] densitometric analysis,[156] correction of gray values,[32] and the manipulation of brightness and contrast.[75] However, none of these approaches has addressed the main deficiencies of accurately elucidating and quantitating the actual existence or progression of periapical bone loss.

Cone-beam computed tomography (CBCT), a new three-dimensional imaging technique requiring only 8% of the effective dose of conventional computed tomography,[119] has been proposed as a means of overcoming the problem of superimposition of tissue layers and structures. The diagnostic values of periapical radiography and CBCT were compared on dogs' teeth using histology as the comparative method of determining apical periodontitis 180 days after root canal treatment.[44] CBCT was found to be significantly more accurate in detecting minor bone lesions compared with two-dimensional radiology.[44,174] Other evidence to support the superior sensitivity of CBCT is available from studies using a variety of simulated defects created in bone in pig[226] or human[165,221] jaws. Although the validity of clinical outcome data derived using conventional radiographic techniques has been questioned,[256] the *routine use* of CBCT is not recommended[62,91,197] owing to its higher radiation dosage.[5,91] The use of CBCT, which may be more sensitive for detection of periapical healing,[114,167] may give lower healing rates and longer durations to complete healing.

Many studies consider the treatment successful only when both radiographic and clinical criteria are satisfied.[69] It has been documented that a small proportion of cases present with persistent symptoms despite complete radiographic resolution of the periapical lesion.[176] Comparison of success rates estimated with or without clinical examination revealed no[93] or only a very small difference (1%).[150]

The widely accepted definition for endodontic success and failure by Strindberg[229] embraces both radiographic and clinical findings (Table 17.2). Friedman and Mor[69] preferred the terms *healed*, *healing*, and *diseased* instead of *success* and *failure* because of the potential of the latter to confuse patients. The "healed" category corresponds to "success" as defined by Strindberg,[229] whereas "healing" corresponds to "success" as defined by

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Bender and colleagues (see Table 17.2).[17,18]

Table 17.2

Criteria for Determination of Periapical Status

Strindberg (1956)	Bender et al. (1966, a and b)	Friedman and Mor (2004)
Success	**Success**	**Healed**
Clinical: No symptoms *Radiographic:* The contours, width, and structure of the periodontal margin were normal OR The periodontal contours were widened mainly around the excess filling	*Clinical:* Absence of pain/swelling; disappearance of fistula; no loss of function; no evidence of tissue destruction *Radiographic:* An eliminated or arrested area of rarefaction after a posttreatment interval of 6 months to 2 years	*Clinical:* Normal presentation *Radiographic:* Normal presentation
Failure		**Diseased**
Clinical: Presence of symptoms *Radiographic:* A decrease in the periradicular rarefaction OR Unchanged periradicular rarefaction OR An appearance of new rarefaction or an increase in the initial rarefaction		Radiolucency has emerged or persisted without change, even when the clinical presentation is normal OR Clinical signs or symptoms are present, even if the radiographic presentation is normal
Uncertain		**Healing**
Radiographic: There were ambiguous or technically unsatisfactory control radiographs that could not, for		*Clinical:* Normal presentation *Radiographic:* Reduced

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some reason, be repeated OR The tooth was extracted prior to the 3-year follow-up owing to the unsuccessful treatment of another root of the tooth		radiolucency

The long duration of the periapical healing process coupled with the reduced recall rates at longer follow-up periods has provided opportunity for setting the thresholds of success at either complete healing or partial healing (reduced lesion size). The success criteria used for complete resolution have been described either as "strict"[152] or "stringent,"[69] whereas the criteria established for reduction in periapical radiolucency size has been described as either "loose"[152] or "lenient."[69] The frequency of adoption of these two thresholds has been similar in previous studies; the expected success rates using "strict" criteria would be expected to be lower than those based on "loose" criteria. The literature finds differences that vary from 4% to 48%.[153]

A periapical index (PAI) consisting of five points on the scale was devised for measuring the periapical status and used in some studies.[157,159] However, the studies only reported the increase or decrease in mean scores for the clinical factors under investigation coupled with the proportion of successful cases. This approach precluded direct comparison of their data with studies reporting conventional dichotomized outcomes. Other studies using the index dichotomized the scores into "healthy" (PAI 1 or 2) or "diseased" (PAI 3 to 5) categories,[160] thus allowing the data to be compared directly with the more traditionally used binary outcomes of success or failure. In this system of designation, given that the PAI score 2 represents periodontal ligament widening, it effectively signals adoption of the "loose" threshold. The longitudinal follow-up[86] of 14 cases presenting with widened apical periodontal ligament (PAI score at 2) for 10 years revealed unfavorable future healing in only a small proportion of the cases (28%, 4/14).

Outcome measures for periapical surgery

Nonsurgical root canal treatment alone may fail to resolve apical periodontitis in a small proportion of cases because it may not allow access to the infection

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or resolution of the cause of the infection (i.e., root fracture or crestal bone communication to the periapical infection). Where the cause is microbial persistence, the infection may be localized intraradicularly (in the apical canal anatomy [Fig. 17.6] or in the apical dentinal tubules [Fig. 17.7] or extraradicularly [Fig. 17.8]). In these instances, a surgical approach to the periapex may be required in addition to the nonsurgical approach to removing such microbes (see Fig. 17.7).

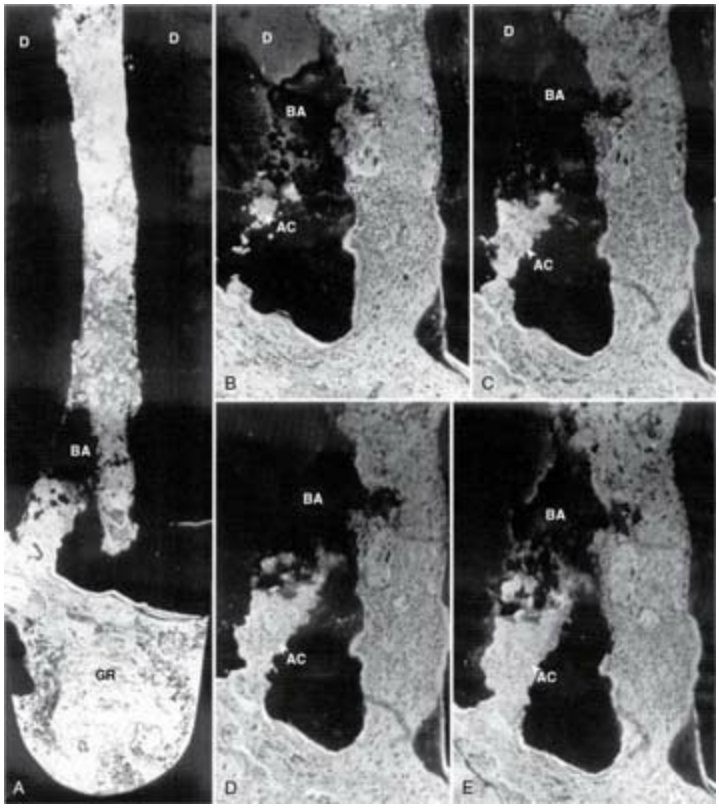

FIG. 17.6 Axial sections through the surgically removed apical portion of the root with a therapy-resistant periapical lesion *(GR)*. Note the cluster of bacteria *(BA)* visible in the root canal. Parts (**B**)–(**E**) show serial semithin sections taken at varying distances from the section plane of **(A)** to reveal the emerging **(B)** and gradually widening **(C–E)** profiles of an accessory root canal *(AC)*. Note that the accessory canal is clogged with bacteria *(BA)*. Original magnification: **A,** ×52; **B–E,** ×62.

Five radiographs marked A through E show bacterial colonies above GR in the radiograph A and increasing accessory canal below BA from radiograph B to E.

Source: (From Nair PN, Sjogren U, Krey G, Sundqvist G: Therapy-resistant foreign body giant cell granuloma at the periapex of a root-

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filled human tooth, *J Endod* 16:589, 1990.)

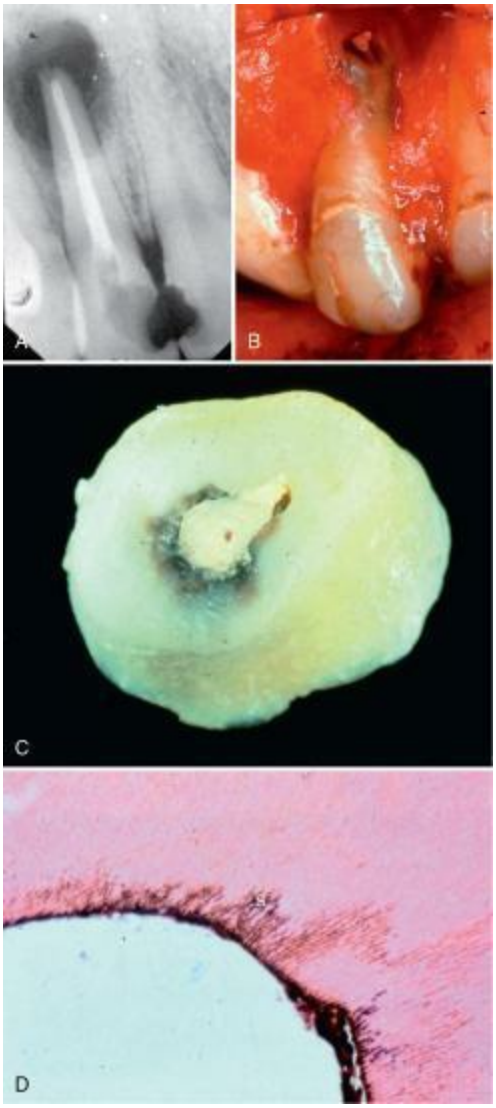

FIG. 17.7 **A,** Adequate root filling demonstrated on radiograph. **B,** View of the periapical surgery and root resection of the tooth shown in **(A)** shows stained root dentine. **C,** Resected root showing stained/infected dentine. **D,** Histologic view of the root end shown in **(C)** showing infected dentinal tubules *(S)*.

A) Radiograph shows two radiolucent patches, one at the root tip of tooth and the other at crown edge.

B) Close-up view shows stained tooth with a wound at the root tip and damaged red tissue at the gum line.

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C) Cut specimen of tooth shows nearly tears drop structure with a brownish black periphery.

D) Large cavity with dens vertical tubules in the edge.

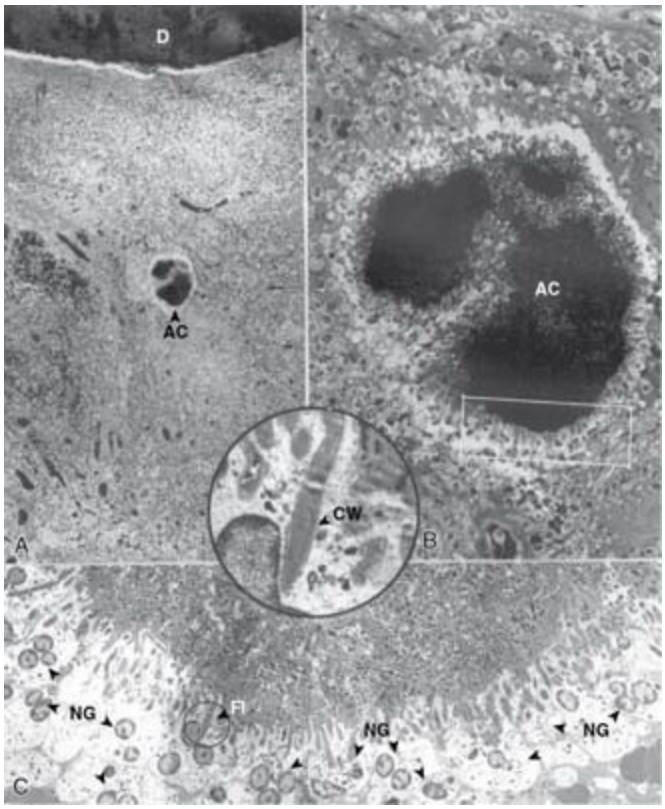

FIG. 17.8 Actinomyces in the body of a human periapical granuloma. The actinomycotic colony *(AC)* in (**A**) is magnified in (**B**). The rectangular area demarcated in **(B)** is magnified in **(C)**. Note the starburst appearance of the colony with needle-like peripheral filaments surrounded by few layers of neutrophilic granulocytes *(NG)*, some of which contain phagocytosed bacteria. A dividing peripheral filament *(FI)* is magnified in the inset. Note the typical gram-positive wall cell wall *(CW)*. *D,* Dentine. Original magnification: **A,** ×60; **B,** ×430; **C,** ×1680; inset, ×6700.

Three electron micrographs marked A through C.

A) An AC colony is shown as ovoid outline with two dark cavities inside the tooth.

B) The AC colony is enlarged showing large cavities with a small cavity.

C) The edge of AC cavity shows an arrow head, marked CW, pointing toward

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a vertical wide filament. Arrow heads pointing toward NG depicted as circular and oval patches.

Source: (From Nair PR, Schroeder H: Periapical actinomycosis, *J Endod* 10:567, 1984.)

The success of periapical surgery has been assessed with the same clinical and radiographic criteria as for nonsurgical root canal treatment. However, the radiographic criteria for successful periapical healing are different from those for nonsurgical root canal treatment (Table 17.3; Figs. 17.9–17.11).[140,186] Moreover, periodontal attachment loss in the form of marginal gingival recession is an additional criterion for measuring the outcome of periapical surgery.

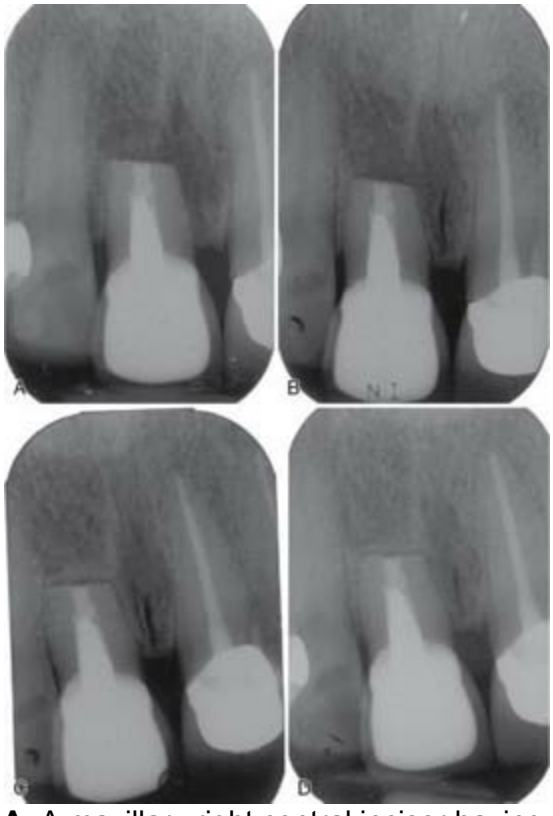

FIG. 17.9 **A,** A maxillary right central incisor having undergone periapical surgery. **B,** Incomplete periapical healing at 1 year postoperatively. Complete healing at 3 **(C)** and 4 years **(D)** postoperatively.

Four radiographs are marked A through D.

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A) It shows incisor with half and filled root canal.

B) It is similar to B, except that the tissue is less dense around the tooth.

C) It has porous tissue below the root.

D) It has slightly more dense tissue below the root.

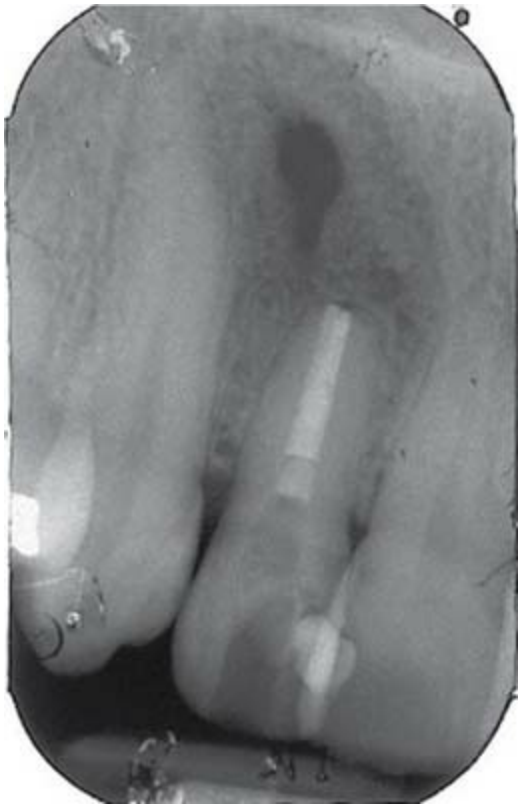

FIG. 17.10 Example of incomplete healing with isolated scar tissue.

Radiograph shows a knob-shaped dark patch slightly away from the root. The tooth has filled root canal, but the central part of crown and neck are radiolucent.

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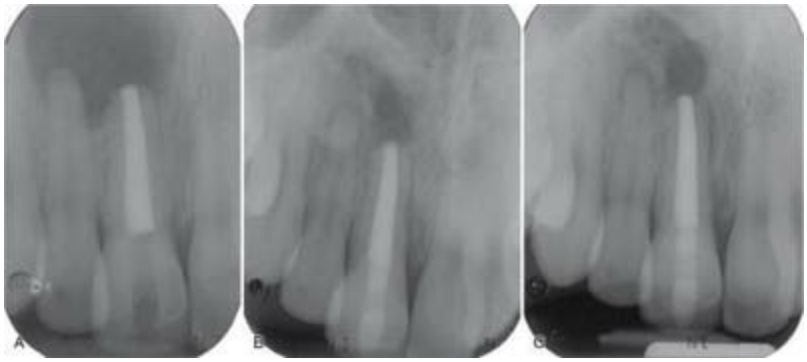

FIG. 17.11 Example of uncertain healing. Periapical radiographs of a maxillary left central incisor taken immediately after periapical surgery **(A)**, at 2 years postoperatively **(B)**, and at 3 years postoperatively **(C)**.

Three radiographs are marked A through C.

A) It has a large dark patch around the root apices of two teeth.

B) It has a triangular dark patch at the root tip of one tooth.

C) It has a circular dark patch at the root tip of one tooth.

Table 17.3

Classification of Periapical Healing Following Apical Surgery

COMPLETE HEALING
Reformation of a Periodontal Space With
Normal width and lamina dura to be followed around the apex Slight increase in width of apical periodontal space but less than twice the width of noninvolved parts of the root Tiny defect in the lamina dura (maximum 1 mm) adjacent to the root filling
Complete Bone Repair With
Bone bordering the apical area does not have the same density as surrounding noninvolved bone No apical periodontal space can be discerned
Incomplete Healing (Scar Tissue)
The Rarefaction Has Decreased in Size or Remained Stationary With
Bone structure recognized within the rarefaction The irregular periphery of the rarefaction and demarcation by a compact bone border The rarefaction located asymmetrically around the apex

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Angular connection between the rarefaction and the periodontal space
An Isolated Scar Tissue in the Bone With Findings Above
Uncertain Healing
The Rarefaction Has Decreased in Size With
The size larger than twice the width of the periodontal space Lamina-dura like bone structures around the border A circular or semicircular periphery Symmetric location around the apex as a funnel-shaped extension of the periodontal space Bone structure discernible within the bony cavity A collar-shaped increase in width of lamina dura coronal to the radiolucency
Unsatisfactory Healing (Failures)
The rarefaction has enlarged or is unchanged If a case still demonstrated "uncertain healing" 4 years postoperatively, the treatment should be considered a failure[186]

From Rud J, Andreasen JO: A study of failures after endodontic surgery by radiographic, histologic and stereomicroscopic methods, *Int J Oral Surg* 1:311-328, 1972; Molven O, Halse A, Grung B: Observer strategy and the radiographic classification of healing after endodontic surgery, *Int J Oral Maxillofac Surg* 16:432-439, 1987.

Now that the parameters have been discussed as to the variables that need to be considered when assessing healing or nonhealing outcomes, the following discussion will describe the expected outcomes of the various endodontic procedures.

Outcomes of vital pulp therapy procedures

The vital pulp therapy procedures discussed in this chapter include those for managing extensive caries with a high risk of pulpal exposure or pulps exposed through caries or traumatic injuries.

Indirect pulp capping (one-step versus stepwise excavation)

The most conservative management for extensive caries is indirect pulp capping using either the one-step or the stepwise excavation approach. The stepwise excavation approach with initial partial caries removal was advocated to reduce the risk of pulpal exposure and is generally considered to be associated with poorer outcomes. The clinical outcomes of the one-step or

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stepwise approaches have been compared in three randomized controlled trials on permanent teeth with deep caries.[19,113,121,122] Stepwise excavation was found to be associated with a lower rate of pulp exposure and higher chance of long-term clinical success than the one-step excavation approach (Table 17.4).[19,113] Maltz and colleagues,[121,122] however, found the contrary, and they attributed the low long-term clinical success rate of the stepwise approach to patients being noncompliant in returning for scheduled completion of treatment. Interestingly, comparison of the cost-effectiveness of the one-step versus the stepwise excavation techniques using health-economic modeling involving simulated treatment of a molar tooth with deep caries in a 15-year-old patient revealed that the one-step procedure accrues lower long-term costs as well as longer tooth retention and pulp vitality.[200]

Table 17.4

Studies Investigating the Clinical/Radiographic Outcome of Indirect Pulp Capping of Permanent Teeth

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Study	Study Design	Type of Indirect Pulp Capping	No. of Teeth	Success (%)	Age	Pretreatment Status	Capping Material	Criteria for Success	Duration After Treatment	Notes
More, 1967	Case series	Stepwise	8	8 (100%)	11–18	Carious Vital Apical pathosis	1st visit: ZOE 2nd visit: CH/Amal	Vital/asymptomatic/intact PDL	6–36 months	
Jordan et al., 1971 (in Hayashi et al., 2011) and 1978	Case series	1-step	243	236 (97.1%)	8–37	Deep caries without pulpitis	CH, CH + cresatin, or ZOE/ZOE + amalgam	Vital/asymptomatic/intact PDL	Not given	24 teeth were followed up for 11 years, 11 remained vital with no evidence of pathosis
Sawusch, 1982 (in Hayashi et al., 2011)	CCT	Stepwise	48	48 (100%)	14 or younger	Deep caries without pulpitis	CH (Dycal versus improved Dycal)/ZOE or ZnPO4	Clinical	6 months	
Fitzgerald and Heys, 1991	Case series	1-step	46	39 (84.8%)	20–60	Deep caries Vital	CH (Dycal versus Life)	Asymptomatic	1 year	The brand of CH did not have significant effect
Nagamine, 1993 (in Hayashi et al., 2011)	CCT	Stepwise	23	21 (91.3%)	17–46	Deep caries without pulpitis	Polycarboxylate cement with tannin-fluoride versus hydraulic temporary sealing material/GIC	Vital	3 months	
Leksell, 1996	RCT	Stepwise	57	47 (82.5%)	6–16	Not given	CH/ZOE then CH/GIC	Asymptomatic/intact PDL	1–11 years (mean = 43 months)	47/57 without exposure duration of CH dressing had no significant influence on exposure
		1-step	70	42 (60.0%)						42/70 without exposure

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Bjorndal and Thylstrup, 1998	Case series	Stepwise	94	87 (92.6%)	Not given	Deep caries	CH/temporary filling	Absence of perforation, asymptomatic	1 year	88/94 without exposur
Bjorndal et al., 2010	RCT	Stepwise	143	106 (74.1%)	29 (25–38)	Deep caries Only provoked pain Vital	CH/GIC	Vital/intact PDL	1 year	Stepwise better than complet preoper pain, older patients reduce success
		1-step	149	93 (62.4%)						
Gruythuysen et al., 2010	Case series	1-step	34	33 (97.1%)	Up to 18	Carious (> 2/3) No spontaneous, persistent pain No apical pathosis	GIC	Survive/asymptomatic/intact PDL/no resorption	3 years	
Maltz et al., 2011	Case series	1-step	26	16 (61.5%)		Vital Deep caries	CH/ZOE then composite	Vital	10 years	
Maltz et al., 2012 (a and b)	RCT (multicenter)	1-step	112	111 (18 months) 102 (91.7%) (3 years)	≥ 6	Vital Carious (> 1/2) No spontaneous, persistent pain No apical pathosis	GIC/amalgam or composite	Vital/intact PDL Vital/intact PDL	18 months, 3 years (clinical and radiographic outcome)	Indirect pulp capping was associat with significa higher success rate than stepwise regardle of duration after treatmer the low success rate of stepwise was attribute to patients not returnin for complet of treatmer

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		Stepwise	101	87 (18 months) 70 (69.3%) (3 years)			CH/ZOE then GIC/amalgam or composite			

CH, Calcium hydroxide; *GIC*, glass ionomer; *PDL*, periodontal ligament; *RCT*, randomized controlled trial; *ZOE*, zinc oxide eugenol.

Meta-analyses of data from studies[19,20,65,81,89,113,120-122, 141] listed in Table 17.4 revealed that the weighted pooled success rate for indirect pulp capping using the one-step approach (81.7%; 95% confidence interval [CI]: 72.7%, 90.6%) (Fig. 17.12) was similar to that for the stepwise approach (81.9%; 95% CI: 72.1%, 91.7%) (Fig. 17.13).

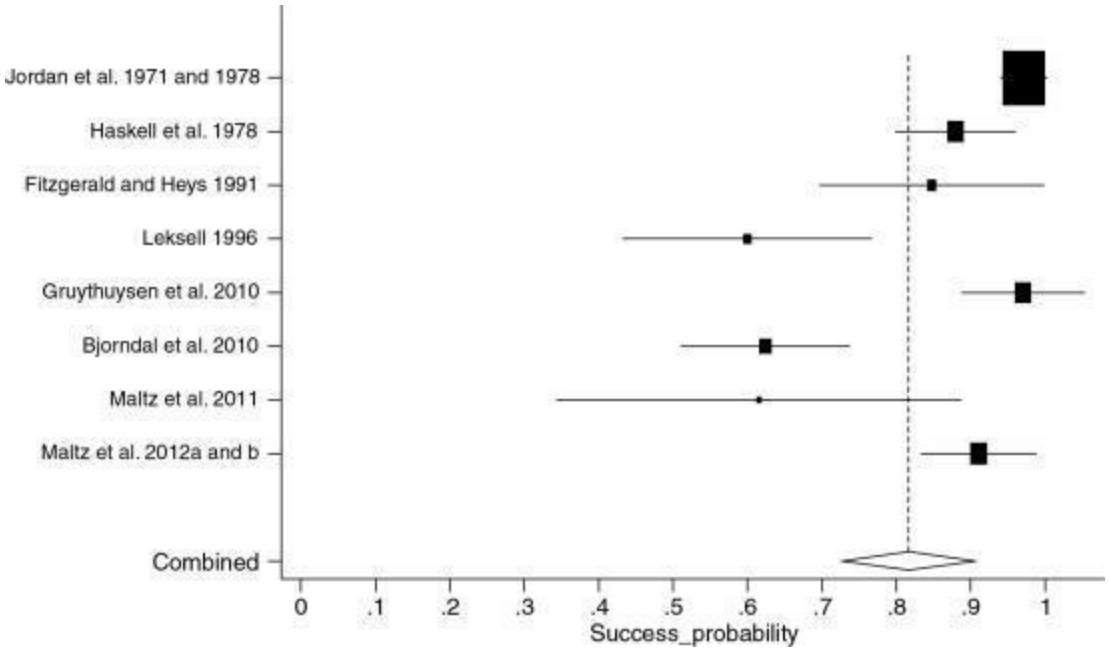

FIG. 17.12 Forest plot showing individual study and weighted pooled probabilities of success for one-step indirect pulp capping.

Forest plot shows individual study against success probability.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.7 5 to 0.9, Maltz et al. 2012a and b: 0.9, Maltz et al. 2011: 0.6 1, Bjorndal et al. 2010: 0.6 2, Gruythuysen et al. 2010: 0.9 6, Leksell 1996: 0.6, Fitzgerald and Heys 1991: 0.8 6, Haskell et al. 1978: 0.8 8, and Jordan et al. 1971 and 1978: 1.

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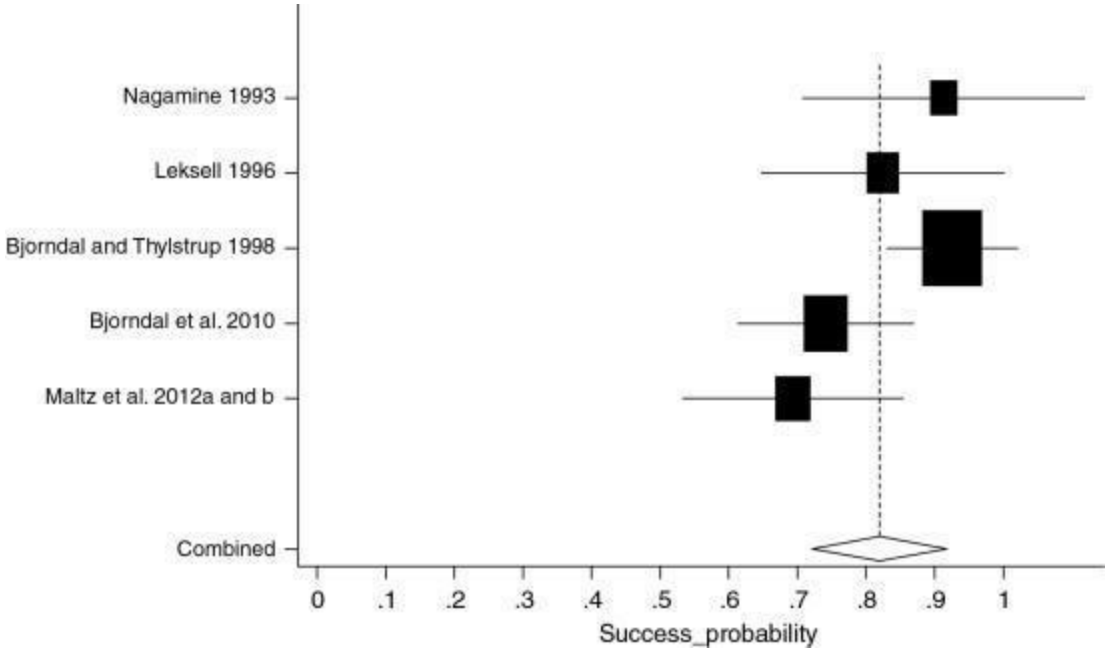

FIG. 17.13 Forest plot showing individual study and weighted pooled probabilities of success for stepwise indirect pulp capping.

Forest plot shows individual study against success probability.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.7 5 to 0.9, Maltz et al. 2012a and b: 0.7, Bjorndal et al. 2010: 0.7 3, Bjorndal and Thylstrup 1998: 0.9 2, Leksell 1996: 0.8 2, and Nagamine 1993: 0.9 2.

Calcium hydroxide cement was the preferred lining material for the pulpal surface, whereas zinc oxide–eugenol–based cement was the preferred base material in most studies. Recently, resin-modified-glass-ionomer liner has also been used, but the type of lining material did not influence the outcome (see Table 17.4). The age of patients, the presence of preoperative pain, and pulpal exposure during excavation were significant negative prognostic factors.[19]

Direct pulp capping

Direct pulp capping is performed on teeth with pulp exposures caused by caries, caries excavation, or traumatic injuries. Most studies investigating the clinical outcome of direct pulp capping on permanent teeth had excluded teeth with signs and symptoms of irreversible pulpitis and apical pathosis (Table 17.5). Saline, sodium hypochlorite, and chlorhexidine have been

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reportedly used to irrigate the exposed pulp and to achieve hemostasis. Calcium hydroxide paste and mineral trioxide aggregate (MTA) were the commonly used capping materials (see Table 17.5).

Table 17.5

Studies Investigating the Clinical/Radiographic Outcome of Direct Pulp Capping of Permanent Teeth

Study	Study Design	No. of Teeth	Success (%)	Age (Years)	Pretreatment Status	Hemostasis	Capping Material	Base/Restorative Material	Criteria for Success	Duration After Treatment	Notes
Weiss, 1966	Case series	160	141 (88.0%)	16–67	Not given	Not given	CH + cresatin	ZOE	Vital/asymptomatic/intact PDL	3 years	
Shovelton et al., 1971	RCT	154 (1-step)	115 (74.7%)	15–44	Carious or traumatic exposure Vital Asymptomatic Molar/premolar	Saline	Corticosteroid + antibiotic, glycerrhetinic acid + antibiotics, ZOE, or CH	ZOE/amalgam	Vital/asymptomatic/intact PDL	24 months	There was no significant difference in success rates among the various capping materials
		53 (2-step)	33 (64.7%)								
Haskell et al., 1978	Case series	133	117 (88.0%)	8–74	Carious exposure Asymptomatic	Not given	CH or penicillin	ZOE	Vital/asymptomatic/intact PDL	5 years	7- to 22-year survival Success rate was not affected by age and tooth type
Gillien and Schuman, 1985	Case series	17	13 (76.4%)	6–9	Carious exposure	Not given	CH	Base material not given/amalgam or crown	Asymptomatic/intact PDL	6 1/2 months	

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Horsted et al., 1985	Case series	510	485 (95.1%)	Not given	Exposure during cavity preparation or caries removal No periapical pathosis No pain	2% chloramine or 0.2% CHX	CH	ZOE	Vital/asymptomatic/intact PDL	1 years	Type of exposu and tooth type had no signific effects Older age had low surv
Fitzgerald and Heys, 1991	Case series	8	6 (75.0%)	20–60	Vital Exposure during caries removal	Sterile cotton pellets	CH	$ZnPO_4$/amalgam or composite	Asymptomatic	12 months	The brand of setting CH paste did not have signific effect
Matsuo et al., 1996	Case series	44	36 (81.2%)	20–69	Carious exposure No intense pain	10% NaOCl and 3% H_2O_2	CH	ZOE/GIC	Vital/asymptomatic/intact PDL	3 years	
Santucci, 1999	Case series	29	15 (51.7%)	Not given	Exposure due to caries, or its removal Sensitive to cold or sweat with no other pain No periapical pathosis	Not given	CH	Composite or cast gold restoration	Asymptomatic	1–5 years	

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Barthel et al., 2000	Case series	123	29 (23.6%)	10–70	Carious exposure	3% H_2O_2	$CH/ZnPO_4$ or other materials	Not given	Vital/asymptomatic/intact PDL	7–10 years	Age, tooth type, site of exposu had no signific effects Immed plac of perr rest had sign high succ rate
Farsi et al., 2006	Case series	30	28 (93.3%)	9–12	Deep caries Potential exposure Reversible pulpitis Intact PDL	Saline	MTA	ZOE/compos	Vital/asymptomatic/intact PDL/continuous root development	2 years	
Bogen et al., 2008	Case series	49	48 (98.0%)	7–45	Carious exposure (0.25–2.5 mm) Reversible pulpitis	5.25/6% NaOCl	MTA	Composite	Dentine bridge/asymptomatic/roo development/intact PDL	1–9 years	
Bjorndal et al., 2010	RCT	22	7 (31.8%)	25–38	Exposure during caries removal Only provoked pain Vital	Saline	CH	GIC	Vital/intact PDL	1 year	

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Mente et al., 2010	Case series	122	86 (70.5%)	8–78	Carious or mechanical exposure	0.12% CHX	MTA or CH	GIC/composite or crown	Vital/no clinical or radiographic evidence of apical pathosis	12–18 months	Use of MTA for cappin and immed placem of perma restora had signific higher success rate Age; sex; toot loca and type exp site and type rest type size and qua did not hav sign effe
Miles et al., 2010	Case series	51	23 (45.1%)	21–85	Carious exposure	2.5% NaOCl	MTA	GIC/composite or amalgam	Vital/asymptomatic/intact PDL	12–27 months	
Hilton et al., 2013	RCT	126	81 (64.3%)	9–90	Carious, traumatic, mechanical exposure	5.25% NaOCl	CH	GIC	Vital/intact PDL/no resorption/not requiring extraction or root canal treatment	2 years	MTA was associa with signific higher success rate than CH Patient den toot pul exp and pul cap cha did not hav sign infl on the resu

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		144	116 (80.6%)	8–89			MTA				

CH, Calcium hydroxide; *GIC*, glass ionomer; *MTA*, mineral trioxide aggregate; *NaOCl*, sodium hypochlorite; *RCT*, randomized controlled trial.

A meta-analysis of data from studies[12,19,21,64,65,72,88,90,92,128,132,133,193,207,254] listed in Table 17.5 revealed the weighted pooled success rate to be 70.1% (95% CI: 59.9%, 80.2%) (Fig. 17.14). The patient's age and sex; tooth location and type; pulp exposure type, size, and its location; and the restoration type, size, and quality did not have a significant influence on success. Although the outcome of direct pulp capping of teeth with immature versus mature apices has not been systematically compared in individual studies, indirect comparison of pooled data from different studies[1] revealed that teeth with immature roots were associated with significantly more successful outcomes.

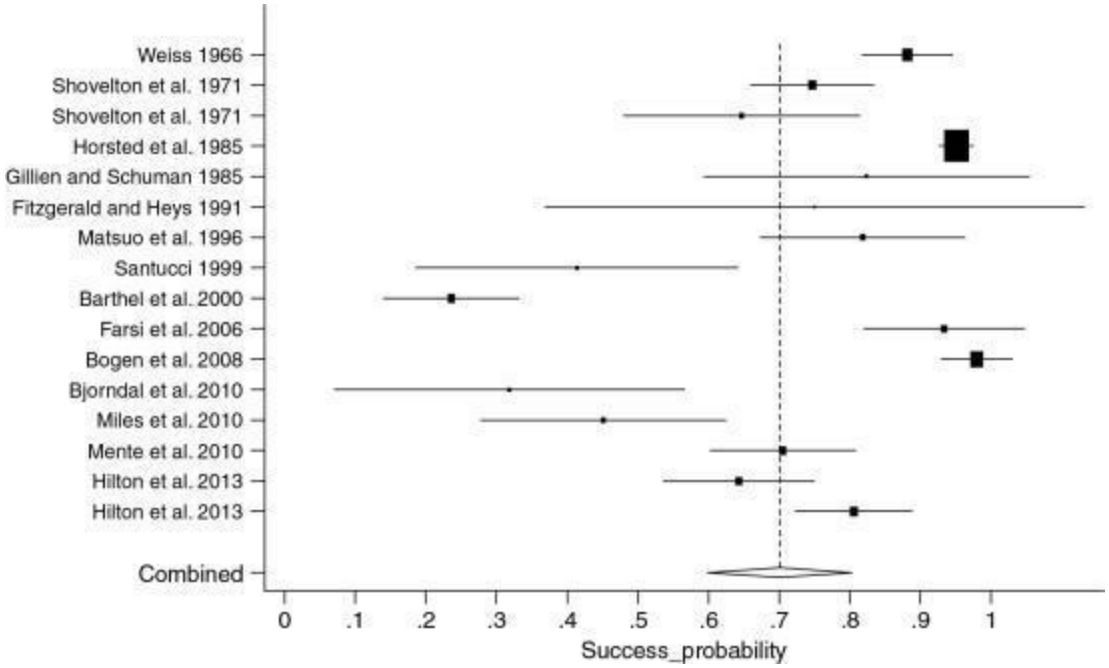

FIG. 17.14 Forest plot showing individual study and weighted pooled probabilities of success for direct pulp capping.

Forest plot shows individual study against success probability.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.6 to 0.8, Hilton et al. 2013: 0.8 1, Hilton et al. 2013: 0.6 4, Mente et al. 2010: 0.7, Miles et al. 2010: 0.3 1, Bjorndal et al.

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2010: 0.3 2, Bogen et al. 2008; 1, Farsi et al. 2006: 0.9 2, Barthel et al. 2000: 0.2 4, Santucci 1999: 0.4, Matsuo et al. 1996: 0.8, Fitzgerald and Heys 1991: 0.7 4, Gillien and Schuman 1985: 0.8 1, Shovelton et al. 1971: 0.6 3, Shovelton et al. 1971: 0.7 6, and Weiss 1966: 0.9 2.

The type of capping material was another significant prognostic factor,[90,197,207] with MTA performing superiorly to calcium hydroxide in a randomized controlled trial[90] and in a systematic review.[1]

Pulpotomy

Earlier studies on the outcome of *partial pulpotomy* (specifying varying degrees of coronal pulp removal) had only included teeth with vital traumatic pulp exposures; however, more recent studies have also included teeth with carious pulp exposures. Teeth with spontaneous or severe pain and signs and symptoms of apical pathosis were excluded (Table 17.6). On the other hand, the outcomes of *full pulpotomies* (completely removing the coronal pulp and retaining the radicular pulp) had only been investigated in teeth with carious exposures (Table 17.7). Saline irrigation has been preferred for achieving hemostasis, whereas calcium hydroxide, and more recently, MTA, were the preferred pulp capping materials (see Table 17.6).

Table 17.6

Studies Investigating the Clinical/Radiographic Outcome of Partial Pulpotomy of Permanent Teeth

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Author	No. of Teeth	Success (%)	Age	Pretreatment Status	Exposure Size	Hemostasis	Pulp Dressing Material	Base/Restorative Material	Criteria for Success	Duration After Treatment	Notes
Cvek, 1978	60	58 (96.7%)	Not given	Traumatic exposure Vital Bleeding wound	0.5–4.0 mm	Saline	1st visit: nonsetting CH 2nd visit: setting CH	1st visit: ZOE 2nd visit: Composite	EPT/asymptomatic/intact PDL/continued root development/hard tissue barrier	1–60 months	Size and duration of exposure root maturity did not affect outcome
Baratieri et al., 1989	26	26 (100%)	12–44	Exposure due to caries or caries removal Pulp tissue bleeding without signs of degeneration	Not given	Calcium hydroxide solution	CH powder then hard set CH cement	Zinc oxide cement	Asymptomatic/vital	1–2 years	
Fuks et al., 1993	44	35 (79.5%)	Not given	Traumatic exposure Vital Bleeding wound	Not given	Saline	CH	ZOE	Asymptomatic/bridge/root development/vital	0.5–4 years	
Mass and Zilberman, 1993	35	32 (91.4%)	7.5–25	Molar Deep caries Asymptomatic No apical pathosis	Less than 1–2 mm diameter; 2–3 mm deep	Saline	CH	ZOE/amalgam or crown	Asymptomatic/intact PDL/root development	1–2 years	
Mejare and Cvek, 1993	31 (2-step)6 (1-step)	29 (93.5%) 4 (66.7%)	Not given	Carious exposure Asymptomatic No apical pathosis	N/A	Saline	CH	ZOE	Asymptomatic/intact PDL/root development	24–140 months	
Barrieshi-Nusair and Qudeimat, 2006	28	21 (75.0%)	7.2–13.1	Carious exposure Molar Reversible pulpitis No apical pathosis	2–4 mm deep	Saline	MTA	GIC/amalgam or crown	Vital/asymptomatic/intact PCL/root development	1–2 years	

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Qudeimat et al., 2007*	23	21 (91.3%)	6.8–13.3	Carious exposure Molar	2–4 mm deep		CH	GIC/amalgam or crown	Absence of signs and symptoms/intact PDL/root development	24.5–45.6 months	There was no signific differer in outcom betwee CH and MTA
	28	26 (92.9%)					MTA				
Bjorndal et al., 2010*	29	10 (34.5%)	25–38	Deep caries Only provoked pain Vital Expose during removal	Not given	Saline	CH	GIC	Vital/intact PDL	1 year	

CH, Calcium hydroxide; *EPT*, electric pulp test; *GIC*, glass ionomer; *MTA*, mineral trioxide aggregate *PDL*, periodontal ligament; *ZOE*, zinc oxide eugenol.
NB: All the studies were case series except those labeled with *.
Qudeimat et al. (2007) is a randomized controlled trial comparing calcium hydroxide versus MTA as pulp dressing material.
Bjørndal et al. (2010) is a randomized controlled trial comparing the outcomes of 1-step versus stepwise indirect pulp capping for teeth without exposure, and direct pulp capping versus partial pulpotomy for teeth with exposure during caries removal.

Table 17.7

Studies Investigating the Clinical/Radiographic Outcome of Full Pulpotomy of Permanent Teeth

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Author	No. of Teeth	Success (%)	Age	Pretreatment Status	Hemostasis	Capping Material	Base/Restorative Material	Criteria for Success	Duration After Treatment	Notes
Masterson, 1966	30	25 (83.3%)	6–39	Not given	Not given	CH	Not given	Vital/asymptomatic	1–78 months	
Russo et al., 1982	30	28 (93.3%)	9–28	Carious exposure No apical pathosis	Not given	CH	Not given	Intact PDL	8 weeks	
Santini, 1983	373	192 (51.4%)	Not given	Carious exposure or near exposure Symptomatic	Cotton wool pellet	CH or CH + Ledermix	ZOE	Vital/bridge/asymptomatic	6 months–5	Sex and medicar had no significa effect Poor heali was assoc with age < 7.5
Caliskan, 1993	24	22 (91.7%)	10–22	Hyperplastic pulpitis	Saline	CH	ZOE/Amalgam or composite	Vital/asymptomatic/intact bridge/intact PDL	1–4 years/dentine	
Caliskan, 1995	26	24 (92.3%)	10–24	Carious exposure Asymptomatic Apical pathosis	Saline	CH	ZOE	Asymptomatic/bridge/root development/vital	16–72 months	
Waly, 1995	20	18 (90.0%)		Carious exposure Molar		CH-glutaraldehyde CH	Not given	Not given	5 years	
Teizeira et al., 2001	41	34 (82.9%)	6–16	Deep caries or exposed pulp With or without apical pathosis	Not given	CH	GIC	Vital/asymptomatic/ bridge/intact PDL	24–32 weeks/dentine	
DeRosa, 2006	26	17 (65.4%)		Not given		CH	Amalgam	Asymptomatic	14–88 months	

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El Meligy and Avery, 2006	15	13 (86.7%)	6–12	Carious or traumatized teeth Immature apex No apical pathosis	Saline	CH	ZOE/Amalgam or composite	Asymptomatic/intact PDL/no resorption/root development	1 year	
	15	15 (100%)				MTA				
Witherspoon et al., 2006	19	15 (79.0%)	7–16	Carious or traumatic exposure Irreversible pulpitis	6% NaOCl	MTA	Not given	Vital/asymptomatic/intact PDL/root development	3 years	
Asgary and Ehsani, 2009	12	12 (100%)	14–62	Carious Irreversible pulpitis	Saline	NEC	Permanent rest	Asymptomatic/intact PDL	13–26 months	

CH, Calcium hydroxide; *GIC*, glass ionomer; *MTA*, mineral trioxide aggregate, *NaOCl*, sodium hypochlorite; *NEC*, new endodontic cement.
NB: All the studies were case series except El Meligy and Avery (2006), which was a randomized controlled trial comparing calcium hydroxide and MTA as pulp dressing material.

A meta-analysis of data from studies listed in Tables 17.6[10,11,19,40,71,125,131,177] and 17.7[6,30,31,52,58,126,187,192,235,251,255] revealed the weighted pooled success rate to be 79.3% (95% CI: 66.7%, 91.8%) for partial pulpotomies (Fig. 17.15) and 82.4% (95% CI: 69.3%, 95.4%) for full pulpotomies (Fig. 17.16).

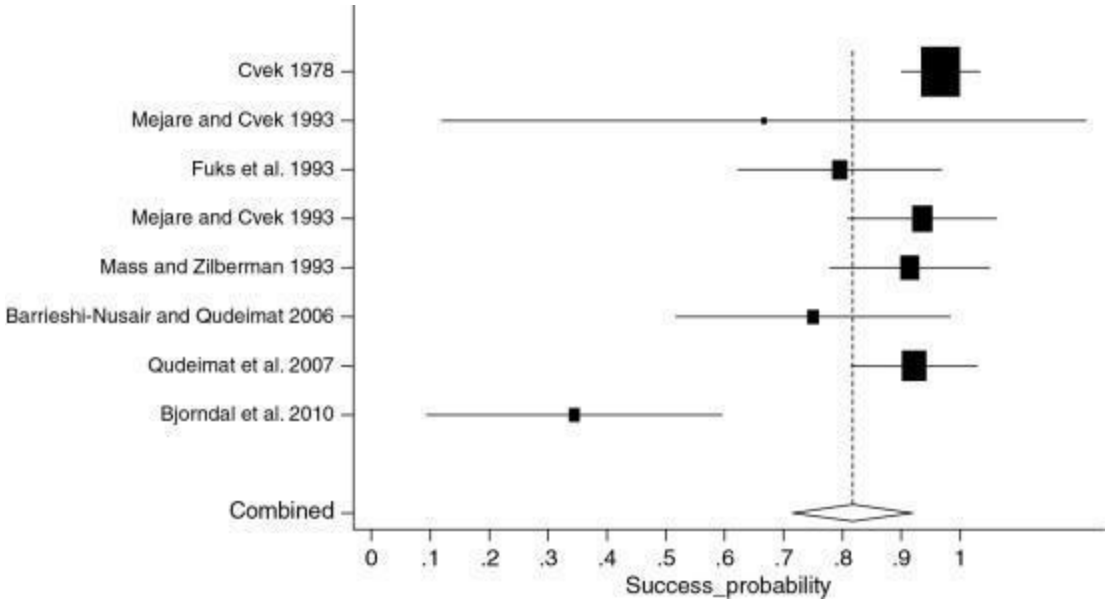

FIG. 17.15 Forest plot showing individual study and weighted pooled probabilities of success for partial pulpotomy.

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Forest plot shows individual study against success probability.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.7 to 9.0, Bjorndal et al. 2010: 0.3 6, Qudeimat et al. 2007: 0.9 4, Barrieshi-Nusair and Qudeimat 2006: 0.7 5, Mass and Zilberman 1993: 0.9 1, Mejare and Cvek 1993: 0.9 3, Fuks et al. 1993: 0.7 9, Mejare and Cvek 1993: 0.7, and Cvek 1978: 0.9 9.

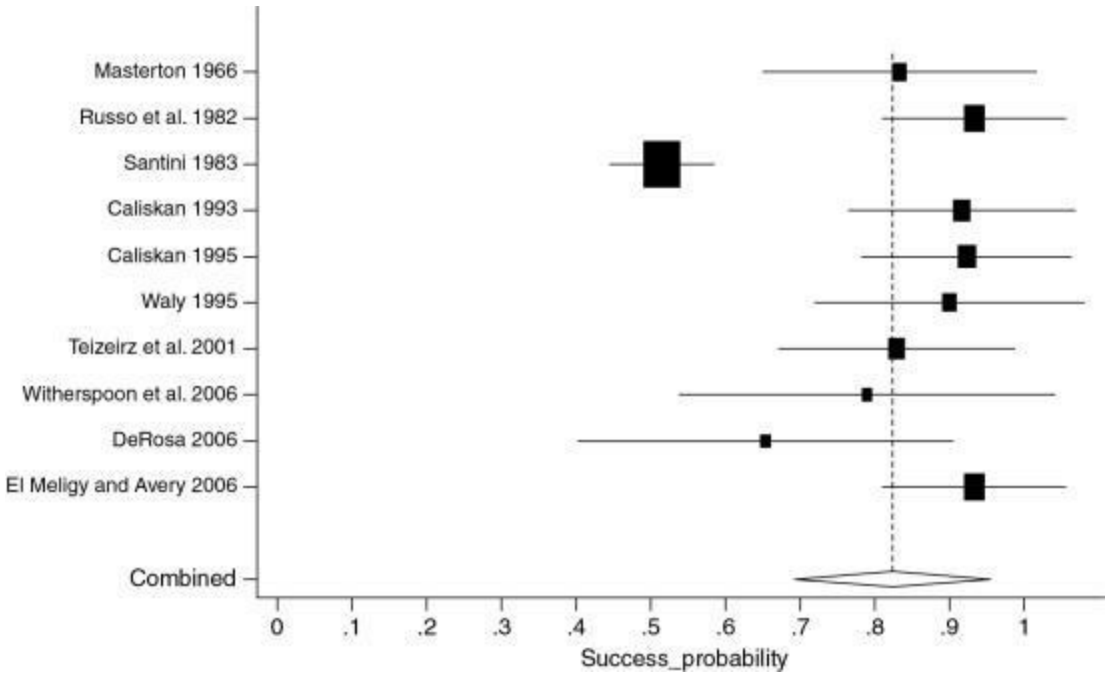

FIG. 17.16 Forest plot showing individual study and weighted pooled probabilities of success for full pulpotomy.

Forest plot shows individual study against success probability.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.7 to 0.9, El Meligy and Avery 2006: 0.9 3, DeRosa 2006: 0.6 6, Witherspoon et al. 2006: 0. 8, Teizeirz et al. 2001: 0.8 3, Waly 1995: 0.9, Caliskan 1995: 0.9 2, Caliskan 1993: 0.9 2, Santini 1983: 0.5 1, Russo et al. 1982: 0.9 2, and Masterton 1966: 0.8 3.

The effects of potential prognostic factors for pulpotomy have not been explored systematically except for the pulp capping material. Randomized controlled trials revealed that MTA achieved similar outcomes in partial[177] or full pulpotomies[58] when compared with calcium hydroxide.

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Summary of prognostic factors for vital pulp therapy

In summary, vital pulp therapy performed under guideline standards with optimal coronal seal achieved promising long-term success in teeth with carious, mechanical, or traumatic exposures of healthy pulps.

The most important factors affecting the outcome of vital pulp therapy are preexisting health of the pulp, adequate removal of infected hard or soft tissues, careful operative technique to avoid damage to residual tissues, and elimination of microbial leakage around the final restoration. It can be difficult to gauge the health of the residual pulp as it is a matter of subjective assessment and relies on experience in pulp diagnosis. The degree of pulp bleeding upon exposure is a more reliable tool to judge the status of the pulp than the preoperative clinical signs and symptoms. Continued bleeding after 10 minutes, even after rinsing with sodium hypochlorite solution, may suggest that the residual pulp was still heavily inflamed and a complete pulpectomy may be a more effective treatment modality. Removal of infected tissue is a matter of subjective experience but may be aided by various dyes. The final factor relies on the correct choice of restorative material and its adequate manipulation to prevent leakage.

Factors such as the age and health of the patient, the size and nature (carious or traumatic) of pulp exposure and its duration of exposure to the oral environment (up to 48 hours) do not in themselves compromise outcomes of vital pulp therapy.

Outcomes of nonsurgical root canal treatment

In contrast to other areas of endodontics, the number of studies and extent of investigation of nonsurgical root canal treatment is more comprehensive, yielding a much greater insight even though the quality and scope of the research does not always reach the highest levels.

A systematic review and meta-analysis of the factors affecting primary root canal treatment outcome conducted by the authors revealed the following: the mean success rate was 83% when vital pulpectomy was performed (Fig. 17.17), which reduced to 72% when the root canal treatment procedure was used to eradicate the established infection associated with a periapical lesion (Fig. 17.18).

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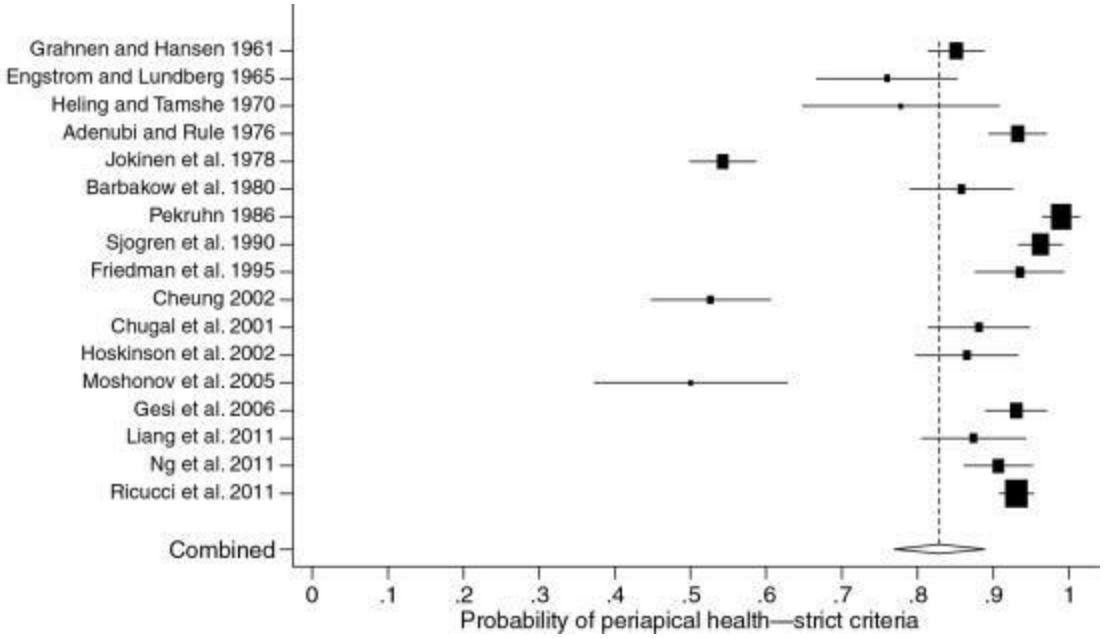

FIG. 17.17 Forest plot showing results of pooled and individual study's probability of maintained periapical health for preoperatively vital teeth undergoing root canal treatment (pooled probability = 0.83; 95% confidence interval: 0.77, 0.89).

Forest plot shows individual study against VITALSS_pr.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.7 8 to 0.8 9, Ricucci et al. 2011: 0.9 3, Ng et al. 2011: 0.9, Liang et al. 2011: 0.8 7, Gesi et al. 2006: 0.9 3, Moshonov et al. 2005: 0.5, Hoskinson et al. 2002: 0.8 7, Chugal et al. 2001: 0.8 8, Cheung 2002: 0.5 2, Friedman et al. 1995: 0.9 3, Sjogren et al. 1990: 0.9 5, Pekruhn 1986: 0.9 9, Barbakow et al. 1980: 0.8 7, Jokinen et al. 1978: 0.5 3, Adenubi and Rule 1976: 0.9 2, Heling and Tamshe 1970: 0.7 7, Engstrom and Lundberg 1965: 0.7 6, and Grahnen and Hansen 1961: 0.8 5.

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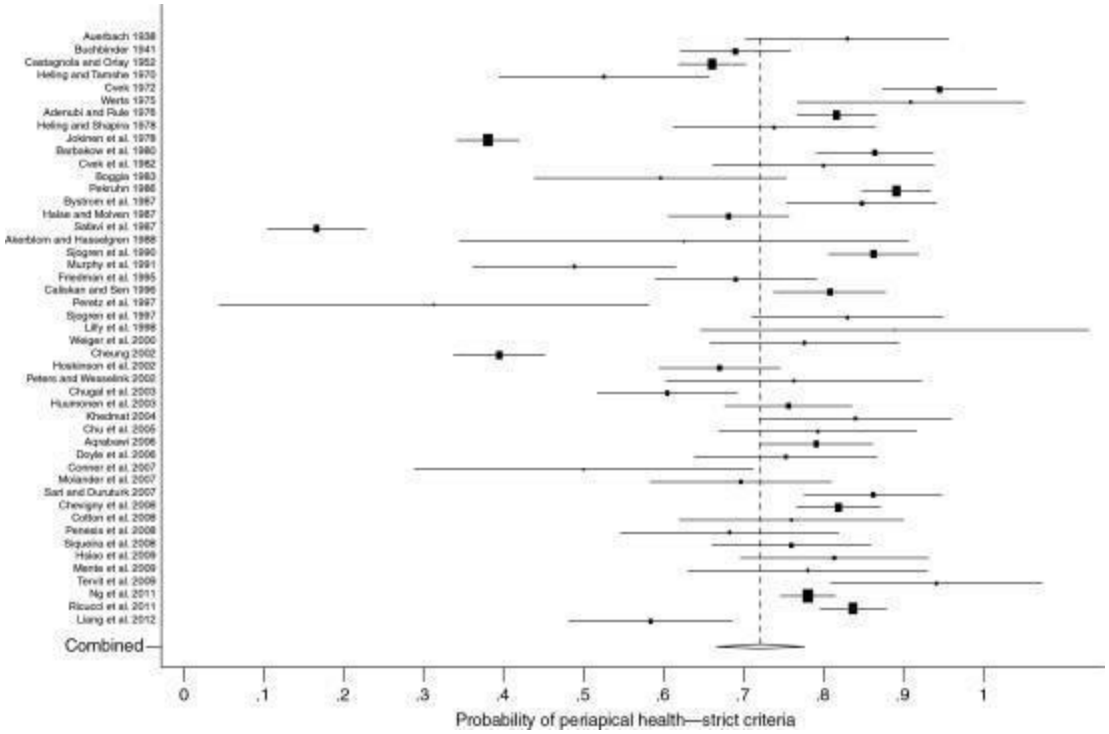

FIG. 17.18 Forest plot showing results of pooled and individual study's probability of periapical health for teeth associated with nonvital pulps and periapical radiolucencies undergoing root canal treatment (pooled probability = 0.72; 95% confidence interval: 0.67, 0.78).

Forest plot shows individual study against NVPASS_pr.

The horizontal axis is marked 0 to 1 with increment of 0.1. The approximate data are as follows: Combined: 0.6 8 to 0.7 8, Liang et al. 2012: 0.5 8, Ricucci et al. 2011: 0.8 3, Ng et al. 2011: 0.7 8, Tervit et al. 2009: 0.9 3, Mente et al. 2009: 0.8 8, Hsiao et al. 2009: 0.8 3, Siqueira et al. 2008: 0.7 6, Penesis et al. 2008: 0.6 9, Cotton et al. 2008: 0.7 8, Chevigny et al. 2008: 0.8 2, Sari and Duruturk 2007: 0.8 7, Molander et al. 2007: 0.7, Conner et al. 2007: 0.5, Doyle et al. 2006: 0.7 6, Aqrabawi 2006: 0.7 9, Chu et al. 2005: 0.8, Khedmat 2004: 0.8, Huumonen et al. 2003: 0.8 5, Chugal et al. 2003: 0.7 6, Peters and Wesselink 2002: 0.6, Hoskinson et al. 2002: 0.7 8, Cheung 2002: 0. 6 6, Weiger et al. 2000: 0.7 9, Lilly et al. 1998: 0.8 9, Sjogren et al. 1990: 0.8 1, Peretz et al. 1997: 0.3 6, Calisken and Sen 1996: 0.8, Friedman et al. 1995: 0.7, Murphy et al. 1991: 0.5, Sjogren et al. 1990: 0.5, Akerblom and Hasselgren 1988: 0.8 7, Safavi et al. 1987: 0.1 8, Halse and Molven 1987: 0.7 6, Bystrom et al. 1987: 0.6 8, Pekruhn 1986: 0.7 8, Boggia 1983: 0.8 6, Cvek et al. 1982: 0.6, Cvek et al. 1982: 0.8, Barbakow et al. 1980: 0.8 9, Jokinen et al. 1978: 0.4, Heling and Shapira 1978: 0.7 5, Adenubi and Rule 1976: 0.8 3, Werts 1975: 0.8 8, Cvek 1972: 0.9 5, Heling and Tamshe 1970: 0.5, Castagnola and Orlay 1952: 0.6, Buchbinder 1941: 0.6 7, and Auerbach 1938: 0.8 4.

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Factors affecting periapical health or healing following root canal treatment

The factors influencing the maintenance of periapical health or periapical healing of preexisting lesions following root canal treatment may be broadly classified into patient factors (age, sex, general health, tooth anatomy, preoperative pulpal and periapical status), treatment factors (operator variables, canal enlargement, irrigation, medication, culture test, and obturation), and restorative factors. Some factors had a profound impact on success rates, whereas others showed a negligible effect. Patient factors characterizing the nature of the disease showed the most significant effect (periapical status), whereas most of the treatment factors were found to exert a less significant effect; exceptions were the apical extent of root canal treatment relative to the root canal terminus. In addition, the quality of the postoperative restorative care also exerted a profound influence on the outcome of treatment.

Patient factors

Patient's age and sex consistently had no significant effect on outcome, whereas some specific health conditions (diabetes,[54,67] compromised immune response[123]) apparently had a significant influence. The evidence for the effect of host immune response characterized by the general health of the patient is, however, weak. Emerging evidence indicates that the host response measured by polymorphisms of various genes involved in periapical healing may have an effect on outcomes (Table 17.8).[142,183,212,213] The importance of the host response to maintenance of periapical health or periapical healing was also supported by the statistically significant clustering effect of multiple teeth within the same patient in a prospective study.[150]

Table 17.8

Type of Genes Investigated in Studies on SNPs and Periapical Healing

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Study	Gene Function	Genes	Findings
Siqueira et al., 2009	Interleukin 1 Fcγ receptor	IL-1α, IL-1β FcγRIIa, FcγRIIIb	No significant association Significant association
Morsani et al., 2011	Interleukin	IL-1β	Significant association
Siqueira et al., 2011	Fcγ receptor	FcγRIIIa	No significant association
Rôças et al., 2014	Pattern recognition receptors	CD14, TLR4	No significant association

SNP, Single nucleotide polymorphism.

The widespread perception that single-rooted teeth with less complicated anatomy should benefit with more predictable and favorable outcomes proves to be untrue. Having accounted for potential confounding factors such as the presence of periapical disease, tooth type does not seem to exert a strong influence on success rates. This would appear to be counterintuitive but may be explained by the logical inference that canal complexities in the apical anatomy probably play a more dominant role than other complexities, such as the number and curvature of canals.

The presence and size of a periapical lesion seem to have the most negative effect on periapical health/healing; it therefore follows that these factors must be accounted for when analyzing the influence of any other factor. The profound change in success rates once a periapical lesion becomes established is interesting, as it is correlated with the establishment of infection in the apical canal anatomy. This seems to suggest that once the apical canal complexities become infected, it may be much more difficult to eradicate the infection. The negative influence of large periapical lesions has a reasonable biologic explanation: the diversity of bacteria (by number of species and their relative abundance) is greater in teeth with larger periapical lesions.[230] An endodontic infection is more likely to persist in canals with a higher number of bacteria present preoperatively.[27] In addition, larger lesions may represent longer-standing root canal infections that may have penetrated deeper into dentinal tubules and accessory anatomy in the complex canal system[206] where mechanical and chemical decontamination procedures may not be so effective. Larger lesions may also represent cystic transformation.[145] Finally, the host response may also play a part, as patients with larger lesions may innately respond less favorably to residual bacteria.[147] This speculation may crystallize into distinct questions for further biologic research into the nature of interactions among host, bacterial infection, and treatment intervention.

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Most of the other investigated preoperative factors (pain, tooth tenderness to percussion, soft-tissue tenderness to palpation, soft-tissue swelling and sinus tract, periodontal probing defect of endodontic origin, root resorption) are in fact different clinical manifestations of periapical disease.[254] They may therefore act as surrogate measures or complement "presence and size of periapical lesion" in measuring the effect of severity of periapical disease within a broad continuous spectrum. Of these, only presence of preoperative pain,[68] sinus tract,[150] swelling,[150] and apical resorption[229] have been found to be significant prognostic factors that have been associated with significantly reduced success rates in root canal treatment.

The biologic explanation for the negative impact of sinus tract and swelling, either in the acute or chronic form, on periapical healing is interesting, as both represent suppuration and the proliferation of microbiota into the periapical tissues, with the inference being that the host tissues must have become locally overwhelmed. The precise reasons for reduced success rates under these conditions remain unclear but must somehow be related to the nature of the host-microbial interaction.

Treatment factors

Operator.

Although the impact of operator qualification and skill has not been specifically investigated, systematic review has shown that the involved clinicians may be grouped into undergraduate students, general practitioners, postgraduate students, and specialists. Studies show a clear trend in superior outcomes by greater experience and training. Clearly, technical skills play an important role, but this is difficult and often impossible to quantify. In addition, the technical abilities must be augmented by the overall understanding of the biologic issues and the quest for superior treatment by the operator. If clinicians do not feel that they can provide optimal treatment, it is incumbent upon them to refer the patient to a more qualified practitioner.

Isolation.

The use of rubber dams in modern root canal treatment is widely accepted, and the justification seems almost empirical. One study on retreatment[244]

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analyzed the influence of rubber dam use compared to cotton roll isolation and found significantly higher success rates with the former approach. Another study reported a significantly higher success rate of root canal–treated teeth[76] when a rubber dam was employed during post placement compared to when it was not. Perhaps as a consequence, the principal justification for rubber dam use is based on medicolegal implications of root canal instrument ingestion or aspiration by the patient.[61]

Magnification and illumination.

Endodontists have repeatedly reinforced the value of magnification and illumination during root canal treatment,[166] but a systematic review failed to draw any objective conclusions on their influence as no article was identified in the current literature that satisfied their inclusion criteria.[46] A prospective study investigated this factor,[150] but researchers found only an insignificant influence on the final outcome. Use of a microscope may sometimes assist location of the second mesiobuccal canal in maxillary molars, but this only made a small difference to the success rates associated with mesiobuccal roots, when a periapical lesion was present.[150] The true benefit of a microscope can only be verified through a randomized controlled trial. However, canal negotiation with less tooth structure removal and fewer procedural accidents is favorable and seems intuitively more consistent with the use of superior magnification and illumination.

Mechanical preparation: Size, taper, extent, and procedural errors.

The root canal system may be mechanically prepared to a requisite size and taper[199] using a variety of instruments of different cutting designs, tips, tapers, and materials of construction. Their efficacy is often tested in laboratory studies, and the instruments and their utility may have well-characterized properties.[95] Investigation of the influence of the type of instrument used for canal enlargement has been undertaken in one nonrandomized prospective study, but the outcome is likely subjective because of many factors, including the protocol adopted for teaching technical skills.[150] In this study, the better success rates for hand or rotary NiTi instruments compared with stainless steel instruments[150] were

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attributable to the fact that tactile skills training was achieved through a preliminary focus on the use of stainless steel files to develop tactile sensitivity and consistency. Only on demonstration of this competency did the trainees progress to NiTi instruments. More importantly, such senior students may also have had a better understanding of the biologic rationale for root canal treatment. The ability to gain and maintain apical patency as well as to avoid procedural errors was better instilled in the senior students, whereas in selected cases, NiTi instruments appear capable of achieving the same in primary root canal treatment undertaken by undergraduates.[175]

A key tenet of the ESE[61] guidelines is that root canal debridement must be extended to the terminus of the canal system, which is expressed variously as extension to the "apical constriction," or to "0.5 to 2 mm from the radiographic apex," or to the "cementodentinal junction." This guideline is broadly supported by the fact that outcome of treatment is compromised by canal obstruction or failure to achieve patency to the canal terminus.[150,216,229] Ng and colleagues[150] reported a twofold reduction in the success of treatment when the patency to the canal terminus was not achieved. It could be speculated that the lack of mechanical negotiability of canals may be due to the presence of obstructions caused by "denticles," tertiary dentine, acute branching or a fine plexus of apical canals, or dentine/organic debris.

The continued debate on the optimal size of apical preparation remains topical in the absence of definitive evidence; the findings from relevant in vitro and clinical studies have been previously reviewed.[15] So far, four clinical outcome studies have considered this issue or have systematically investigated the effect of apical size of canal preparation on treatment outcome.[93,102,150,188,223,229] One randomized controlled trial revealed that enlargement of the canal to three sizes larger than the first apical binding file was adequate[188] (the mean final size was ISO #30). The observational studies[95,150,229] had not designed their investigation with apical canal size as their principal focus and neither had they found a statistically significant influence from this factor; nevertheless, they all reported the same inverse trend of decreasing success rates with an increase in size of apical preparation. It was speculated that canal preparation to larger apical sizes may compromise treatment success by generation of more apical dentine debris, which in the absence of an adequate irrigation regimen serves to block

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apical canal exits that may still be contaminated with bacteria. Continued generation of dentine debris, in the absence of sufficient irrigation, may lead to what is termed *dentine mud,* which ultimately creates a blockage. The impatient or neophyte clinician fails to resist the temptation to force the instrument back to length, resulting in the classically described procedural errors of apical transportation, canal straightening, and perforation. An alternative mechanism is required to explain the higher failures in initially large canals; it is likely that immature roots present a different debridement challenge, where the canal shape is not amenable to planing of the main portions of the canal by conventional instruments. Perhaps an intracanal brush may be a more suitable cleaning device in such teeth. The findings from these studies therefore do not concur with views that more effective bacterial debridement may be achieved with larger apical preparations.[33,164,184]

The issue of apical preparation size should be considered together with that of the size and taper of the rest of the canal preparation. Again, there is a paucity of sufficient direct evidence for the influence of degree of canal taper on root canal treatment outcome. The ESE guidelines[63] recommend only that canal preparation should be tapered from crown to apex without stipulating any particular degree of taper. Three studies have analyzed the influence of canal preparation taper on primary treatment and retreatment outcome, although again, none had focused their investigation primarily on this factor.[93,150,220] Smith and coworkers,[220] using loose criteria for determination of success, found that a “flared” preparation (wide taper) resulted in a significantly higher success rate compared with a “conical” preparation (narrow taper); the exact degree of taper was not reported and the effects of other treatment and nontreatment parameters were not controlled. In contrast, Hoskinson and associates[93] and Ng and colleagues,[150] using strict criteria, did not find any significant difference in treatment outcome between narrow (0.05) and wide (0.10) canal tapers. The controlled use of stainless steel instruments in a step-back technique may create 0.05 (1 mm step-back) or 0.10 (0.5 mm step-back) tapers, although, of course, uncontrolled use of such instruments may generate a variety of shapes. Ng and colleagues[150] also compared these (0.05 and 0.10) preparation tapers with 0.02, 0.04, 0.06, and 0.08 tapers (generally achieved by using greater taper nickel-titanium

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instruments) and found no significant effect on treatment outcome. They cautioned that their investigation of the influence of canal preparation taper without randomization could be influenced by the initial size of the canal, the type of instrument used, and operator experience.

Triangulation of the data on the effects of canal preparation size and taper on treatment outcome may intuitively lead to the conclusion that as far as current best evidence indicates, it is not necessary to overenlarge the canal to achieve periapical healing. An apical preparation size of ISO 30 with a 0.05 taper for stainless steel instrumentation or 0.06 taper for NiTi instrumentation is sufficient. Precisely what biologic and hydrodynamic mechanisms underpin such sufficiency is more difficult to define based on available evidence. Although a number of laboratory studies[4,84,112] have investigated the interaction between canal dimensions and irrigation or obturation dynamics, the precise physical, chemical, or biologic mechanisms that ultimately enable periapical healing remain unknown, although collaborations with fluid dynamics specialists[84] and (micro)biologists[82] may ultimately yield a clearer picture.

Procedural errors during root canal preparation include canal blockage, ledge formation, apical zipping and transportation, straightening of canal curvature, tooth or root perforation at the pulp chamber or radicular level, and separation of instruments. Instrument separation during treatment has been found to reduce the success rate significantly[150,229]; however, the reported prevalence of instrument separation was low (0.5% to 0.9%) in these studies, precluding an analysis of causative factors. A case-control study[225] revealed no significant difference in success rates between periapically involved teeth with or without retained separated instruments. The stage of canal debridement at which instrument separation occurred and the justification for their retention may have implications on the outcome. The coronoapical location of a separated instrument and whether the instrument was successfully bypassed were found to have no effect on treatment outcome.

Irrigant.

Different chemical agents have been used as irrigants for root canal treatment, singly or in various combinations, both in clinical practice and in the studies reviewed. They have included water, saline and solutions of local

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anesthetic, sodium hypochlorite, iodine, chloramine, sulfuric acid, EDTA, hydrogen peroxide, organic acid, Savlon, urea peroxide, and Biosept (a quaternary ammonium compound).[153] Most of the studies had used sodium hypochlorite as an irrigant,[153] regardless of whether it was primary treatment or retreatment. This is consistent with the ESE guidelines[61] for irrigation, which recommend a solution possessing disinfectant and tissue-dissolving properties.

One prospective study[150] systematically investigated the effect of the irrigant on the success rates of root canal retreatment, which, although not a randomized controlled trial, revealed interesting new findings on the effects of irrigants. Even though a higher concentration of sodium hypochlorite made negligible difference to treatment outcome, the additional use of other specific irrigants had a significant influence on success rates.[150] The finding of a lack of improvement in periapical healing with the use of a higher concentration NaOCl solution is consistent with previous clinical/microbiologic findings.[29,41] Comparing 0.5% to 5.0% NaOCl solution for irrigation, it was found that the concentration of solution, per se, did not appear to increase the proportion of teeth rendered culture-negative[29] or associated with greater periapical healing.[41] As iodine and sodium hypochlorite are both halogen-releasing agents and attack common key protein groups,[129] the finding that the additional use of 10% povidone-iodine for irrigation had no additional influence on treatment success was as expected. Surprisingly, however, the additional use of 0.2% chlorhexidine solution for irrigation was found to reduce the success of treatment significantly.[150] This finding was in complete contrast to previous reports[211,252] on its equivalent or superior in vivo antibacterial efficacy when compared with sodium hypochlorite solution. The use of chlorhexidine as a final irrigant following sodium hypochlorite irrigation had been recommended[107] and was justified on several grounds, including its substantivity in root dentin (i.e., prolonged antibacterial effect),[185] relative lack of toxicity,[117] and broad-spectrum efficacy.[130] Not until recently has alternate irrigation with sodium hypochlorite and chlorhexidine solution raised serious concerns because of their interaction product. The interaction product is thought to be an insoluble precipitate containing para-

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chloroaniline, which is cytotoxic and carcinogenic.[14,24] Apart from mutually depleting the active moiety in the two solutions for bacterial inactivation, the precipitate may cause persistent irritation to the periapical tissue and block dentinal tubules and accessory anatomy, possibly explaining the observed lower success rate when chlorhexidine was used as an additional irrigant.

Ng and associates[150] also found that the additional use of EDTA had a profound effect on improving radiographically observed periapical healing associated with root canal treatment (OR = 1.5 [1.1, 2.0]). In contrast, the observed synergistic effect of sodium hypochlorite and EDTA had been previously demonstrated in terms of bacterial load reduction[26] but not periapical healing. The long-term (≥2 years) outcome of their cases stratified by canal disinfection protocols[25] did not support their microbiologic findings. Their reported success rate for alternate irrigation with sodium hypochlorite and EDTA solutions (67%) was low when compared to the success rate for irrigation using saline (91%), 0.5% sodium hypochlorite (92%), or 5% sodium hypochlorite (86%) solutions.[162] The reported outcome data were unexpected, as preobturation negative bacterial culture was achieved in all cases. Given the complexity of their study design (clinical and microbiologic), their sample size was restricted to 11 to 15 teeth per group, limiting their outcome data. The synergistic effect of the two disinfectants has been attributed to the chelating properties of the sodium salts of EDTA, and their roles have been reviewed by Zehnder.[261] EDTA solution assists negotiation of narrow or sclerosed canals by demineralization of root dentine and helps in the removal of compacted debris from noninstrumented canal anatomy. It may also facilitate deeper penetration of sodium hypochlorite solution into dentine by opening dentinal tubules and removing the smear layer from the instrumented surface. Lastly, it may help detach or break up biofilms adhering to root canal walls.[85]

Medicament.

Most previous treatment outcome studies have not standardized the type of root canal medicament used in the interappointment period, but the use of several different medicaments has been reported. The list was consistent with that recommended in the ESE guidelines for a medicament with disinfectant properties and included calcium hydroxide, creosote, and iodine solutions.[153]

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However, there is an absence of studies investigating the influence of this factor on treatment outcome.

The use of a mixture of calcium hydroxide and chlorhexidine has been tested based on the speculation that the mixture would be more effective against *E. faecalis.*[13,78,198]

Root canal bacterial culture results prior to obturation.

In the past, in various centers of endodontic excellence, completion of root canal treatment by obturation would only be acceptable after a negative culture test was obtained from the canal, confirming the absence of bacteria in the part of the root canal system that could be sampled.[23,70,143] This practice has fallen out of clinical favor because of the perceived predictability and good prognosis of root canal treatment without microbiologic sampling. Sampling procedures are considered to be lengthy, difficult, and often inaccurate, requiring laboratory support and having low benefit-to-cost ratio.[138,139] A preobturation negative culture result may increase treatment success twofold (Fig. 17.19). One large study[201] helped contribute to the demise of the canal-culture test; however, even this study showed a 10% difference in success in favor of the negative culture test when periapical disease was present. The outcome is even worse when a positive culture test result combines with the presence of a periapical lesion.

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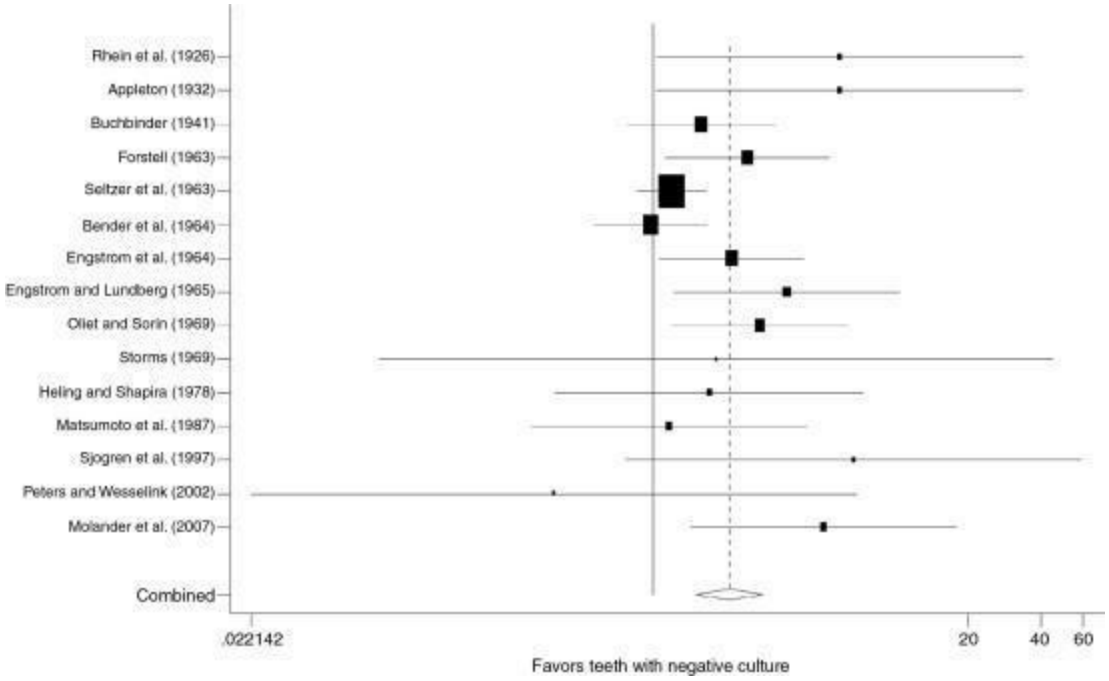

FIG. 17.19 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with preobturation negative versus positive culture test results (pooled OR = 2.1; 95% CI: 1.5, 2.9).

Forest plot shows individual study against Logor_negvspos_culture.

The horizontal axis is marked.022142, 20, 40, and 60. The vertical axis from bottom to top is marked as follows: Combined, Molander et al. (2007), Peters and Wesselink (2002), Sjogren et al. (1997), Matsumoto et al. (1987), Heling and Shapira (1978), Storms (1969), Oliet and Sorin (1969), Engstrom and Lundberg (1965), Engstrom et al. (1964), Bender et al. (1964), Seltzer et al. (1963), Forstell (1963), Buchbinder (1941), Appleton (1932), and Rhein et al. (1926). The data points for all studies lie in-between near the center and three-fourth length of the horizontal axis before the point 20.

Numerous studies[2,7,10,16,26-29,33,37,41,42,60,77,79,99,104,108,110,124,137,154,161,163,168,169,173,179,181,208,209,210,214,2] have evaluated the effect of different stages of root canal treatment on the intraradicular microbiota, both qualitatively and quantitatively (Table 17.9). Some studies merely report positive culture tests, whereas others have identified and quantified intraradicular microbiota before and after various stages of treatment.

Table 17.9

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Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Presence by Culture

			PERCENTAGE OF SAMPLES WITH BACTERIAL PRESENCE		
Study	**Year**	**Sample Size**	**At Baseline**	**After Preparation ± Irrigation**	**Next Visit (After Dressing±)**
Auerbach	1953	60 teeth	93% (56/60)	**Chlorinated soda (double strength):** 22% (12/56)	—
Ingle and Zeldow	1958	89 teeth	73% (65/89)	**H_2O:** 70% (62/89) Some initially −ve became +ve after treatment	—
Stewart et al.	1961	77 teeth	100% (77/77)	**0.5% NaOCl + Gly-oxide:** 2% (1/44) **0.5% NaOCl + 3% H_2O_2:** 9% (3/33)	**No dressing:** 0.5% NaOCl + Gly-oxide: 34% (15/44) 0.5% NaOCl + 3% H2O2: 39% (17/33)
Nicholls	1962	155 teeth	100% (155/155)	**Alkaline chloramine:** 53% (39/74) **H_2O_2 and 2% NaOCl:** 50% (30/60) **H_2O and 2% NaOCl:** 71% (15/21)	—
Grahnén and Krasse	1963	97 teeth	77% (75/97)	**NaCl:** 72% (23/32) **Biocept:** 66% (21/32) **Nebacin:** 36% (12/33) Some initially −ve became +ve after Tx	**No dressing:** NaCl: 47% (15/32) Biocept: 47% (15/32) Nebacin: 18% (6/33)
Engström	1964	223 teeth (untreated or retreated)	60% (134/223)	**Biosept or Iodophor, plus alcohol, chloroform, and 0.5% NaOCl:** No data	**5% I_2 in 10% IKI:** 2nd visit: 43% (58/134); 3rd visit: 22% (29/134); 4th visit: 8% (9/134); 5th visit: 3% (4/134); 6th visit: 2% (3/134); 7th visit: 16% (22/134)

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Olgart	1969	207 teeth	72% (149/207)	**H_2O_2 and 0.5% NaOCl or H_2O_2 and 1% NaOCl:** 43% (88/207)	**No dressing:** 34% (70/207)
Bence et al.	1973	33 teeth	100% (33/33)	**Pre-irritation:** 1st file: 93%, enlargement with #3: 14%, #4: 11%, #5: 21% (32% of instruments showed +ve culture, regardless of size) **5.25% NaOCl:** 48-h culture: 4% dentine, 10% pp 5-day culture: 8% dentine, 26% pp	**No dressing:** 8% dentine, 12% pp samples of teeth with negative culture after irrigation
Akpata	1976	20 extracted teeth	100% (20/20)	**NaCl:** 65% (13/20)	**38% CMCP:** 20% (2/10) When PP sample −ve, crushed tooth yielded −ve culture; When PP +ve, crushed teeth yielded +ve or −ve cultures
Cvek et al.	1976	108 teeth	NaCl group: 53% (18/34) 0.5% NaOCl group: 63% (29/46) 5% NaOCl group: 79% (22/28)	**NaCl:** 83% (15/18) **0.5% NaOCl:** 59% (17/29) **5% NaOCl:** 68% (15/22)	—
Byström and Sundqvist	1981	15 teeth	100% (15/15)	**Saline:** 100% (15/15)	**No dressing:** 47% (7/15) (5th visit) Where initial bacteria load high, difficult to eliminate
Byström and Sundqvist	1983	15 teeth	100% (15/15)	**0.5% NaOCl:** 87% (13/15)	**No dressing:** 20% (3/15) (5th visit)
Byström and Sundqvist	1985	60 teeth	100% (60/60)	**0.5% NaOCl:** No data **5% NaOCl:** No data **5% NaOCl + 15% EDTA:** No data	**No dressing:** 0.5% NaOCl: 12/20 (2nd visit); 8/20 (3rd visit) 5% NaOCl: 10/20 (2nd visit); 6/20 (3rd visit) 5% NaOCl + 15% EDTA: 11/20 (2nd visit); 3/20 (3rd visit)

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Byström et al.	1985	65 teeth	100% (65/65)	**0.5% NaOCl:** No data **5.0% NaOCl:** No data	**CH:** 0/35 (1 month), 1/35 (2–4 days) **CP/CMCP** (2 weeks): 10/30
Sjogren and Sundqvist	1987	31 teeth	100% (31/31)	**0.5% NaOCl plus ultrasonic debridement:** No data	**No dressing:** 29% (9/31) at 2nd visit; 23% (7/31) at 3rd visit
Koontongkaew et al.	1988	15 teeth	100% (15/15)	**3% H_2O_2/5.25% NaOCl:** No data	**CMCP**: 1-day dressing: 40% (2/5); 3-day dressing: 20% (1/5); 7-day dressing 10% (1/10) **No dressing:** 60% (3/5) after 1 day, 20% (1/5) after 3 or 7 days
Reit and Dahlen	1988	35 teeth	91% (32/35)	**0.5% NaOCl:** No data	**CH**: After 14 days: 23% (8/35); after 21 days: 26% (9/35)
Molander et al.	1990	25 teeth	96% (24/25)	**0.04% iodine:** No data	**Clindamycin**: After 14 days: 16% (4/25); after 21 days: 24% (6/25)
Sjogren et al.	1991	30 teeth	100% (30/30)	**0.5% NaOCl:** 50% (15/30)	**CH:** 10 min: 50% (6/12) at 1 week later 7 day: 0% (0/18) (none after 1–5 weeks later without dressing)
Orstavik et al.	1991	23 teeth	96% (22/23)	**NaCl** irrigation and enlarged to: #20–25: 87% (20/23) further to #35–80: No data	**CH**: 34% (8/23); #35/40: 40% (6/15) #>40: 25% (2/8)
Yared and Bou Dagher	1994	60 teeth	100% (60/60)	**1% NaOCl:** Enlarged to #25: 73% (22/30) Enlarged to #40: 23% (7/30)	**CH:** 0% (0/60)
Gomes et al.	1996	42 root canals: Untreated (n = 15) Retreated (n = 27)	95% (40/42)	**2.5% NaOCl:** No data	**Empty canal** (7-10 days): 73% (29/40)
Sjogren et al.	1997	55 teeth (single canal)	100% (55/55)	**0.5% NaOCl:** 40% (22/55)	—

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Dalton et al.	1998	46 teeth	100% (46/46)	NaCl + NiTi files: 68% (15/22); NaCl + K-files: 75% (18/24)	—
Reit et al.	1999	50 teeth	84% (42/50)	Enlarged to #35 (curved) or #50 (straight) with **0.5% NaOCl**: No data	**5% IKI** (5–7 days): 44% (22/50) **Empty** (7 days): 44% (22/50)
Peciuliene et al.	2000	25 teeth	80% (20/25);	**2.5% NaOCl and 17% EDTA:** No data	**Medication unknown:** 28% (7/25)
Shuping et al.	2000	42 teeth	98% (41/42)	**1.25% NaOCl:** 38% (16/42)	**CH:** 8% (3/40)
Lana et al.	2001	31 teeth	87% (27/31)	**2.5% NaOCl:** No data	**CH:** 13% (4/31) **Empty for 7 days:** 23% (7/31)
Peciuliene et al.	2001	40 teeth	83% (33/40)	**2.5% NaOCl and 17% EDTA:** 30% (10/33)	**CH (10–14 days):** 25% (5/20) **IKI: 2% I_2** in 4% KI (10 min): 5% (1/20)
Peters et al.	2002	42 teeth	Instrumentation to #20: 100% (42/42)	**Enlarged to #35 with 2% NaOCl:** 23% (10/42)	**CH** (4 weeks): 71% (15/21); further irrigation: 43% (9/21)

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Card et al.	2002	40 mandibular teeth/canals	95% (38/40)	**1% NaOCl** Profile instrumentation (0.04 taper): 0/13 of cuspids and bicuspids, 5/27 of mesiobuccal canals Further LightSpeed instrumentation to size 57.5–65: 3/27 mesiobuccal canals of molars Only 1/16 of those mesiobuccal canals with detectable communication with the mesiolingual canals had +ve culture after the first preparation using ProFile instruments	No data
Kvist et al.	2004	96 teeth	98% (94/96)	**0.5% NaOCl:** 63% (60/96)	**CH** (7 days): 36% (16/44) **IPI** (10 min): 29% (15/52)
Chu et al.	2006	88 canals	99% (87/88)	**0.5% NaOCl:** No data	**CH, Septomixine forte, or Ledermix**: 36% (32/88) Exposure of pulp, tooth type, acute versus chronic condition, size of lesion, and type of medication had no significant effect
Paquette et al.	2007	22 teeth (single canal)	100% (22/22)	**2.5% NaOCl:** 68% (15/22)	**2% CHX**: 45% (10/22)
Siqueira et al.	2007a	11 teeth (single root)	100% (11/11)	**2.5% NaOCl:** 55% (6/11)	**CH/CPMC:** 9% (1/11)
Siqueira et al.	2007b	11 teeth (single root)	100% (11/11)	**2.5% NaOCl:** 45% (5/11)	**CH:** 18% (2/11)

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Vianna et al.	2007	24 teeth (single root)	100% (24/24)	**Saline + 2% CHX gel:** 33% (8/24)	**2% CHX, CH or mixture:** 54% (13/24) Type of medication had no significant effect
Wang et al.	2007	43 canals	91% (39/43)	**Saline + 2% CHX gel:** 8% (4/39)	**2% CHX + CH:** 8% (3/36) Size of apical preparation (40 versus 60) had no significant effect.
Markvart et al.	2012	24 teeth	88% (21/24)	**2.5% NaOCl:** 63% (15/24)	**17% EDTA irrigation and 10 min 5% IKI medication:** 50% (12/24) Box preparation (#60): 67% (8/12) Cone preparation (#25–30): 33% (4/12)
Xavier et al.	2013	48 teeth (single canal)	100% (40/40)	**1% NaOCl:** 75% (9/12) **2% CHX:** 75% (9/12)	**CH:** 75% (18/24)

CH, Calcium hydroxide; *CP*, camphorated phenol; *CMCP*, camphorated monochlorophenol; *NaOCl*, sodium hypochlorite; *PP*, paper point sample; *r culture*, culture test before obturation.

The effect of the "mechanical preparation" of the canal(s) on the microbiota has been tested using only water or saline as the irrigant. Taken collectively, the studies show that negative cultures were achieved on a weighted pooled average in 31% of the cases (range 0% to 79%). When sodium hypochlorite (concentration range 0.5% to 5%) irrigation supplemented the "mechanical preparation," the frequency of negative cultures immediately increased to a weighted pooled average of 52% (range 13% to 95%) (see Table 17.9).

Most studies report culture reversals during the interappointment period when active antibacterial dressing is not used in the root canal system between appointments. The reversals are due to regrowth of residual bacteria or recontamination by bacteria from coronal restoration leakage. When active interappointment antibacterial dressing is used, negative cultures in the subsequent visit were achieved on average in 71% of cases (range 25% to 100%) (see Table 17.9).

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Effect of persistent bacteria on root canal treatment outcome.

The bacteria present in preobturation cultures have included *Enterococcus*, *Streptococcus*, *Staphylococcus*, *Lactobacillus*, *Veillonella*, *Pseudomonas*, *Fusobacterium* species, and yeasts. Studies have been variable as to the relationship between individual species and treatment failure. Although the overall failure rate for cases with positive cultures was 31%, teeth testing positive for *Enterococcus* species had a failure rate of 55%, and teeth with positive cultures for *Streptococcus* species had 90% failures.[70] In another study, good-quality root canal treatment on 54 teeth with asymptomatic periapical disease gave an overall success rate of 74%, but teeth with positive cultures for *Enterococcus faecalis* achieved a success rate of only 66%.[231] The success rate for teeth with no bacteria was 80%, whereas that for teeth with bacteria in the canal before obturation was 33%. These associations cannot be regarded as direct cause-and-effect associations, but they further emphasize the need to determine a relationship between microbial diversity and treatment outcome.

A monkey-model study[63] used a four- or five-strain infection to test the effect of debridement and obturation procedures on outcome. When bacteria remained after chemomechanical debridement, 79% of the root canals were associated with nonhealed periapical lesions, compared with 28% when no bacteria were found to remain. Combinations of several residual bacterial species were more frequently related to nonhealed lesions than were single strains. When no bacteria remained at the end of chemomechanical debridement, healing occurred independently of the quality of the root filling. In contrast, when bacteria remained in the canal system, there was a greater correlation with nonhealing associated with poor-quality root fillings than in technically well-performed fillings. In root canals where bacteria were found after removal of the root filling, 97% had not healed, compared with 18% for those root canal systems with no bacteria detected upon removal of root filling. The study emphasizes the importance of reducing bacteria below detection limits before permanent root filling in order to achieve optimal healing conditions for the periapical tissues. It also reinforces the view that obturation does indeed play a role when residual infection is present.

Regardless of the technique used for obtaining a sample for a canal culture, the presence of a negative culture seems to have a positive impact on

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treatment outcome. The association of specific species with treatment failure is not well established but the identity of the small group of species isolated from positive cultures is relatively constant and may hold answers to treatment resistance and failure. However, it is important to understand that there are many other factors that can influence root canal treatment outcome.

Root filling material and technique.

The interrelationship among the core root filling material, sealer (for filling the gaps between the core material and canal surface), and technique for their placement complicates the investigation of the effect of obturation and technique on treatment outcome. In previous studies on treatment outcome, the most commonly used core root filling material was gutta-percha with various types of sealer or gutta-percha softened in chloroform (chloropercha).[153] The sealers used may be classified into zinc oxide–eugenol–based, glass ionomer–based, and resin-based types.[153] Materials such as Resilon, SmartSeal, and MTA have been adopted but have not significantly penetrated the market, except for the use of MTA in surgical repairs or repairs for immature apices. In any case, there is no evidence to show that the nature of root filling material and the technique used for placement has any significant influence on treatment outcome.

Apical extent of root filling.

Of the many intraoperative factors associated with success and failure of root canal treatment, the apical extent of the root canal filling material has been the most frequently and thoroughly investigated. In these previous studies, the apical extent of root fillings has been classified into three categories for statistical analyses: more than 2 mm short of radiographic apex (short), 0 to 2 mm within the radiographic apex (flush), and extended beyond the radiographic apex (long).[153] The apical extent of root filling was found to have a significant influence on the success rates of treatment, regardless of the periapical status.[150,153] Flush root fillings were associated with the highest success rates, whereas long root fillings were associated with the lowest success rates.

Most previous retrospective studies could not distinguish between the effects of apical extent of instrumentation versus the apical extent of

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obturation; however, the London Eastman study[150] was able to separate the effect of these two factors and found them both to independently and significantly affect periapical healing. The factors correlated with each other, consistent with the fact that canals are normally filled to the same extent as canal preparation.

Extrusion of cleaning, medication, or filling materials beyond the apical terminus into the surrounding tissues may result in delayed healing or even treatment failure due to a foreign body reaction.[105,146,218,260] Magnesium and silicon from the talc-contaminated extruded gutta-percha were found to induce a foreign body reaction, resulting in treatment failure.[146] An animal study has shown that large pieces of subcutaneously implanted gutta-percha in guinea pigs were well encapsulated in collagenous capsules, but fine particles of gutta-percha induced an intense, localized tissue response.[218] The inference that perhaps extrusion of large pieces of gutta-percha may not impact periapical healing was not supported by data from previous studies.[150,153] The discrepancy may possibly be accounted for by bacterial contamination of the extruded gutta-percha in the clinical data.

The radiographic evidence of “sealer puffs” extruding through the main apical foramina and lateral/accessory canals has been pursued with vigor by some endodontists based on the undaunted belief of its value as “good practice.” Their perception is that this represents a measure of root canal system cleanliness, and they ardently argue that healing would follow, albeit with some delay. The published evidence on the effects of sealer extrusion into the periapical tissues has been contradictory. Friedman and colleagues[68] found that extrusion of a glass ionomer-based sealer significantly reduced success rates. In contrast, Ng and associates[150] reported that extrusion of a zinc oxide–eugenol based–sealer had no significant effect on periapical healing. The discrepancy may be attributed to the difference in sealer type and the duration of treatment follow-up. The radiographic assessment of the presence or resorption of sealer may be complicated by the radiolucent property of its basic components and the insufficient sensitivity of the radiographic method used to detect small traces of it.[150] It is possible that, in some cases, the radiographic disappearance of extruded sealer may simply be due to resorption of the radio opaque additive, barium sulfate, or its uptake by macrophages, still resident in the vicinity.[146]

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Extruded glass ionomer–based[68] zinc oxide–eugenol–based,[98] silicone-based[98] sealers or endomethasone[22] were not found to be resorbed/absorbed by periapical tissues after 1 year. Traces of calcium hydroxide–based sealer (Sealapex) could still be detected after 3 years.[194] In the latter study, treatments were carried out on primary molar teeth and the canals were obturated with Sealapex without gutta-percha. With longer duration of follow-up, complete resorption of extruded zinc oxide–eugenol–based sealers (Procosol, Roth Elite)[8] and a resin-based sealer (AH Plus, Dentsply/DeTrey, Konstanz, Germany)[194] was demonstrated in 69% and 45% of the cases after 4 and 5 years, respectively. Ng and associates[150] advanced two explanations for the difference between the effect of extruded core gutta-percha and the zinc oxide–eugenol sealer: the latter is antibacterial and may kill residual microorganisms, whereas it is also more soluble and readily removed by host cells compared to gutta-percha.

Quality of root filling.

Another much investigated parameter of obturation in retrospective studies has been the radiographic measure of the "quality of root filling." The rationale for complete obturation of the root canal system is to prevent recontamination by colonization from the residual infection or newly invading bacteria. Both are supposedly prevented by a "tight" seal with the canal wall and an absence of voids within the body of the material. Quality of root filling may therefore be regarded either as poor root-filling technique or as a surrogate measure of the quality of the entire root canal treatment, because good obturation relies on properly executed preliminary steps in canal preparation. A systematic review[153] reported that the criteria for judging the quality of root fillings have not been well defined in previous studies.[43,93,216] An unsatisfactory root filling has been defined as "inadequate seal," "poor apical seal," or "radiographic presence of voids." Nevertheless, satisfactory root fillings were found to be associated with significantly higher success rates than unsatisfactory root fillings.[153]

Acute exacerbation during treatment.

The causes for interappointment "flare-up" or pain have not been precisely determined, and several hypothetical mechanisms involving chemical,

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mechanical, or microbial injury to the periradicular tissues, as well as psychological influences, have been suggested as contributory to postpreparation pain or swelling.[202,203] Although these factors have not been specifically studied in the context of periapical healing, acute "flare-ups" during treatment were not found to be significantly associated with periapical healing in two studies.[102,216] In contrast, the London Eastman study[150] found that pain or swelling occurred in 15% of cases after chemomechanical debridement and was found to significantly reduce success as measured by periapical healing. This interesting finding may be explained by the hypothesis that "flare-ups" were caused by extrusion of contaminated material during canal preparation. Such material may elicit a foreign body reaction or (transient) extraradicular infection, resulting in treatment failure in a proportion of such cases. Alternatively, acute symptoms may be the result of incomplete chemomechanical debridement at the first appointment, leading to a shift in canal microbial ecology favoring the growth of more virulent microorganisms, leading to further postpreparation pain and treatment failure. The exact biologic mechanisms of failure in these cases remain obscure and warrant further investigation.

Number of treatment visits.

The number of treatment visits for completing root canal treatment and its effect on periapical healing remains an ongoing controversy. Generally, the argument for single-visit treatment centers around better patient acceptability and cost-effectiveness versus the preference of multiple-visit treatments based on biologic rationale.[224] The premise for multiple-visit treatments has been that primary debridement is not completely effective in eliminating all the adherent bacterial biofilm,[147] and the residual bacteria may multiply and recolonize the canal system.[25,29] Therefore the proponents consider it desirable to use the interappointment period to dress the canal with a long-lasting or slow-release antibacterial agent capable of destroying or incapacitating residual bacteria, as well as to take the opportunity to gauge the initial periapical response before root filling. Calcium hydroxide has served in this capacity for many years because of its ability to dissolve organic tissue, kill bacteria, detoxify antigenic material, and act as a slow-release agent because of its low solubility-product in an aqueous

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environment. However, its antibacterial ability has come under close scrutiny, with advocates suggesting that the material is not suitable for this purpose.[195] A final resolution to this debate is awaited based on robust clinical evidence. Most of the published randomized controlled trials found no significant influence of healing attributable to number of treatment visits, but they all lacked robust statistical power.

The debate about the merits of single- or multiple-visit treatments will continue unabated given the respective strengths of the motivational drivers among the opposing groups. The issue may only be resolved by properly documented, large randomized controlled trials (which are currently unavailable) because undocumented variables (i.e., operator skill, biologic or technical case complexity, and patient compliance) would continue to bias the outcome.

Post root canal treatment restorative factors

Effect of quality and type of restoration.

The placement of a coronal restoration after root canal obturation is the final step in the management of teeth undergoing root canal treatment. It has been shown to have a major influence on endodontic outcomes. Teeth with satisfactory coronal restorations were found to have significantly better periapical healing compared with those with unsatisfactory restorations (OR = 3.31; 95% CI: 1.07, 10.30).[153] The term *satisfactory restorations* has been defined as a restoration with no evidence of marginal discrepancy, discoloration, or recurrent caries with absence of a history of decementation.[93,182]

Given that one of the roles of coronal restorations is to prevent postoperative root canal reinfection via coronal leakage, the criteria for unsatisfactory restoration given by Hoskinson and colleagues[93] could not infer coronal leakage when the inner core was still intact. Consequently, the London Eastman study[150] adopted a different classification and definition for unsatisfactory restorations in order to illustrate obvious and potential coronal leakage more accurately. The two groups of unsatisfactory restorations were defined as those with (1) obvious signs of exposed root filling and (2) potential leakage indicated by marginal defects and history of decementation.

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It is perhaps this strategy that contributed to the finding of a profound effect (OR = 10.7; 95% CI: 3.7, 31.5) of coronal leakage on the endodontic outcome.

A number of investigations have been performed based on comparisons between the types of post root canal treatment restorations, including permanent versus temporary restorations,[43,68,150] crown versus acrylic restorations,[43,68,150,216] presence versus absence of posts,[68,150] and nonabutment versus abutment.[150,216] Teeth that had been permanently restored were associated with significantly higher success rates than their temporarily restored counterparts in some studies[43,68] but not in others.[38,150] The type of permanent restoration[43,68,150,216] was found to have no significant influence on the outcome of treatment.

It has often been recommended that it would be wise to provide a subseal over the root filling in case of loss of a permanent or temporary restoration; the subseal would be glass ionomer (GIC) or zinc oxide–eugenol cement.[196] The placement of a GIC or zinc oxide–eugenol (IRM) cement lining coronal to the gutta-percha filling and underneath the permanent core in order to provide an additional antibacterial coronal seal was found in a prospective study to have no beneficial effect on treatment success.[150]

In summary, the preceding findings overall support the ESE guidelines[61] that an adequate restoration should be placed after root canal treatment to prevent subsequent bacterial recontamination. Therefore the provision of a good-quality coronal restoration, regardless of type, should be considered the final part of the root canal treatment procedure following obturation.

Use of root treated teeth as abutments for prostheses and occlusal contacts.

Mechanical stress on restorations is a function of the role of individual teeth in the occlusal scheme. The pattern of occlusal loading both in static and dynamic occlusion is dictated by whether the teeth are involved as single units or abutments (bridge/denture) and whether they have holding or guiding contacts. It is reasonable to expect that bridge and denture abutments may be placed under unfavorable loads, as may last-standing teeth in the dental arch.[127] These teeth may therefore be expected to have lower success rates because of a potential increase in the development of cracks and fractures due

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to fatigue. This observation has been confirmed for teeth functioning as bridge abutments compared with those restored as individual units following root canal treatment.[216]

Summary of factors influencing periapical healing following nonsurgical root canal treatment

The following factors are considered as having a major impact on periapical health subsequent to root canal treatment:

- Presence (Fig. 17.20) and size (Fig. 17.21) of periapical lesion
- Patency at the canal terminus (achieving patency significantly increased the chance of success twofold)[150]
- Apical extent of chemomechanical preparation in relation to the radiographic apex (Fig. 17.22)
- Outcome of intraoperative culture test (see Fig. 17.19)
- Iatrogenic perforation (if present, reduces the odds of success by 30%)[150]
- Quality of root canal treatment judged by the radiographic appearance of the root filling (Fig. 17.23)
- Quality of the final coronal restoration (Fig. 17.24)

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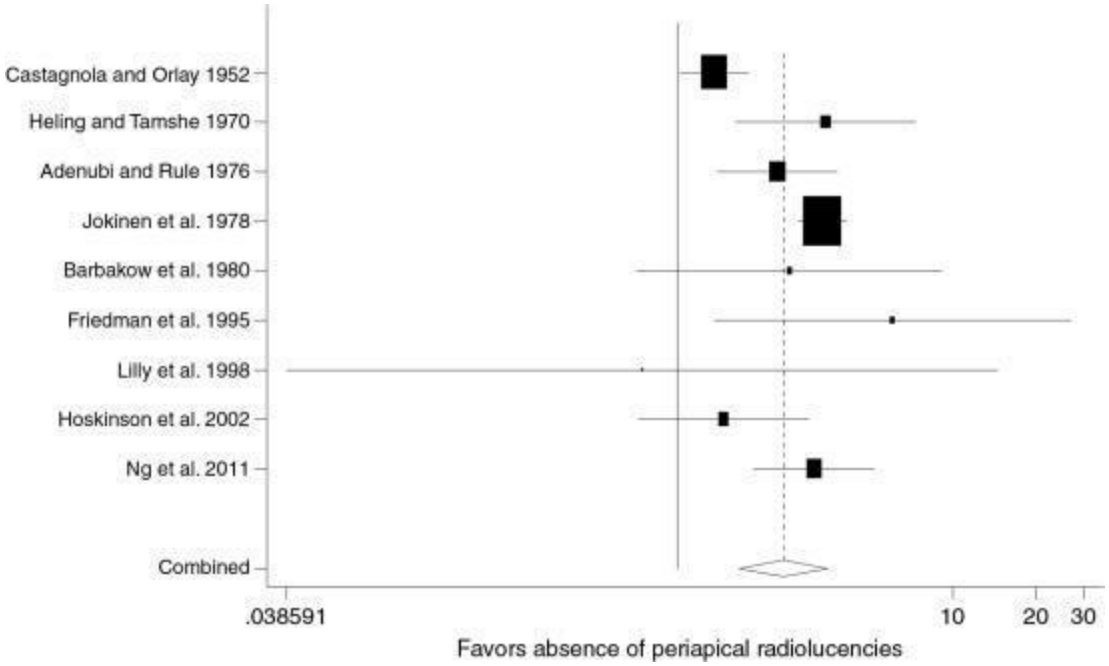

FIG. 17.20 Forest plot showing pooled and individual study's odds ratio *(OR)* for periapical health of teeth undergoing root canal treatment with preoperative nonvital pulps and absence of periapical radiolucencies versus teeth with nonvital pulps and presence of periapical radiolucencies (pooled OR = 2.4; 95% CI: 1.7, 3.5).

Forest plot shows combined and individual study against LogOR_nonvital_nopa_pa.

The horizontal axis is marked.038591, 10, 20, and 30. The vertical axis from bottom to top is marked as follows: Combined, Ng et al. 2011, Hoskinson et al. 2002, Lilly et al. 1998, Friedman et al. 1995, Barbakow et al. 1980, Jokinen et al. 1978, Adenubi and Rule 1976, Heling and Tamshe 1970, Castagnola and Orlay 1952. The data points for each study lie in-between near the center and three-fourth length of the horizontal axis before the point 10.

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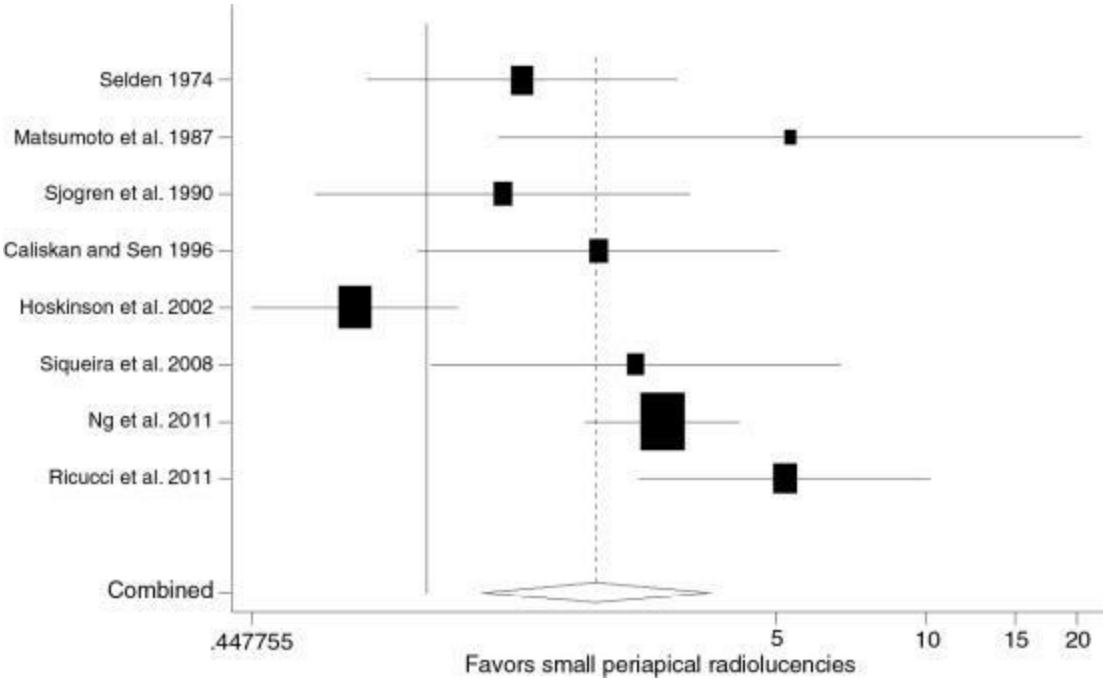

FIG. 17.21 Forest plot showing pooled and individual study's odds ratio *(OR)* for periapical health of teeth undergoing root canal treatment with preoperative large (>5 mm) versus small (<5 mm) periapical radiolucencies (pooled OR = 2.2; 95% CI: 1.3, 3.7).

Forest plot shows combined and individual study against LogOR_small_largelesion_all.

The horizontal axis is marked.0447755, 5, 10, 15, and 20. The approximate data are as follows: Combined: 2.5 to 4, Ricucci et al. 2011: 5.1, Ng et al. 2011: 3.7, Siqueira et al. 2008: 3.6, Hoskinson et al. 2002: 1.5, Calisken and Sen 1996: 2.8, Sjogren et al. 1990: 2.5, Matsumoto et al. 1987: 5.1, and Selden 1974: 2.7.

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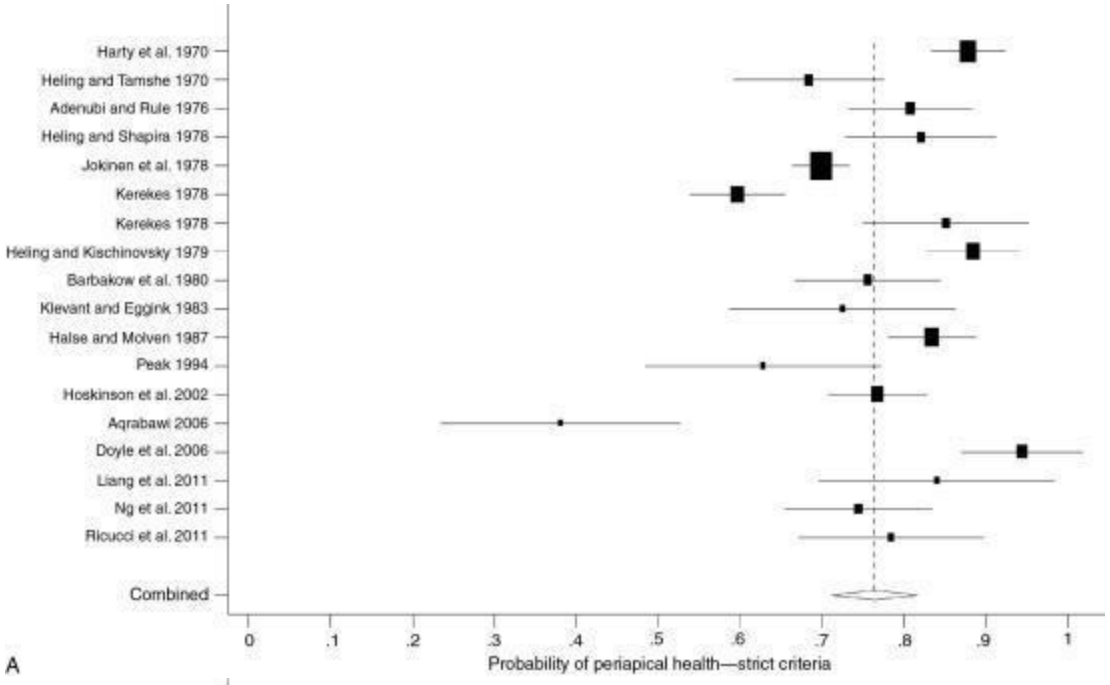
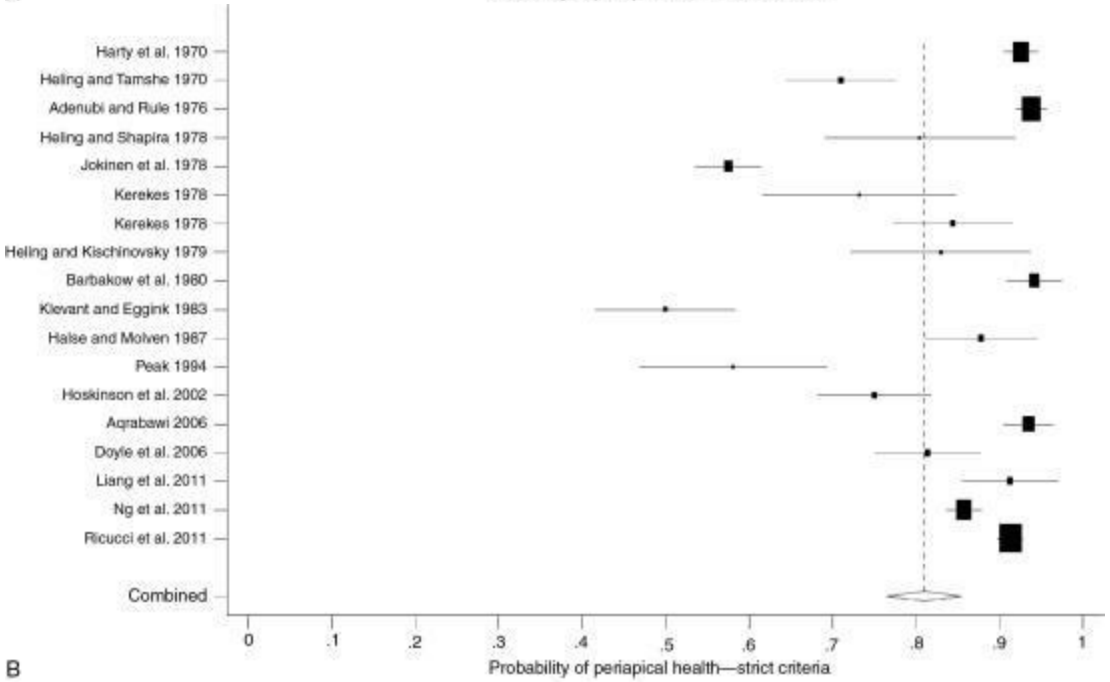


A



B

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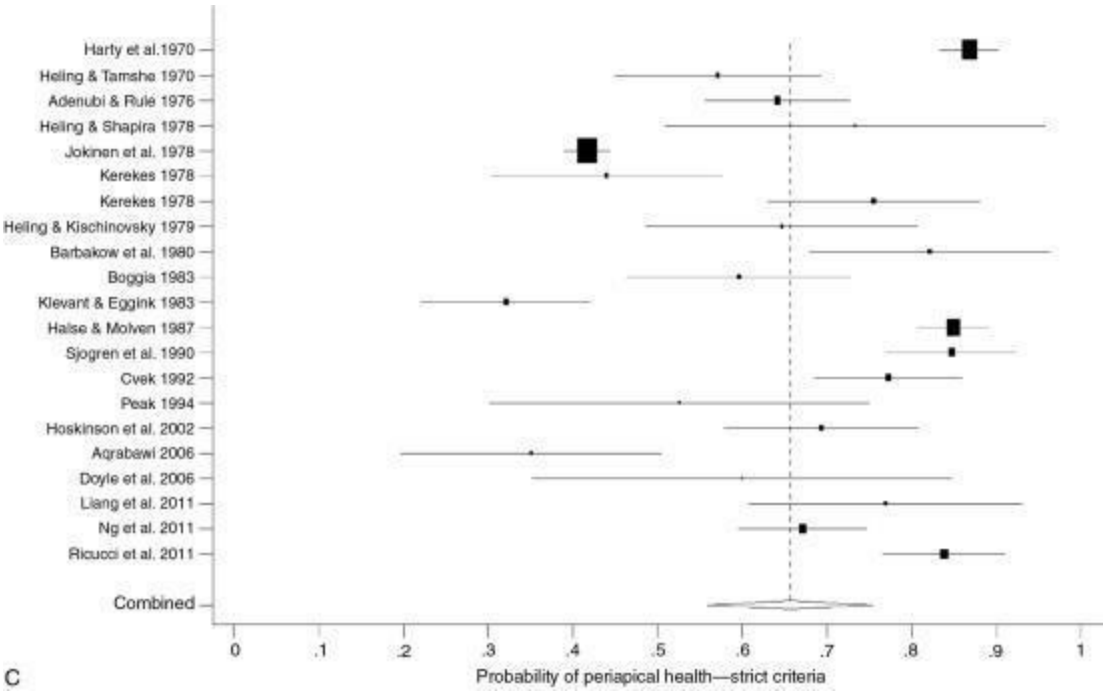

FIG. 17.22 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment with underextended (0.76 [0.71, 0.82]) **(A)**, flush (0.81 [0.76, 0.86]) **(B)**, or overextended (0.66 [0.56, 0.75]) (**C)** root fillings.

Forest plot shows combined and individual study against UNDERFSS_pr.

The horizontal axis ranges from 0 (near the origin) to 1 in increments of 0.9. The approximate data are as follows: Combined: 0.7 1 to 0.8 1, Ricucci et al. 2011: 0.7 8, Ng et al. 2011: 0.7 4, Liang et al. 2011: 0.8 4, Doyle et al. 2006: 0.9 5, Aqrabawi 2006: 0.3 9, Hoskinson et al. 2002: 0.7 6, Peak 1994: 0.6 1, Halse and Molven 1987: 0.8 3, Klevant and Eggink 1983: 0.7 1, Barbakow et al. 1980: 0.7 5, Heling and Kischinovsky 1979: 0.8 8, Kerekes 1978: 0.8 4, Kerekes 1978: 0.5 9, Jokinen et al. 1978: 0.7 9, Heling and Shapira 1978: 0.8 3, Adenubi and Rule 1976: 0.8 2, Heling and Tamshe 1970: 0.7 9, and Harty et al. 1970: 0.8 9.

B) Forest plot shows combined and individual study against FLUSHSS_pr.

The horizontal axis ranges from 0 (near the origin) to 1 in increments of 0.9. The approximate data are as follows: Combined: 0.7 7 to 0.8 6, Ricucci et al. 2011: 0.9 1, Ng et al. 2011: 0.8 6, Liang et al. 2011: 0.9 1, Doyle et al. 2006: 0.8 1, Aqrabawi 2006: 0.9 3, Hoskinson et al. 2002: 0.7 5, Peak 1994: 0.5 7, Halse and Molven 1987: 0.8 7, Klevant and Eggink 1983: 0.4 9, Barbakow et al. 1980: 0.5, Heling and Kischinovsky 1979: 0.9 2, Kerekes 1978: 0.8 2, Kerekes 1978: 0.8 4, Jokinen et al. 1978: 0.5 7, Heling and Shapira 1978: 0.7 9, Adenubi and Rule 1976: 0.9 2, Heling and Tamshe 1970: 0.7 2, and Harty et al. 1970: 0.9.

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C) Forest plot shows combined and individual study against OVERFSS_pr.

The horizontal axis ranges from 0 (near the origin) to 1 in increments of 0.9. The approximate data are as follows: Combined: 0.5 7 to 0.7 5, Ricucci et al. 2011: 0.8 4, Ng et al. 2011: 0.6 8, Liang et al. 2011: 0.7 8, Doyle et al. 2006: 0.6, Aqrabawi 2006: 0.3 4, Hoskinson et al. 2002: 0.7 5, Peak 1994: 0.5 2, Cvek 1992: 0.7 8, Sjogren et al. 1990: 0.8 5, Halse and Molven 1987: 0.8 5, Klevant and Eggink 1983: 0.3 1, Boggia 1983: 0.5 8, Barbakow et al. 1980: 0.8 1, Heling and Kischinovsky 1979: 0.6 4, Kerekes 1978: 0.7 6, Kerekes 1978: 0.4 3, Jokinen et al. 1978: 0.4, Heling and Shapira 1978: 0.7 3, Adenubi and Rule 1976: 0.6 3, Heling and Tamshe 1970: 0.5 4, and Harty et al. 1970: 0.8 5.

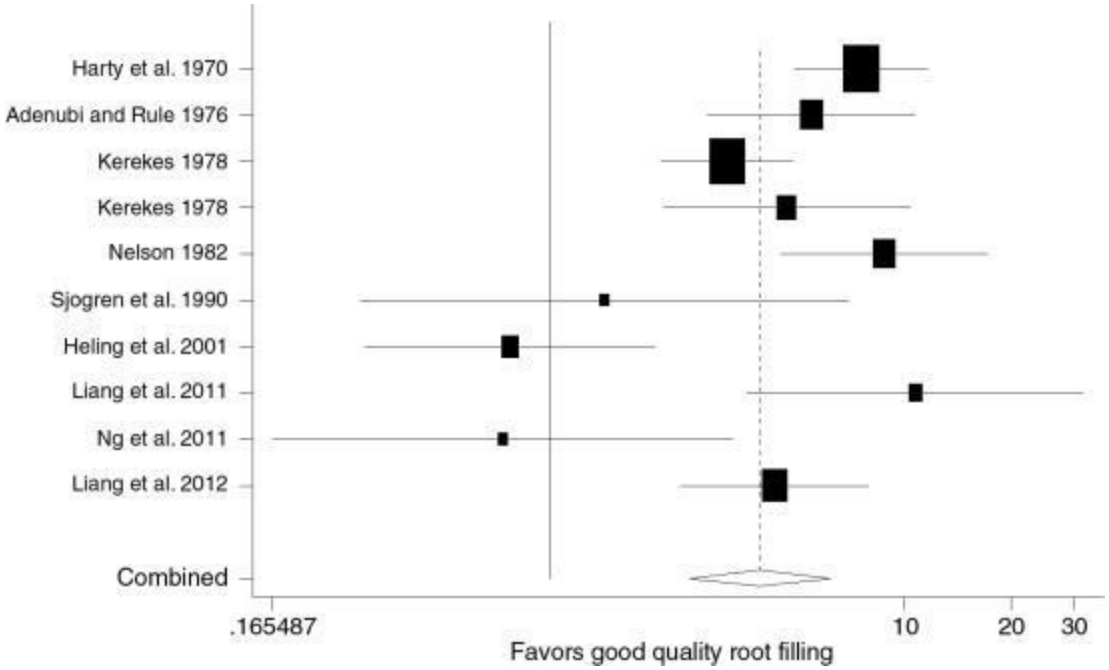

FIG. 17.23 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with good quality versus suboptimal quality root filling (pooled OR = 3.9; 95% CI: 2.5, 6.2).

Forest plot shows combined and individual study against Logor_satRF_unsatRF.

The horizontal axis is marked at 0.1 6 5 4 8 7, 10, 20, and 30. The vertical axis is marked from bottom to top as follows: Combined, Liang et al. 2012, Ng et al. 2011, Liang et al. 2011, Heling et al. 2001, Sjogren et al. 1990, Nelson 1982, Kerekes 1978, Kerekes 1978, Adenubi and Rule 1976, and Harty et al. 1970. The data points for all studies lie in-between one-fourth length of the

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horizontal axis and 10.2.

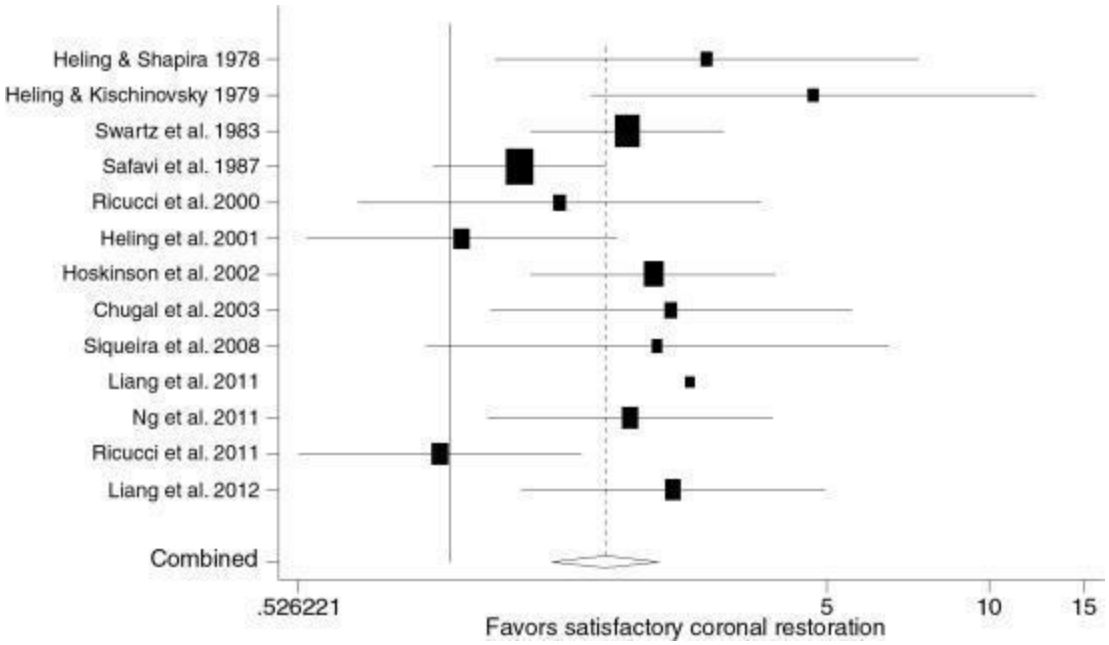

FIG. 17.24 Forest plot showing pooled and individual study's odds ratio (OR) of periapical health for teeth undergoing root canal treatment with satisfactory versus unsatisfactory coronal restoration at follow-up (pooled OR = 1.9; 95% CI: 1.5, 2.5).

Forest plot shows combined and individual study against Logor_satrest_unsatrest.

The horizontal axis is marked at 0.5 2 6 2 2 1, 5, 10, and 15. The vertical axis is marked from bottom to top as follows: Combined, Liang et al. 2012, Ricucci et al. 2011, Ng et al. 2011, Liang et al. 2011, Siqueira et al. 2008, Chugal et al. 2003, Hoskinson et al. 2002, Heling et al. 2001, Ricucci et al. 2000, Safavi et al. 1987, Swartz et al. 1983, Heling & Kischinovsky 1979, Heling & Shapira 1978. The data points for all studies lie in-between near one-fourth length of the horizontal axis and 5 (three-fourth length of the horizontal axis).

The following factors are considered as having minimal impact on root canal treatment outcome:

- Age of patient
- Gender of patient
- Tooth morphologic type

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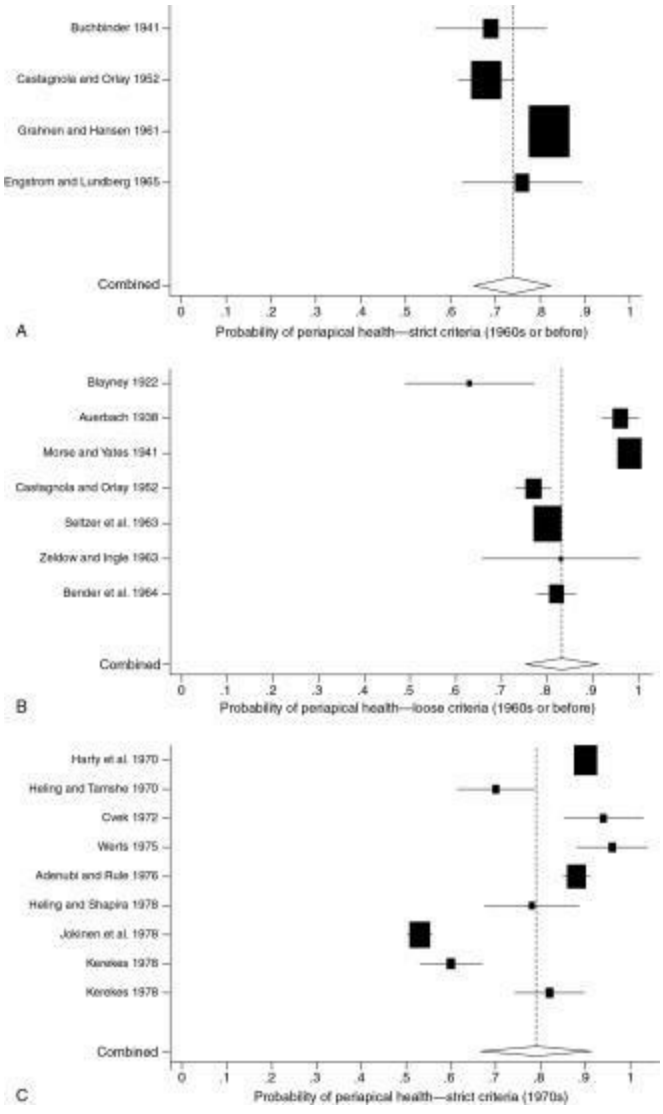
- Specific root canal treatment protocol and technique (preparation, irrigation, and obturation material and technique)

Contemporary improvements in mechanical and chemical canal preparation have not resulted in an increase in the success rate for root canal treatment over the past century (Fig. 17.25). This observation may be explained by the currently available techniques not being effective in eliminating the infection in the apical canal anatomy.



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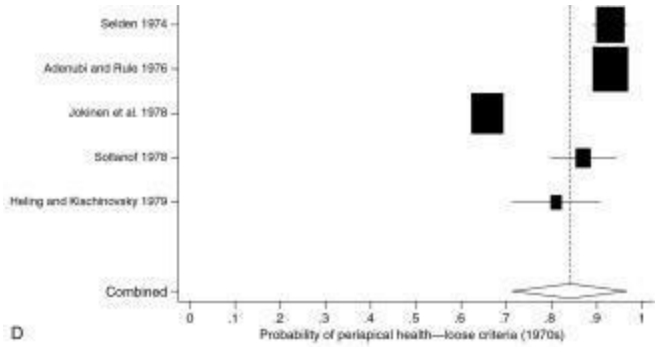

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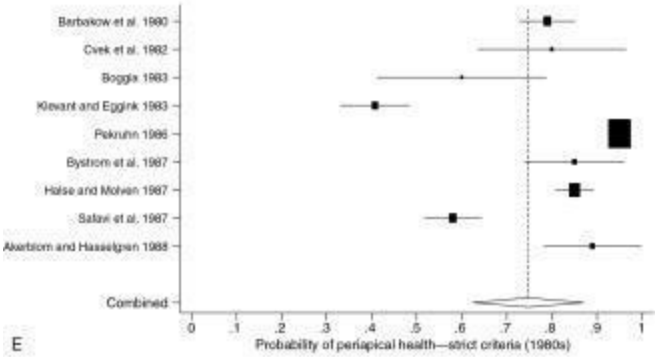

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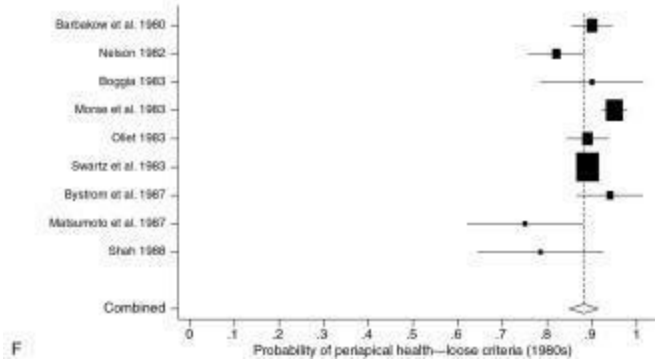

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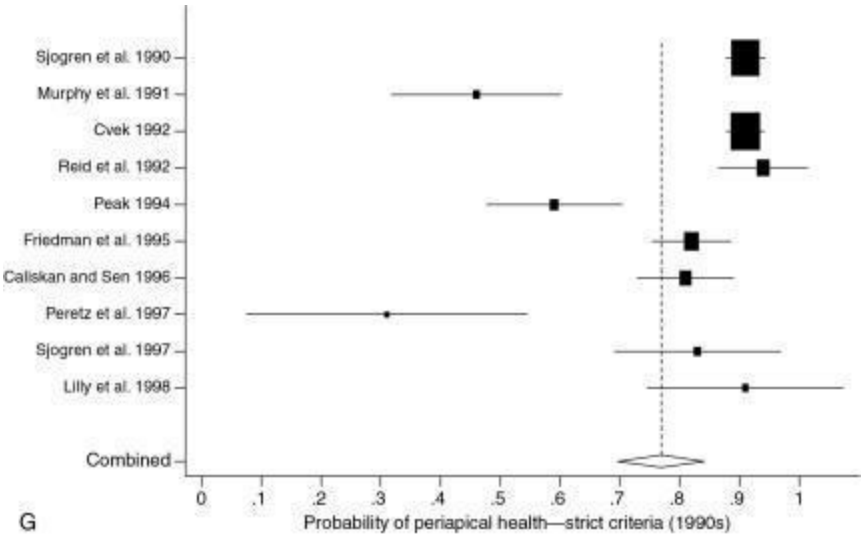

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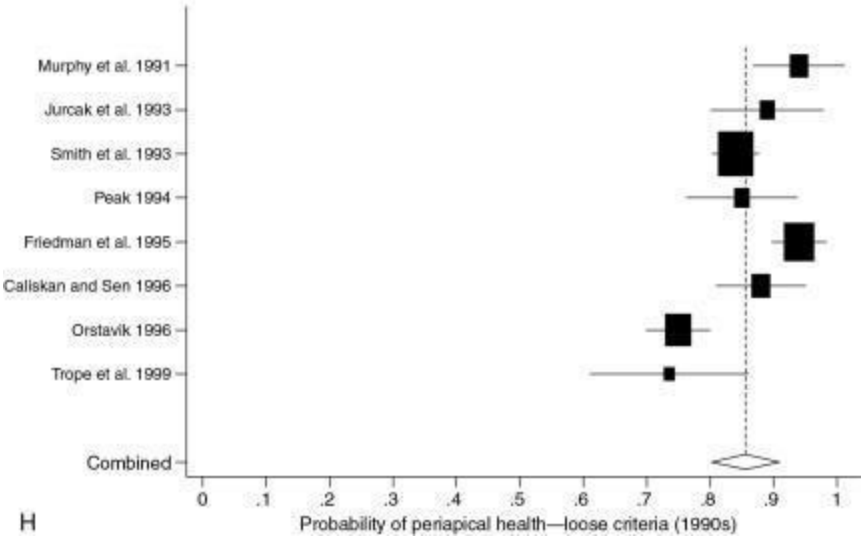

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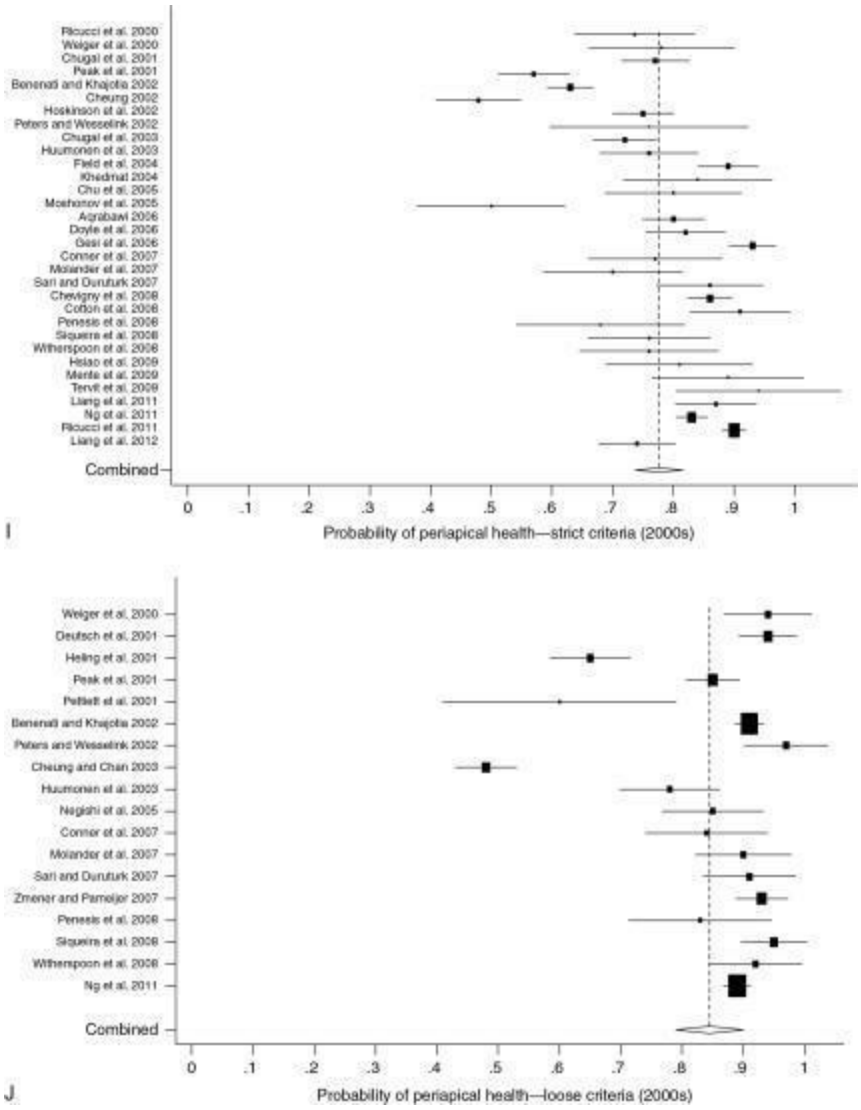

FIG. 17.25 Forest plot showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment by "decade of publication" and "criteria for success."

A) Forest plot shows combined and individual study against Probability of success—strict.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.6 6 to 0.8 2, Engstrom and Lundberg 1965: 0.7 6, Grahnen and Hansen 1961: 0.8 2, Castagnola and Orlay 1952: 0.6 5, and Buchbinder 1941: 0.6 2.

B) Forest plot shows combined and individual study against Probability of success—loose.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.7 6 to 0.9 1, Bender et al. 1964: 0.8 2, Zeldow and Ingle 1963: 0.8 3, Seltzer et al. 1963: 0.8, Castagnola

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and Orlay 1952: 0.7 6, Morse and Yates 1941: 0.9 8, Auerbach 1938: 0.9 5, and Blayney 1922: 0.6.

C) Forest plot shows combined and individual study against Probability of success—loose.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.6 8 to 0.9, Kerekes 1978: 0.8 2, Kerekes 1978: 0.6, Jokinen et al. 1978: 0.5 2, Heling and Shapira 1978: 0.7 9, Adenubi and Rule 1976: 0.8 8, Werts 1975: 0.9 5, Cvek 1972: 0.9 4, Heling and Tamshe 1970: 0.7, and Harty et al. 1970: 0.9.

D) Forest plot shows combined and individual study against Probability of success—loose.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined:.7 1 to 0.9 6, Heling and Kischinovsky 1979: 0.8 1, Soltanof 1978: 0.8 9, Jokinen et al. 1978: 0.6 3, Adenubi and Rule 1976: 0.9 2, and Selden 1974: 0.9 2.

E) Forest plot shows combined and individual study against Probability of success—strict.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.6 2 to 0.8 8, Akerblom and Hasselgren 1988: 0.9, Safavi et al. 1987: 0.5 9, Halse and Molven 1987: 0.8 5, Bystrom et al. 1987: 0.8 5, Pekruhn 1986: 0.9 5, Klevant and Eggink 1983: 0.4 1, Boggia 1983: 0.5 9, Cvek et al. 1982: 0.8, and Barbakow et al. 1980: 0.7 9.

F) Forest plot shows combined and individual study against Probability of success—loose.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.8 6 to 0.9 1, Shah 1988: 0.7 9, Matsumoto et al. 1987: 0.7 6, Bystrom et al. 1987: 0.9 4, Swartz et al. 1983: 0.8 8, Oliet 1983: 0.8 9, Morse et al. 1983: 0.9 4, Boggia 1983: 0.9, Nelson 1982: 0.8, and Barbakow et al. 1980: 0.9.

G) Forest plot shows combined and individual study against Probability of success—strict.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.7 to 0.8 3, Lilly et al. 1998: 0.9, Sjogren et al. 1997: 0.8 2, Peretz et al. 1997: 0.3, Calisken and Sen 1996: 0.8, Friedman et al. 1995: 0.8 2, Peak 1994: 0.5 9, Reid et al. 1992: 0.9 2, Cvek 1992: 0.9, Murphy et al. 1991: 0.4 4, and Sjogren et al. 1990: 0.9.

H) Forest plot shows combined and individual study against Probability of success—loose.

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The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.8 to 0.9, Trope et al. 1999: 0.7 3, Orstavik 1996: 0.7 5, Calisken and Sen 1996: 0.9 8, Friedman et al. 1995: 0.9 4, Peak 1994: 0.8 4, Smith et al. 1993: 0.8 3, Jurcak et al. 1993: 0.8 8, and Murphy et al. 1991: 0.9 2.

I) Forest plot shows combined and individual study against Probability of success—strict.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.7 4 to 0.8 1, Liang et al. 2012: 0.7 3, Ricucci et al. 2011: 0.9, Ng et al. 2011: 0.8 3, Liang et al. 2011: 0.8 8, Tervit et al. 2009: 0.9 3, Mente et al. 2009: 0.8 9, Hsiao et al. 2009: 0.8 1, Witherspoon et al. 2008: 0.7 5, Siqueira et al. 2008: 0.7 5, Penesis et al. 2008: 0.6 8, Cotton et al. 2008: 0.9, 2008: 0.8 6, Sari and Duruturk 2007: 0.8 6, Molander et al. 2007: 0.6 9, Conner et al. 2007: 0.7 8, Gesi et al. 2006: 0.9 2, Doyle et al. 2006: 0.8 2, Aqrabawi 2006: 0.7 9, Moshonov et al. 2005: 0.5, Chu et al. 2005: 0.8, Khedmat 2004: 0.8 3, Field et al. 2004: 0.8 9, Huumonen et al. 2003: 0.7 6, Chugal et al. 2003: 0.7, Peters and Wesselink 2002: 0.7 5, Hoskinson et al. 2002: 0.7 4, Cheung 2002: 0.5, Benenati and Khajotia 2002: 0.6 6, Peak et al. 2001: 0.5 5, Chugal et al. 2001: 0.7 7, Weiger et al. 2000: 0.7 9, and Ricucci et al. 2000: 0.7 3.

J) Forest plot shows combined and individual study against Probability of success—loose.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.8 9, Ng et al. 2011: 0.8 9, Witherspoon et al. 2008: 0.9 1, Siqueira et al. 2008: 0.9 5, Penesis et al. 2008: 0.8 2, Zmener and Pameijer 2007: 0.9 3, Sari and Duruturk 2007: 0.9 1, Molander et al. 2007: 0.9, Conner et al. 2007: 0.8 3, Negishi et al. 2005: 0.8 4, Huumonen et al. 2003: 0.7 8, Cheung and Chan 2003: 0.4 9, Peters and Wesselink 2002: 0.9 7, Benenati and Khajotia 2002: 0.9, Pettiett et al. 2001: 0.6, Peak et al. 2001: 0.8 3, Heling et al. 2001: 0.6 4, Deutsch et al. 2001: 0.9 2, and Weiger et al. 2000: 0.9 2.

It is notable that all factors that have a strong influence on periapical health after root canal treatment are associated in some way with a persistent root canal infection. Further improvements in root canal treatment outcomes may therefore be achieved by understanding the nature of the root canal infection (especially apically) and the manner in which the microbiota is altered or eliminated during root canal treatment.

Factors affecting tooth survival following root canal treatment

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A systematic review and meta-analysis has shown that 93% of endodontically treated teeth survive at 2 years postoperatively; but this survival reduced to 88% at 10 years following treatment (Fig. 17.26). The most common reasons for such tooth loss were due to problems of endodontic origin, tooth/root fracture, or restoration failure.[149,151] Consistent with the studies on periapical healing, the considerations affecting the survival of endodontically treated teeth may be divided into patient, intraoperative, and restorative factors.

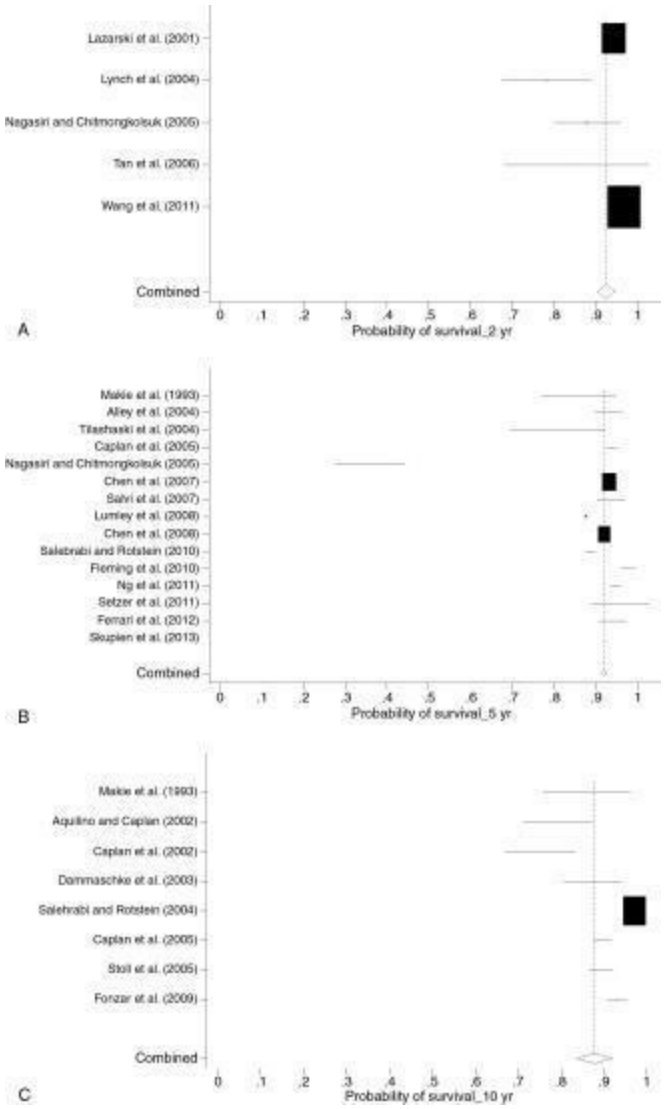

FIG. 17.26 Forest plot showing pooled and individual study's probability of 2-year (pooled probability = 0.93; 95% CI: 0.90, 0.95) **(A)**, 5-year (pooled probability = 0.92; 95% CI: 0.91, 0.93) **(B)**, and 10-year (pooled probability = 0.88; 95% CI: 0.84, 0.92) **(C)** survival of teeth undergoing root canal treatment.

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A) Forest plot shows combined and individual study against Rate_2 year.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.9 2 to 0.9 3, Wang et al. (2011): 0.9 7, Tan et al. (2006): 0.8 2, Nagasiri and Chitmongkolsuk (2005): 0.8 5, Lynch et al. (2004): 0.7 8, and Lazarski et al. (2001): 0.9 3.

B) Forest plot shows combined and individual study against Rate_5 year.

The approximate data are as follows: Combined: 0.9 2, Skupien et al. (2013): 0.9 3, Ferrari et al. (2012): 0.9 4, Setzer et al. (2011): 0.9 6, Ng et al. (2011): 0.9 4, Fleming et al. (2010): 0.9 7, Salebrabi and Rotstein (2010): 0.8 9, Chen et al. (2008): 0.9 2, Lumley et al. (2008): 0.8 8, Salvi et al. (2007): 0.9 2, Chen et al. (2007): 0.9 2, Nagasiri and Chitmongkolsuk (2005): 0.3 5, Caplan et al. (2005): 0.9 4, Tilashaski et al. (2004): 0.8, Alley et al. (2004): 0.9 3, and Makie et al. (1993): 0.8 2.

C) Forest plot shows combined and individual study against Rate_10 year.

The approximate data are as follows: Combined: 0.8 4 to 0. 9 1, Fonzar et al. (2009): 0.9 2, Stoll et al. (2005): 0.8 8, Caplan et al. (2005): 0.8 9, Salehrabi and Rotstein (2004): 0.9 6, Dammaschke et al. (2003): 0.8 4, Caplan et al. (2002): 0.7 3, Aquilino and Caplan (2002): 0.7 8, and Makie et al. (1993): 0.8 4.

Patient factors

Ng and colleagues[151] found that teeth in patients suffering from diabetes or receiving systemic steroid therapy had a higher chance of being extracted after root canal treatment. The negative influence of diabetes on tooth survival is consistent with the report by Mindiola and associates,[135] whereas the influence of steroid therapy had never been reported previously. It may be argued that patients with diabetes are more susceptible to periodontal disease[74] or have a lower success rate of root canal treatment[67] because of being immunologically compromised. However, the researchers reported that over 50% of such teeth were extracted due to persistent pain. Some of these observations may be explained by the presence of neuropathy, a debilitating painful complication of diabetes.[57] It is interesting to note that systemic steroid therapy is often prescribed to control such chronic pain.[39,51,101]

Tooth morphologic type and location

Tooth types (i.e., location within the arch) may vary with respect to their

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susceptibility to tooth fracture, a common reason for tooth loss after endodontic treatment. Ng and coworkers[149] found that tooth type had a significant influence on survival. Maxillary premolars and mandibular molars were found to have the highest frequency of extraction, with tooth fracture being the most common reason. The observation is consistent with previous reports on fracture incidence of maxillary premolars and mandibular molars.[56,109] The factors "proximal contacts" and "terminal (last standing) teeth" were found to affect tooth survival considerably,[149,151] but they were significantly correlated with molar teeth. Most of the extractions of terminal teeth or teeth with one or fewer proximal contacts were due to tooth fracture.[151] The observation may be explained by the unfavorable distribution of occlusal forces and higher nonaxial stress on terminal teeth and those with fewer than two proximal contacts. Other possible reasons for their higher rate of loss are (1) failure of root canal treatment in a terminal tooth may be accepted more willingly because of little perceived aesthetic value and (2) clinicians may be less likely to offer further treatment on terminal molar teeth due to difficult access. Therefore when restoring molar teeth, the favorable distribution of occlusal forces must be considered, especially on teeth with one or fewer adjacent teeth or on terminal teeth.

Preoperative conditions of teeth

The presence of preoperative periapical lesions, which is the most significant prognostic factor for periapical healing, was found to have no significant influence on tooth survival.[151] On the other hand, the presence of preoperative periodontal probing defects of endodontic origin, preoperative pain, and preoperative sinus tracts were found to reduce tooth survival.[151] These observations are consistent with a previous report that the mere presence of a persistent periapical lesion was not a sufficient reason for dentists and patients to opt for further treatment, either retreatment or extraction.[180] The negative impact of preoperative pain on survival outcome highlights the importance of accurate pain diagnosis. In some instances, the pain may have been of nonendodontic origin and therefore would persist after treatment.[176] In other instances, preoperative pain of endodontic origin may persist following treatment, as a result of peripheral or central sensitization. Therefore effective pain diagnosis and management for patients presenting

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with preoperative pain are crucial.

The presence of preoperative cervical resorption and perforation was also found to significantly reduce tooth survival.[151] This was as expected because tooth fracture and reinfection due to leakage through the resorption and perforating defects are likely sequelae in such cases. In the presence of reinfection, clinicians are more inclined to suggest extraction due to the intuitive perception of poor long-term prognosis of such teeth.

Treatment factors

Considering all of the intraoperative factors, the "lack of patency at apical foramen" and the "extrusion of gutta-percha root filling" were found to be the most significant intraoperative factors in reducing tooth survival.[151] In the presence of persistent problems and knowing that the treatment objective of cleaning to the canal terminus could not be achieved, patients and dentists may be more likely to opt for extraction sooner than later.

Restorative factors

Protection of teeth with crowns or cast restorations has not been shown to influence periapical healing; however, the placement of good cores had a positive effect on endodontic outcome. In contrast, placement of crowns or cast restorations was found to improve tooth survival.[149,151,189] This suggests that crowns and cast restorations help prevent tooth fracture, whereas the mere placement of a satisfactory core is sufficient to prevent reinfection after endodontic treatment. Unfortunately, the study was not able to investigate the interrelationship between tooth morphologic type, the extent of tooth tissue loss after treatment, or the type of final restoration. Although the clinical inference from these findings is that cast restorations should preferably be placed on all teeth after root canal treatment, this is probably a gross exaggeration of the true need. On the basis of laboratory[178] and clinical findings,[144] posterior teeth with compromised marginal ridges (mesial or distal), together with heavy occlusal loading evidenced by faceting, may benefit from full coverage restorations. The restoration design should attempt to preserve as much remaining tooth tissue as possible; the implication is that the so-called nonaesthetic but technically demanding partial veneer onlays and partial coverage crowns would be the restorations of choice for root-

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treated teeth. In anterior teeth, the missing tooth tissue may often be adequately replaced with composite restorative materials. A crown is only indicated when tooth structure or aesthetics become compromised.

The use of cast post and cores for retention of restorations have also been found to reduce tooth survival.[149,151] It may be speculated that the presence of a post has different effects on anterior versus posterior teeth as they are subjected to different directions and amount of occlusal force. It has been reported that only 12% of the extracted teeth with cast post and cores were incisors or canines. Therefore the inference is that the use of such retention should be particularly avoided in premolar and molar teeth. Alternative treatment options should be considered for molar or premolar teeth lacking sufficient tooth structure.

Ng and colleagues[151] observed that teeth functioning as prosthetic abutments had poorer survival rates; however, the number of teeth (n = 94) in the study that functioned as abutments was too small a sample to be considered statistically significant. As previously described, the explanation may reside in the excessive and unfavorable distribution of occlusal stresses on abutment teeth. If possible, root-treated teeth should be avoided as abutments for prostheses or in provision of occlusal guidance in excursive movements.

Summary of factors influencing the survival of teeth following root canal treatment

The following conditions have been found to significantly improve tooth survival following root canal treatment:

- Nonmolar teeth (Fig. 17.27)
- Teeth with both mesial and distal adjacent teeth (Fig. 17.28)
- Teeth not located as the distal-most tooth in the arch[151]
- Teeth (molar) with cast restorations after treatment (Fig. 17.29)
- Teeth not requiring cast post and core for support and retention of restoration[111,151]
- Teeth not functioning as abutments for fixed prosthesis[3,111,191,151]
- Absence of preoperative deep periodontal probing defects, pain, sinus tract, or perforation[151]

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■ Achievement of patency at canal terminus and absence of root-filling extrusion during treatment[151]

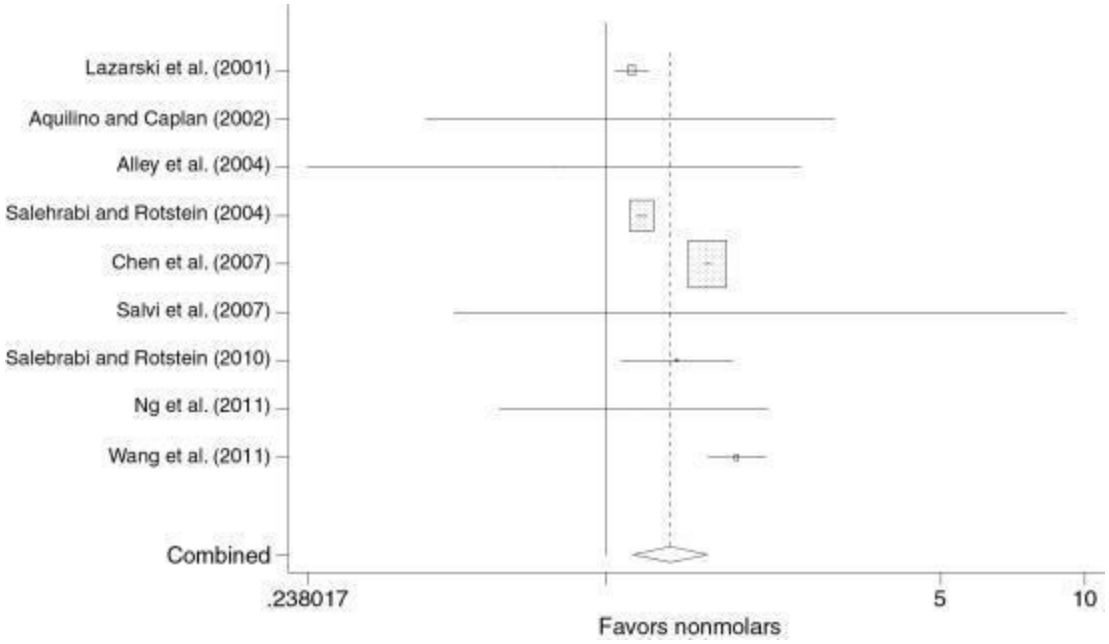

FIG. 17.27 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for nonmolar versus molar teeth (pooled OR = 1.4; 95% CI: 1.1, 1.6).

Forest plot shows combined and individual study against Logor_nonmolar_molar.

The horizontal axis is marked at 0.2 3 8 0 1 7, 5, and 10. The vertical axis is marked from bottom to top as follows: Combined, Wang et al. (2011), Ng et al. (2011), Salebrabi and Rotstein (2010), Salvi et al. (2007), Chen et al. (2007), Salehrabi and Rotstein (2004), Alley et al. (2004), Aquilino and Caplan (2002), and Lazarski et al. (2001). The data points for all studies lie in between approximately 2.5 to 3.7.

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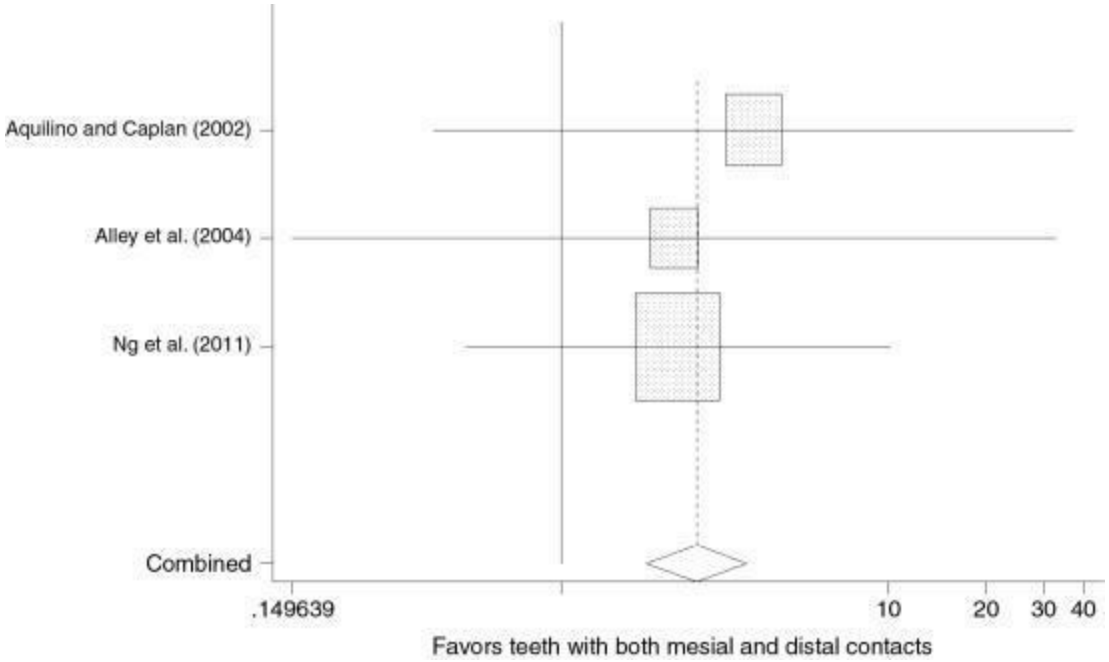

FIG. 17.28 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth with both mesial and distal contacts present versus absent (pooled OR = 2.6; 95% CI: 1.8, 3.7).

Forest plot shows combined and individual study against Logor_proximal2_01.

The horizontal axis is marked at 0.1 4 9 6 3 9, 10, 20, 30, and 40. The vertical axis is marked from bottom to top as follows: Combined, Ng et al. (2011), Alley et al. (2004), and Aquilino and Caplan (2002). The data points for all studies lie in between two-fourth and three-fourth length of the horizontal axis.

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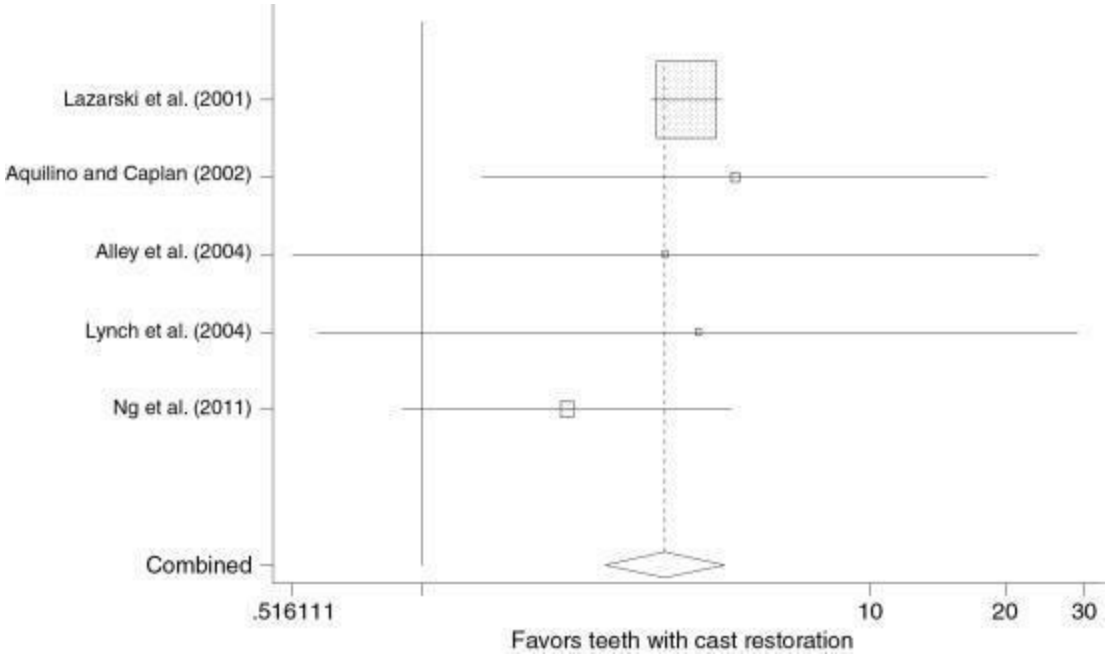

FIG. 17.29 Forest plot showing pooled and individual study's odds ratio (OR) of survival probability for teeth restored with cast versus plastic restoration (pooled OR = 3.5; 95% CI: 2.6, 4.7).

Forest plot shows combined and individual study against Logor_crown_nocrown.

The horizontal axis is marked at 0.5 1 6 1 1 1, 10, 20, and 30. The vertical axis is marked from bottom to top as follows: Combined, Ng et al. (2011), Lynch et al. (2004), Alley et al. (2004), Aquilino and Caplan (2002), and Lazarski et al. (2001). The data points for all studies lie in between lie near and at the center of horizontal axis.

In addition, it is important to ensure favorable distribution of occlusal forces when designing restorations for endodontically treated teeth.

Impact of root canal treatment on quality of life

The impact of root canal treatment on the OHRQoL of patients has been evaluated using the short form (OHIP-14) or the modified version (OHIP-17) of the Oral Health Impact Profile (OHIP-14) (Table 17.10).[219] The distinctly positive impact of root canal treatment was apparent, regardless of cultural background of the patient group or the measure used.[55,73,87,115] As expected, physical pain, psychological discomfort (feeling tense), and disability (difficulty in relaxing) were the most improved domains following treatment.

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Table 17.10

The OHIP-14 and Modified Oral Health Impact Profile Instrument (OHIP-17)

OHIP-#	Item Question
OHIP-1	Have you had trouble pronouncing words because of your teeth and mouth?
OHIP-2	Have you felt that your sense of taste has worsened because of your teeth or mouth?
OHIP-3	Have you had painful aching in your mouth?
OHIP-4	Have you found it uncomfortable to eat any foods because of your teeth or mouth?
OHIP-5*	Have you had to alter the temperature of the foods that you eat because of your teeth or mouth?
OHIP-6	Have you been self-conscious because of your teeth or mouth?
OHIP-7	Have you felt tense because of your teeth or mouth?
OHIP-8	Has your diet been unsatisfactory because of your teeth or mouth?
OHIP-9	Have you had to interrupt meals because of your teeth or mouth?
OHIP-10	Have you found it difficult to relax because of your teeth or mouth?
OHIP-11*	Have you found it difficult to fall asleep because of your teeth or mouth?
OHIP-12*	Have you ever been awakened by problems with your teeth or mouth?
OHIP-13	Have you been embarrassed because of your teeth or mouth?
OHIP-14	Have you been irritable with other people because of your teeth or mouth?
OHIP-15	Have you had difficulty doing your usual jobs because of problems with your teeth or mouth?
OHIP-16	Have you felt that life in general was less satisfying because of your teeth or mouth?
OHIP-17	Have you been totally unable to function because of your teeth or mouth?

OHIP-14 contains all the items except those with an asterisk (*).
OHIP, Oral Health Impact Profile.

From Dugas NN, Lawrence HP, Teplitsky P, Friedman S: Quality of life and satisfaction

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outcomes of endodontic treatment, *J Endod* 28:819-827, 2002.

Outcome of nonsurgical retreatment

When root canal treatment fails to resolve periapical disease, it is often considered appropriate to retreat the tooth using conventional approaches first, especially when the previous treatment is technically deficient (Fig. 17.30). This requires removal of the previous root-filling material and any other material placed for restorative reasons. Correction of any procedural errors may also be required, if possible. All materials must be removed in their entirety to ensure delivery of antibacterial agents to all surfaces of the root canal dentin (see Fig. 17.30). The periapical healing rates of root canal retreatment are generally perceived to be lower compared to primary treatment for the following reasons:

- Obstructed access to the apical infection
- A potentially more resistant microbiota

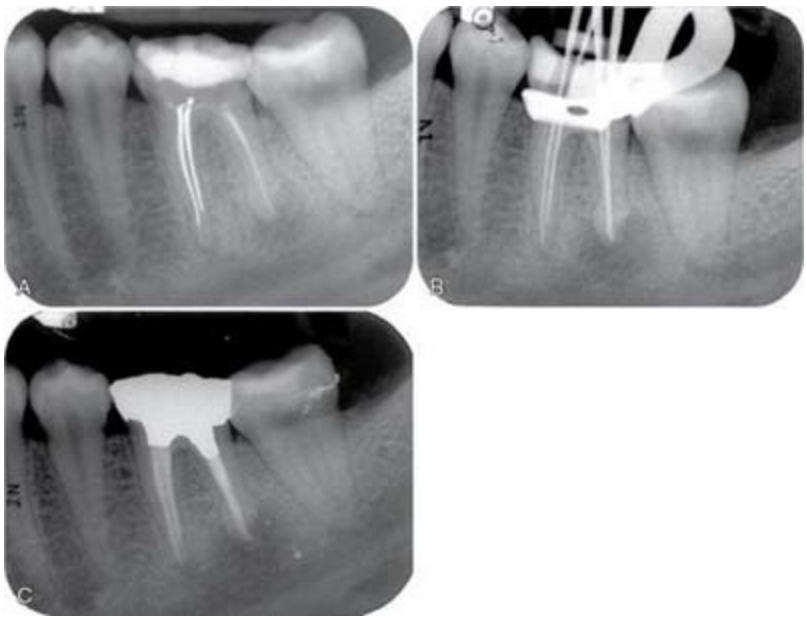

FIG. 17.30 Tooth with technically deficient root canal treatment **(A)**; having undergone (**B** and **C**) root canal retreatment.

Three radiographs are marked A through C.

A) Four teeth are shown. The third tooth has incomplete root canals. The

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crown has a radiopaque patch.

B) Third tooth is undergoing root canal treatment.

C) Third tooth has filled root canals and radiopaque crown.

The outcomes from a range of studies show that the mean weighted success rate of nonsurgical root canal retreatment is 66% (Figs. 17.31 and 17.32), which is about 6% lower than in the case of primary treatment on teeth with apical periodontitis.[148,150] However, it has also been shown that the survival rate of teeth having undergone nonsurgical root canal retreatment may be similar to that for primary root canal treatment.[151]

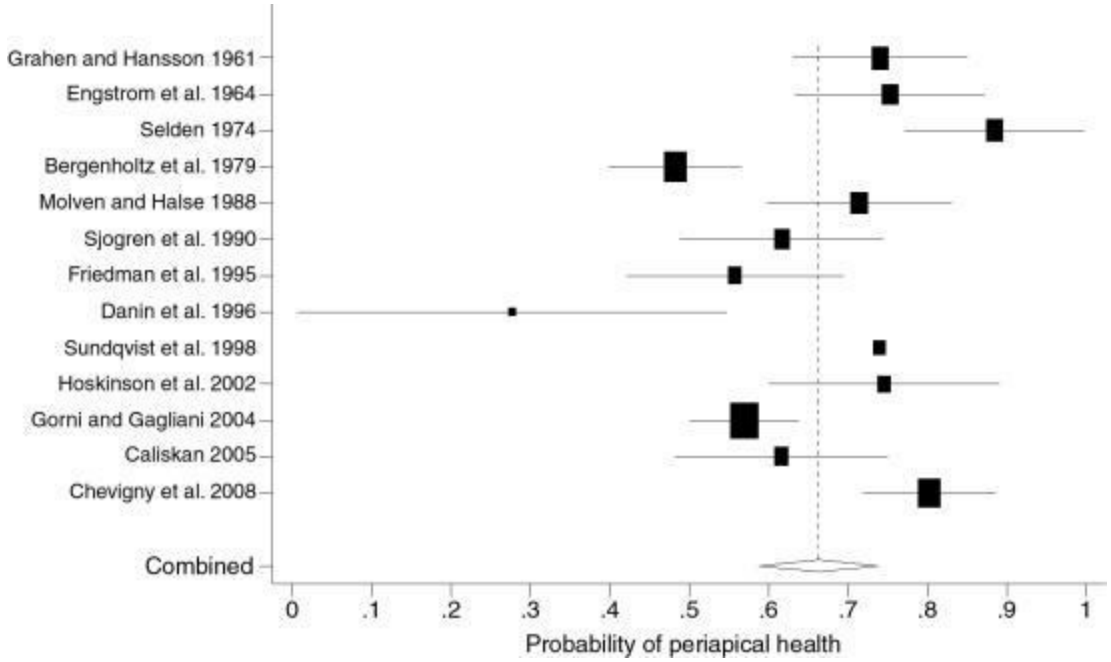

FIG. 17.31 Forest plot showing pooled and individual study's probability of complete periapical healing following root canal retreatment.

Forest plot shows combined and individual study against pr_success_pa.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The approximate data are as follows: Combined: 0.5 9 to 0. 7 3, Chevigny et al. 2008: 0.8, Caliskan 2005: 0.6 1, Gorni and Gagliani 2004: 0.5 8, Hoskinson et al. 2002: 0.7 2, Sundqvist et al. 1998: 0.7 2, Danin et al. 1996: 0.2 8, Friedman et al. 1995: 0.5 7, Sjogren et al. 1990: 0.6, Molven and Halse 1988: 0.7, Bergenholtz et al. 1979: 0.4 9, Selden 1974: 0.8 9, Engstrom et al. 1964: 0.7 8, and Grahen and Hansson 1961: 0.7 7.

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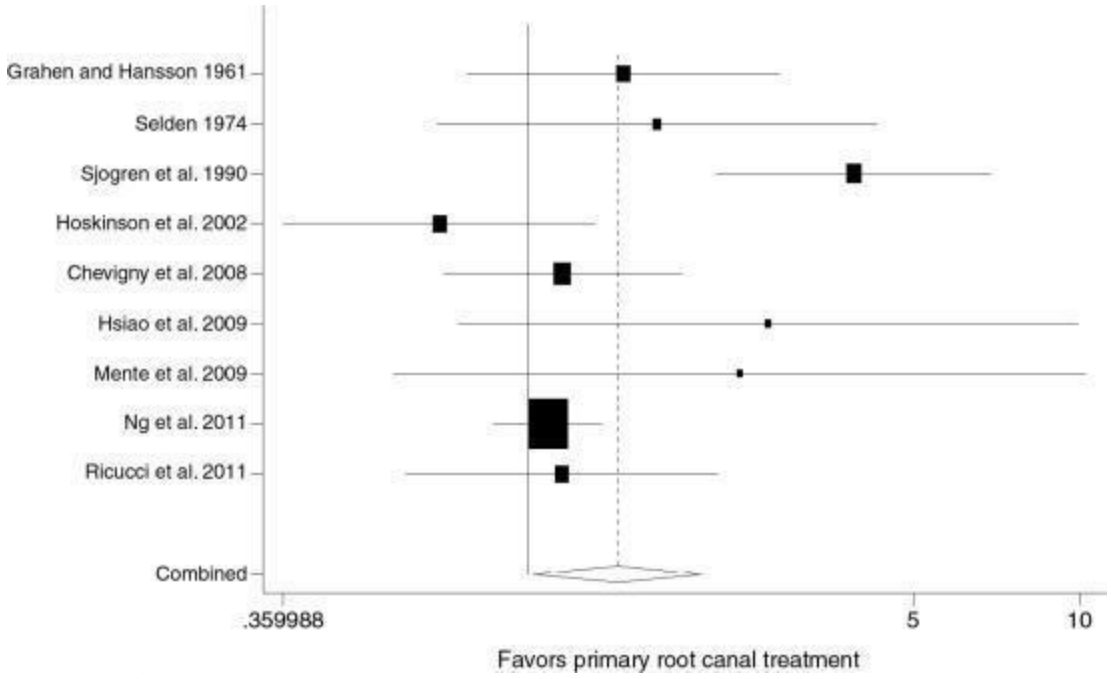

FIG. 17.32 Forest plot showing pooled and individual study's odds ratio of periapical health for teeth with preoperative periapical radiolucency undergoing primary root canal treatment versus root canal retreatment (pooled OR = 1.5; 95% CI: 1.0, 2.1).

Forest plot shows combined and individual study against Logor_pa1st_pa2nd.

The horizontal axis is marked at 0.3 5 9 9 8 8, 5, and 10. The vertical axis is marked from bottom to top as follows: Combined, Ricucci et al. 2011, Ng et al. 2011, Mente et al. 2009, Hsiao et al. 2009, Chevigny et al. 2008, Hoskinson et al. 2002, Sjogren et al. 1990, Selden 1974, and Grahen and Hansson 1961. The data points for all studies lie in between one-fourth length of the horizontal axis to the point 5.

The factors affecting outcomes of periapical health and tooth survival following root canal retreatment are otherwise identical to those affecting primary root canal treatment. Of the potential prognostic factors unique to retreatment cases, the most significant factor influencing the outcome of treatment is the ability to remove or bypass preexisting root-filling material or separated instruments during retreatment. This is understandable because it would have a direct impact on the ability to achieve canal patency and canal disinfection at the apical terminus.[150]

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Outcome of surgical retreatment

Factors affecting periapical health or healing following periapical surgery and root-end filling

There are a number of published systematic reviews on the prognostic factors for periapical surgery with apical filling.[45,47,48,155,204,205,239-241,250] The major limitations of this pooled data include the variable length of time for evaluating success following treatment as well as the radiographic criteria used to assess healing. An unpublished meta-analysis[130] of data from the studies listed in Table 17.11 reveals that the weighted pooled probability of success from periapical surgery with retrograde restorations, based on complete radiographic healing, is 67.5% (95% CI: 62.9%, 72.0%) (Fig. 17.33). The trend shows higher success rates reported in more recent studies. This observation is consistent with the much higher pooled success rate of 92% (95% CI: 86%, 95%) revealed in the meta-analysis of prospective outcome data of surgical endodontic treatments performed by a “modern” technique (using magnification, root-end resection with minimal or no bevel, retrograde cavity preparation with ultrasonic tips, and modern retrograde root canal filling materials).[240,241] In congruence with these findings, Setzer and colleagues reported that the pooled success rate of treatment using a microsurgical approach (94%; 95% CI: 89%, 98%) was more favorable than traditional root-end surgery (59%; 95% CI: 55%, 63%).[205] However, the studies included in the latter meta-analysis differed in design, case selection, duration after treatment, and the provision of preoperative nonsurgical treatment, which may bias the higher success rate of the contemporary treatment.

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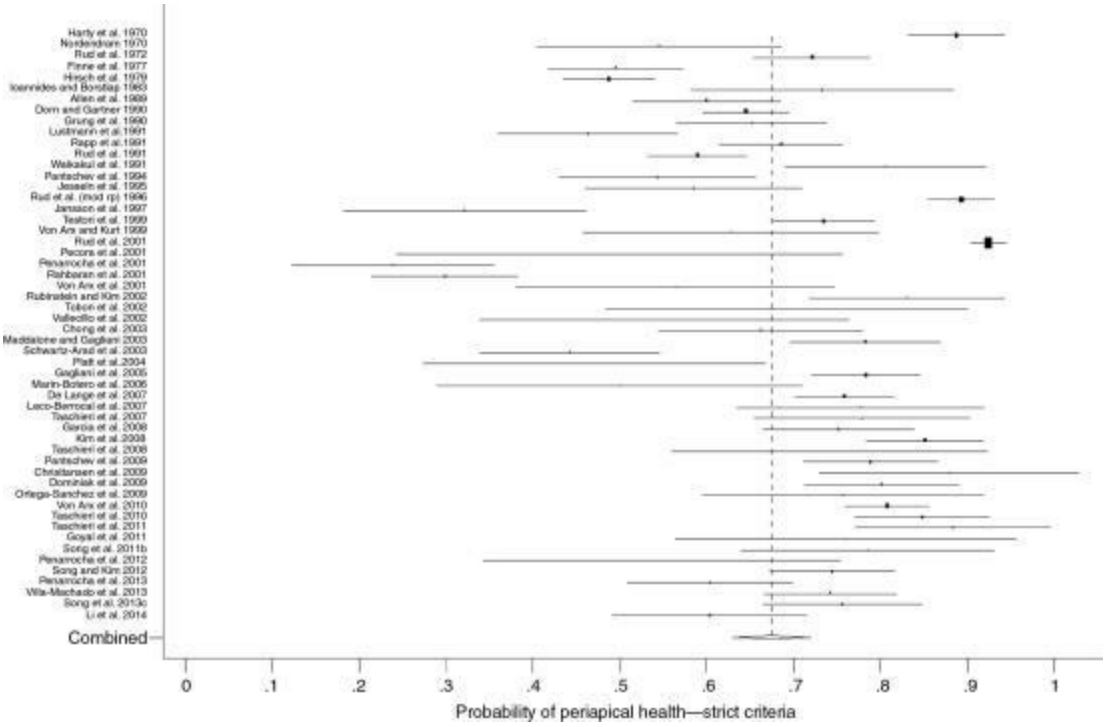

FIG. 17.33 Forest plot showing pooled and individual study's probability of complete periapical healing following apical surgery.

Forest plot shows combined and individual study against Strict_s_pr.

The horizontal axis ranges from 0 (near origin) to 1 in the increments of 0.1. The vertical axis is marked from bottom to top as follows: Combined, Li et al. 2014, Song et al. 2013c, Villa-Machado et al. 2013, Penarrocha et al. 2013, Song and Kim 2012, Penarrocha et al. 2012, Song et al. 2011b, Goyal et al. 2011, Taschieri et al. 2011, Taschieri et al. 2010, Von Arx et al. 2010, Ortega-Sanchez et al. 2009, Dominiak et al. 2009, Christiansen et al. 2009, Pantschev et al. 2009, Taschieri et al. 2008, Kim et al. 2008, Garcia et al. 2008, Taschieri et al. 2007, Leco-Berrocal et al. 2007, De Lange et al. 2007, Marin-Botero et al. 2006, Gagliani et al. 2005, Platt et al. 2004, Schwartz-Arad et al. 2003, Maddalone and Gagliani 2003, Chong et al. 2003, Vallecillo et al. 2002, Tobon et al. 2002, Rubinstein and Kim 2002, Von Arx et al. 2001, Rahbaran et al. 2001, Penarrocha et al. 2001, Pecora et al. 2001, Rud et al. 2001, Von Arx and Kurt 1999, Testori et al. 1999, Jansson et al. 1997, Rud et al. (mod rp) 1996, Jesseln et al. 1995, Pantschev et al. 1994, Waikakul et al. 1991, Rud et al. 1991, Rapp et al.1991, Lustmann et al.1991, Grung et al. 1990, Dorn and Gartner 1990, Allen et al. 1989, Ioannides and Borstlap 1983, Hirsch et al. 1979, Finne et al. 1977, Nordendram 1970, and Harty et al. 1970. The data points for all studied lie in-between 0.1 and 0. 9 5.

Table 17.11

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Studies Investigating Periapical Healing Following Apical Surgery

					SAMPLE SIZE			
Author	**Design**	**Examination**	**Type of Radiograph**	**Radiographic Criteria for Success**	**Patients**	**Teeth**	**Roots**	**Duration After Treatment (Months)**
Harty et al., 1970	Retrospective	C & R	Pa	Other		169		6–60
Nordendram, 1970	Prospective	C & R	Pa	Rud et al., 1972	66	66		6–24
Rud et al., 1972	Prospective	R	Pa	Rud et al., 1972		237		12–180
Finne et al., 1977	Prospective	C & R	Pa	Persson, 1973	156	218		36
Hirsch et al., 1979	Prospective	C & R	Pa	Rud et al., 1972	467	467		6–36
Ioannides and Borstlap, 1983	Retrospective	C & R	Pa	Other	50	50	45	6–60
Allen et al., 1989	Retrospective	C & R	Pa	Rud et al., 1972		175		12–60
Amagasa et al., 1989	Prospective	C & R	Pa	Other	42	64		12–90
Dorn and Gartner, 1990	Retrospective	R	Pa	Other		488		6–120
Grung et al., 1990	Prospective	C & R	Pa	Rud et al., 1972		161		12–96
Lustmann et al., 1991	Retrospective	C & R	Pa	Rud et al., 1972			123	6–96
Rapp et al., 1991	Retrospective	R	Pa	Rud et al., 1972	331	226		6–24
Rud et al., 1991	Retrospective	C & R	Pa	Rud et al., 1972	388	388		12

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Waikakul et al., 1991	Prospective	C & R	Pa	Other	34	62		6–24
Pantschev et al., 1994	Prospective	C & R	Pa	Persson, 1973	79	103		36
Jesselen et al., 1995	Prospective	C & R	Pa	Other	67	82		12–60
Rud et al., 1996	Prospective	R	Pa	Rud et al., 1972			347	12–48
Sumi et al., 1996	Retrospective	C & R	Pa	Other	86	157		6–36
Jansson et al., 1997	Retrospective	C & R	Pa	Other	59	59		11–16
Testori et al., 1999	Retrospective	R	Pa	Rud et al., 1972	130	181	302	12–72
Von Arx and Kurt, 1999	Prospective	C & R	Pa	Von Arx and Kurt, 1999	38	43		12
Zuolo et al., 2000	Prospective	C & R	Pa	Molven et al., 1987	106	102		12–48
Pecora et al., 2001	RCT	R	Pa	Rud et al., 1972	20	20		6
Penarrocha et al., 2001	Retrospective	R	Pa	Von Arx and Kurt, 1999	30	31	71	12
Rahbaran et al., 2001	Retrospective	C & R	Pa	Other	154	154		48–108
Rud et al., 2001	Prospective	C & R	Pa	Rud et al., 1972		520	834	6–150
Von Arx et al., 2001	Prospective	C & R	Pa	Other	24	25	39	12
Jensen et al., 2002	RCT	C & R	Pa	Rud et al., 1972		122		
Rubinstein and Kim, 2002	Prospective	C & R	Pa	Rud et al., 1972	52	59	59	68
Tobon et al., 2002	RCT	R	Pa	Rud et al., 1972	25	26		12

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Vallecillo et al., 2002	Prospective	R	Pa	Other	29	29		12
Chong et al., 2003	RCT	C & R	Pa	Molven et al., 1987	86	86		12–24
Maddalone and Gagliani, 2003	Prospective	C & R	Pa	Molven et al., 1987	79	120		3–36
Schwartz-Arad et al., 2003	Prospective	R	Pa	Other	101	122		6–45
Platt et al., 2004	RCT	C & R	Pa	Molven et al., 1987	28	34		12
Gagliani et al., 2005	Prospective	C & R	Pa	Rud et al., 1972	164	168	231	60
Lindeboom et al., 2005	RCT	C & R	Pa	Rud et al., 1972	100	100		12
Marti-Bowen et al., 2005	Retrospective	C & R	DPT	Von Arx and Kurt, 1999	52	71	95	6–12
Taschieri et al., 2005	Prospective	C & R	Pa	Rud et al., 1972	32	46		12
Taschieri et al., 2007	Prospective	C & R	Pa	Molven et al., 1987	28	28		12
Tsesis et al., 2006	Retrospective	C & R	Pa	Rud et al., 1972	71	88		6–48
Marin-Botero et al., 2006	RCT	C & R	Pa	Rud et al., 1972	30	30		12
Taschieri et al., 2006	RCT	C & R	Pa	Molven et al., 1987	53	71		12
De Lange et al., 2007	RCT	C & R	Pa	Rud et al., 1972	290	290		12
Leco-Berrocal et al., 2007	Prospective	R	Pa and DPT	Other	45	45		6–24

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Penarrocha et al., 2007	Prospective	C & R	Pa	Von Arx and Kurt, 1999	235	333	384	6–144
Taschieri et al., 2007a	Prospective	C & R	Pa	Molven et al., 1987	17	27		12
Taschieri et al., 2007b	Prospective	C & R	Pa	Molven et al., 1987	41	59		12
Von Arx et al., 2007	Prospective	C & R	Pa	Rud et al., 1972	200	177		12
Walivaara et al., 2007	Prospective	C & R	Pa	Rud et al., 1972	54	55		12
Garcia et al., 2008	Prospective	C & R	DPT	Von Arx and Kurt, 1999	92	106	129	6–12
Kim et al., 2008	Prospective	C & R	Pa	Molven et al., 1987		148		24
Penarrocha et al., 2008	Prospective	C & R	DPT	Von Arx and Kurt, 1999	278	278		12
Taschieri et al., 2008b	Prospective	C & R	Pa	Molven et al., 1987	27	31		12
Taschieri et al., 2008a	RCT	C & R	Pa	Molven et al., 1987	61	100		25
Christiansen et al., 2009	RCT	C & R	Pa	Molven et al., 1987		25		12
Dominiak et al., 2009	Retrospective	C & R	Pa	Other	106	106		12
Ortega-Sanchez et al., 2009	Retrospective	C & R	DPT	Von Arx and Kurt, 1999	30	30	37	
Pantschev et al., 2009	Retrospective	C & R	Pa	Other		147		12
Waalivaara et al., 2009	RCT	C & R	Pa	Molven et al., 1987	131	147		12
Barone et al., 2010	Prospective	C & R	Pa	PAI		129		48–120
Garcia-Mira et al., 2010	Retrospective	C & R	DPT	Von Arx and Kurt, 1999	75	87		12

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Taschieri et al., 2010	Retrospective	C & R	Pa	Molven et al., 1987	76	112		48
Von Arx et al., 2010	Prospective	C & R	Pa	Rud et al., 1972		339		12
Goyal et al., 2011	RCT	C & R	Pa	Rud et al., 1972	25	25		12
Penarrocha et al., 2011	Retrospective	C & R	DPT	Von Arx and Kurt, 1999	150	178	178	12
Song et al., 2011a	Retrospective	C & R	Pa	Molven et al., 1987		441		3–12
Song et al., 2011b	Prospective	C & R	Pa	Molven et al., 1987	42	42		12
Taschieri et al., 2011	Retrospective	C & R	Pa	Molven et al., 1987	33	43		12–48
Waalivaara et al., 2011	RCT	C & R	Pa	Molven et al., 1987	153	194		12–21
Penarrocha et al., 2012	Prospective	C & R	DPT	Von Arx and Kurt, 1999	23	31		12–19
Song and Kim, 2012	RCT	C & R	Pa	Molven et al., 1987	192	192		12
Von Arx et al., 2012	Prospective	C & R	Pa	Rud et al., 1972		170		12–60
Kreisler et al., 2013	Prospective	C & R	Pa	Rud et al., 1972	255	281		6–12
Penarrocha et al., 2013	Retrospective	C & R	Pa and DPT	Von Arx and Kurt	96	139		6–12
Song et al., 2013a	Prospective	C & R	Pa	Molven et al., 1987		344		12–120
Song et al., 2013b	Prospective	C & R	Pa	Molven et al., 1987		135		12
Song et al., 2014	Retrospective	C & R	Pa	Rud et al., 1972		115		12–96
Taschieri et al., 2013	Retrospective	C & R	Pa	Molven et al., 1987		86		6–12
Villa-Machado et al., 2013	Retrospective	C & R	Pa	PAI	154	171		12–192
Li et al., 2014	Retrospective	C & R	Pa	Molven et al., 1987	82	101		48

C, Clinical; *DPT*, dental pantomogram; *Pa*, periapical; *PAI*, periapical index; *R*, radiographic; *RCT*, randomized controlled trial.

The unpublished meta-analysis[130] stratified by duration following treatment reveals that the pooled probability of success based on complete healing plateaued after 2 years postoperatively, being 51% (95% CI: 42%, 60%) after 6 months, 68% (95% CI: 63%, 73%) after 12 months, 76% (95% CI: 67%, 84%) after 24 months, 74% (95% CI: 52%, 95%) after 48 months,

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and 74% (95% CI: 66%, 82%) after more than 48 months. It is therefore advisable to follow up on cases that have undergone periapical surgery for a minimum period of 2 years and up to 4 years, as suggested in quality guidelines.[61]

The factors having a major impact on outcome of periapical surgery with retrograde cavities and fillings in teeth with preoperative periapical radiolucencies revealed in the (as yet) unpublished meta-analyses[130] of data from studies listed in Table 17.11 are as follows:

- Small (≤5 mm) versus large (>5 mm) periapical lesion (risk ratio = 1.2; 95% CI: 1.1, 1.3)
- Periapical lesion involving one versus both cortical plates (risk ratio = 1.2; 95% CI: 1.0, 1.5)
- Absence versus presence of previous surgery (risk ratio = 1.2; 95% CI: 1.1, 1.3)
- Using magnification versus without the use of magnification during surgery (risk ratio = 1.5; 95% CI: 1.3, 1.8)
- Root-end resection with minimum versus obvious bevel (risk ratio = 1.3; 95% CI: 1.2, 1.4)
- Use of ultrasonic tip versus bur for retro-cavity preparation (risk ratio = 1.3; 95% CI: 1.2, 1.4)
- Use of retro-filling material with mineral trioxide aggregate (MTA) cement, super ethoxybenzoic acid (EBA) cement, or intermediate restorative material (IRM) versus amalgam; the use of MTA resulted in a similar outcome to SuperEBA (risk ratio = 1.0; 95% CI: 0.99, 1.1) or IRM (risk ratio = 1.1; 95% CI: 0.98, 1.1); SuperEBA and IRM were associated with significantly higher chances of success than amalgam (risk ratio = 1.2; 95% CI: 1.1, 1.3) as the retro-filling material

The preoperative presence of signs or symptoms (risk ratio = 1.2; 95% CI: 1.1, 1.3),[249] periodontal status (risk ratio = 2.1; 95% CI: 1.1, 3.8),[222] and quality of the coronal restoration (risk ratio = 1.6; 95% CI: 1.2, 2.1)[249] have also been revealed as significant prognostic factors by individual studies.

The following factors have minimal effect on surgical retreatment[43]:

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- Age of patient
- Gender of patient
- General health of patient
- Tooth type
- Quality of the preexisting root canal filling, as judged radiographically
- Histologic diagnosis of the biopsied periapical lesion (cyst or granuloma)

The use of guided periodontal tissue regenerative membrane or grafting material has been advocated for cases with through-and-through defects (missing buccal and palatal cortical plates), but there are conflicting reports as to their benefits.[53,162,170,232-234,237,249] The unpublished meta-analyses[130] of data from these studies did not reveal a significant influence of such approaches on the periapical healing outcome.

Factors affecting periodontal incisional wound healing

Periodontal attachment level after periapical surgery is an additional physical outcome measure compared to nonsurgical root canal treatment. Studies have compared the effect of different soft-tissue incision techniques (e.g., intrasulcular with or without involving interproximal papilla, submarginal, papilla-base).[106,245-247] As expected, all concluded that negligible marginal recession could be achieved by adopting a flap design that avoided reflection of the interproximal papilla.

The improvement in outcomes of periapical surgery has been attributed to modern surgical techniques plus greater biologic awareness of clinicians.[205,250] In addition, and perhaps even more importantly, case selection may be more critical in excluding potential failures, inferring that prognostication may have improved.

Factors affecting tooth survival following periapical surgery and root-end filling

Unlike nonsurgical root canal treatment, the outcome measure of tooth survival had only been adopted by one study[253] on surgical cases at the time of this writing. The reported median survival time for first-time surgery was

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92.1 months (95% CI: 40.9, 143.4) and that for resurgery was 39.1 months (95% CI: 6.1, 72.1).[253] The failure events, however, included tooth extraction and clinical and radiographic signs of periapical disease after treatment.

Impact of periapical surgery on quality of life

The impact of periapical surgery on the patient's quality of life has only been evaluated using a questionnaire including three domains: physical function (chewing, talking, sleeping, daily routine, and work), physical pain, and other physical symptoms (swelling, bleeding, nausea, bad taste/breath).[49] It was concluded that the papilla-base incision flap design resulted in a lower impact on physical pain and other symptoms within the first week postoperation. The impact on physical pain has also been explored by two other studies[35,36]; both reported the postoperative pain was of relatively short duration, with the intensity peaking at 3 to 5 hours postoperatively and progressively decreasing with time with no significant influencing factors identified.

Concluding remarks

The procedures used to maintain pulp vitality and for prevention and treatment of periapical disease are able to achieve excellent outcomes. The outcome data and potential prognostic factors should be considered during treatment options appraisal and planning. Despite the fact that most important prognostic factors are beyond the control of clinicians, optimal outcomes for individual cases may still be achieved by performing the procedure to guideline standards. From a health and economic perspective, conventional root canal treatment is a highly cost-effective treatment as a first-line intervention to extend the life of the tooth with a preoperative periapical lesion.[103,171] If a root canal treatment subsequently fails, nonsurgical and surgical retreatments are also more cost-effective than replacement with a prosthesis.[103,171] Ultimately, all sources of evidence must be assessed for biasing influences based on the local cultures, expertise, treatment predilection, and funding sources.

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References

1. Aguilar P, Linsuwanont P. *Vital pulp therapy in vital permanent teeth with cariously exposed pulp: a systematic review*, J Endod 2011;37: 581.
2. Akpata ES. *Effect of endodontic procedures on the population of viable microorganisms in the infected root canal*, J Endod 1976;2: 369.
3. Alley BS, Kitchens GG, Alley LW, et al: *A comparison of survival of teeth following endodontic treatment performed by general dentists or by specialists*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 115.
4. Allison DA, Weber CR, Walton RE. *The influence of the method of canal preparation on the quality of apical and coronal obturation*, J Endod 1979;5: 298.
5. Arai Y, Honda K, Iwai K, et al: *Practical model "3DX" of limited cone-beam X-ray CT for dental use*, Comput Assist Radiol Surg 2001;1230: 671.
6. Asgary S, Ehsani S. *Permanent molar pulpotomy with a new endodontic cement: a case series*, J Conserv Dent 2009;12: 31.
7. Auerbach M. *Antibiotics vs. instrumentation in endodontics*, N Y State Dent J 1953;19: 225.
8. Augsburger RA, Peters DD. *Radiographic evaluation of extruded obturation materials*, J Endod 1990;16: 492.
9. Bader JD, Shugars DA. *Variation, treatment outcomes, and practice guidelines in dental practice*, J Dent Educ 1995;59: 61.
10. Baratieri LN, Monteiro S Jr, Caldeira de Andrada MA. *Pulp curettage: surgical technique*, Quintessence Int 1989;20: 285.

Telegram: @dental_k

www.konkur.in

11. Barrieshi-Nusair KM, Qudeimat MA. *A prospective clinical study of mineral trioxide aggregate for partial pulpotomy in cariously exposed permanent teeth*, J Endod 2006;32: 731.
12. Barthel CR, Rosenkranz B, Leuenberg A, et al: *Pulp capping of carious exposures: treatment outcome after 5 and 10 years: a retrospective study*, J Endod 2000;26: 525.
13. Basrani B, Tjaderhane L, Santos JM, et al: *Efficacy of chlorhexidine- and calcium hydroxide-containing medicaments against Enterococcus faecalis in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 618.
14. Basrani BR, Manek S, Sodhi RN, et al: *Interaction between sodium hypochlorite and chlorhexidine gluconate*, J Endod 2007;33: 966.
15. Baugh D, Wallace J. *The role of apical instrumentation in root canal treatment: a review of the literature*, J Endod 2005;31: 333.
16. Bence R, Madonia JV, Weine FS, et al: *A microbiologic evaluation of endodontic instrumentation in pulpless teeth*, Oral Surg Oral Med Oral Pathol 1973;35: 676.
17. Bender IB, Seltzer S, Soltanof W. *Endodontic success: a reappraisal of criteria, 1*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1966;22: 780.
18. Bender IB, Seltzer S, Soltanof W. *Endodontic success: a reappraisal of criteria. 2*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1966;22: 790.
19. Bjorndal L, Reit C, Bruun G, et al: *Treatment of deep caries lesions in adults: randomized clinical trials comparing stepwise vs. direct complete excavation, and direct pulp capping vs. partial pulpotomy*, Eur J Oral Sci 2010;118: 290.

Telegram: @dental_k

www.konkur.in

20. Bjorndal L, Thylstrup A. *A practice-based study on stepwise excavation of deep carious lesions in permanent teeth: a 1-year follow-up study* Community Dent Oral Epidemiol 1998;26: 122.
21. Bogen G, Kim JS, Bakland LK. *Direct pulp capping with mineral trioxide aggregate: an observational study*, J Am Dent Assoc 2008;139: 305- quiz 305.
22. Boggia R. *A single-visit treatment of septic root canals using periapically extruded endomethasone*, Br Dent J 1983;155: 300.
23. Buchbinder M, Wald A. *An improved method of culturing root canals*, J Am Dent Assoc 1939;26: 1697.
24. Bui TB, Baumgartner JC, Mitchell JC. *Evaluation of the interaction between sodium hypochlorite and chlorhexidine gluconate and its effect on root dentin*, J Endod 2008;34: 181.
25. Byström A. Evaluation of endodontic treatment of teeth with apical periodontitis 1986; University of Umeå Sweden.
26. Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 1985;1: 170.
27. Bystrom A, Sundqvist G. *Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy*, Scand J Dent Res 1981;89: 321.
28. Bystrom A, Sundqvist G. *Bacteriologic evaluation of the effect of 0.5 percent sodium hypochlorite in endodontic therapy*, Oral Surg Oral Med Oral Pathol 1983;55: 307.
29. Bystrom A, Sundqvist G. *The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic*

Telegram: @dental_k

www.konkur.in

therapy, Int Endod J 1985;18: 35.
30. Caliskan MK. *Success of pulpotomy in the management of hyperplastic pulpitis*, Int Endod J 1993;26: 142.
31. Caliskan MK. *Pulpotomy of carious vital teeth with periapical involvement*, Int Endod J 1995;28: 172.
32. Camps J, Pommel L, Bukiet F. *Evaluation of periapical lesion healing by correction of gray values*, J Endod 2004;30: 762.
33. Card SJ, Sigurdsson A, Orstavik D, et al: *The effectiveness of increased apical enlargement in reducing intracanal bacteria*, J Endod 2002;28: 779.
34. Chambers D. *Outcomes-based practice: how outcomes-based practices get better*, Dental Economics 2001;3: 34.
35. Chong BS, Pitt Ford TR. *Postoperative pain after root-end resection and filling*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 762.
36. Christiansen R, Kirkevang LL, Horsted-Bindslev P, et al: *Patient discomfort following periapical surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: 245.
37. Chu FC, Leung WK, Tsang PC, et al: *Identification of cultivable microorganisms from root canals with apical periodontitis following two-visit endodontic treatment with antibiotics/steroid or calcium hydroxide dressings*, J Endod 2006;32: 17.
38. Chugal NM, Clive JM, Spangberg LS. *Endodontic treatment outcome: effect of the permanent restoration*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 576.
39. Colman I, Friedman BW, Brown MD, et al: *Parenteral dexamethasone for acute severe migraine headache: meta-analysis of randomised controlled trials for preventing recurrence*, BMJ 2008;336: 13591.

Telegram: @dental_k

www.konkur.in

40. Cvek M. *A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture*, J Endod 1978;4: 232.
41. Cvek M, Hollender L, Nord CE. *Treatment of non-vital permanent incisors with calcium hydroxide. VI. A clinical, microbiological and radiological evaluation of treatment in one sitting of teeth with mature or immature root*, Odontol Revy 1976;27: 93.
42. Dalton BC, Orstavik D, Phillips C, et al: *Bacterial reduction with nickel-titanium rotary instrumentation*, J Endod 1998;24: 763.
43. de Chevigny C, Dao TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study–phase 4: initial treatment*, J Endod 2008;34: 258.
44. de Paula-Silva FW, Wu MK, Leonardo MR, et al: *Accuracy of periapical radiography and cone-beam computed tomography scans in diagnosing apical periodontitis using histopathological findings as a gold standard*, J Endod 2009;35: 10092.
45. Del Fabbro M, Taschieri S. *Endodontic therapy using magnification devices: a systematic review*, J Dent 2010;38: 269.
46. Del Fabbro M, Taschieri S, Lodi G, et al: *Magnification devices for endodontic therapy*, Cochrane Database Syst Rev 2009; CD005969.
47. Del Fabbro M, Taschieri S, Lodi G, et al: *Magnification devices for endodontic therapy*, Cochrane Database Syst Rev 2009; CD005969.
48. Del Fabbro M, Taschieri S, Testori T, et al: *Surgical versus non-surgical endodontic retreatment for periradicular lesions*, Cochrane Database Syst Rev 2007; CD005511.

Telegram: @dental_k

www.konkur.in

49. Del Fabbro M, Taschieri S, Weinstein R. *Quality of life after microscopic periradicular surgery using two different incision techniques: a randomized clinical study*, Int Endod J 2009;42: 360.
50. Delano EO, Tyndall D, Ludlow JB, et al: *Quantitative radiographic follow-up of apical surgery: a radiometric and histologic correlation*, J Endod 1998;24: 420.
51. DePalma MJ, Slipman CW. *Evidence-informed management of chronic low back pain with epidural steroid injections*, Spine J 2008;8: 45.
52. DeRosa TA. *A retrospective evaluation of pulpotomy as an alternative to extraction*, Gen Dent 2006;54: 37.
53. Dominiak M, Lysiak-Drwal K, Gedrange T, et al: *Efficacy of healing process of bone defects after apicectomy: results after 6 and 12 months*, J Physiol Pharmacol Suppl 8, 2009;60: 51.
54. Doyle SL, Hodges JS, Pesun IJ, et al: *Retrospective cross sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants*, J Endod 2006;32: 822.
55. Dugas NN, Lawrence HP, Teplitsky P, et al: *Quality of life and satisfaction outcomes of endodontic treatment*, J Endod 2002;28: 819.
56. Eakle WS, Maxwell EH, Braly BV. *Fractures of posterior teeth in adults*, J Am Dent Assoc 1986;112: 215.
57. Edwards JL, Vincent AM, Cheng HT, et al: *Diabetic neuropathy: mechanisms to management*, Pharmacol Ther 2008;120: 1.
58. El-Meligy OA, Avery DR. *Comparison of mineral trioxide aggregate and calcium hydroxide as pulpotomy agents in young permanent teeth (apexogenesis)*, Pediatr Dent 2006;28: 399.

Telegram: @dental_k

www.konkur.in

59. American Association of Endodotists: *Evolution of the root canal* www.aae.org/uploadedfiles/news_room/press_releases/rcav Accessed June 2015.
60. Engström B. *The significance of enterococci in root canal treatment*, Odontol Revy 1964;15: 87.
61. European Society of Endodontology. *Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology*, Int Endod J 2006;39: 921.
62. Patel S, Durack C.et al: European Society of Endodontology. *European Society of Endodontology position statement: the use of CBCT in endodontics*, Int Endod J 2014;47: 502.
63. Fabricius L, Dahlen G, Sundqvist G, et al: *Influence of residual bacteria on periapical tissue healing after chemomechanical treatment and root filling of experimentally infected monkey teeth*, Eur J Oral Sci 2006;114: 278.
64. Farsi N, Alamoudi N, Balto K, et al: *Clinical assessment of mineral trioxide aggregate (MTA) as direct pulp capping in young permanent teeth*, J Clin Pediatr Dent 2006;31: 72.
65. Fitzgerald M, Heys RJ. *A clinical and histological evaluation of conservative pulpal therapy in human teeth*, Oper Dent 1991;16: 101.
66. Ford TP. *Frequency of radiological review in pulp studies*, Int Endod J 2008;41: 931.
67. Fouad AF, Burleson J. *The effect of diabetes mellitus on endodontic treatment outcome: data from an electronic patient record*, J Am Dent Assoc 2003;134: 43- quiz 117.
68. Friedman S, Lost C, Zarrabian M, Trope M. *Evaluation of*

Telegram: @dental_k

www.konkur.in

success and failure after endodontic therapy using a glass ionomer cement sealer, J Endod 1995;21: 384.

69. Friedman S, Mor C. *The success of endodontic therapy—healing and functionality*, J Calif Dent Assoc 2004;32: 493.
70. Frostell G. *Clinical significance of the root canal culture*, Transactions of Third International Conference of Endodontics 1963; 112-122.
71. Fuks AB, Gavra S, Chosack A. *Long-term followup of traumatized incisors treated by partial pulpotomy*, Pediatr Dent 1993;15: 334.
72. Gallien GS Jr, Schuman NJ. *Local versus general anesthesia: a study of pulpal response in the treatment of cariously exposed teeth*, J Am Dent Assoc 1985;111: 599.
73. Gatten DL, Riedy CA, Hong SK, et al: *Quality of life of endodontically treated versus implant treated patients: a University-based qualitative research study*, J Endod 2011;37: 903.
74. Genco RJ, Loe H. *The role of systemic conditions and disorders in periodontal disease*, Periodontol 2000 1993;2: 98.
75. Gesi A, Hakeberg M, Warfvinge J, et al: *Incidence of periapical lesions and clinical symptoms after pulpectomy —a clinical and radiographic evaluation of 1- versus 2-session treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 379.
76. Goldfein J, Speirs C, Finkelman M, et al: *Rubber dam use during post placement influences the success of root canal-treated teeth*, J Endod 2013;39: 14814.
77. Gomes BP, Lilley JD, Drucker DB. *Variations in the susceptibilities of components of the endodontic microflora to biomechanical procedures*, Int Endod J 1996;29: 235.

Telegram: @dental_k

www.konkur.in

78. Gomes BP, Souza SF, Ferraz CC, et al: *Effectiveness of 2% chlorhexidine gel and calcium hydroxide against Enterococcus faecalis in bovine root dentine in vitro*, Int Endod J 2003;36: 267.
79. Grahnen H, Krasse B. *The effect of instrumentation and flushing of non-vital teeth in endodontic therapy*, Odontol Rev 1963;14: 167.
80. Gratt BM, Sickles EA, Gould RG, et al: *Xeroradiography of dental structures. IV. Image properties of a dedicated intraoral system*, Oral Surg Oral Med Oral Pathol 1980;50: 572.
81. Gruythuysen RJ, van Strijp AJ, et al: *Long-term survival of indirect pulp treatment performed in primary and permanent teeth with clinically diagnosed deep carious lesions*, J Endod 2010;36: 14903.
82. Gulabivala K. Species richness of gram-positive coccoid morphotypes isolated from untreated and treated root canals of teeth associated with periapical disease 2004; University of London London.
83. Gulabivala K, Ng Y. *Endodontology*, Wilson N Clinical dental medicine 2020 2009; Quintessence New Malden, Surrey 147-182.
84. Gulabivala K, Ng YL, Gilbertson M, et al: *The fluid mechanics of root canal irrigation*, Physiol Meas 2010;31: R49.
85. Gulabivala K, Patel B, Evans G, et al: *Effects of mechanical and chemical procedures on root canal surfaces*, Endodontic Topics 2005;10: 103.
86. Halse A, Molven O. *Increased width of the apical periodontal membrane space in endodontically treated teeth may represent favourable healing*, Int Endod J 2004;37: 552.

Telegram: @dental_k

www.konkur.in

87. Hamasha AA, Hatiwsh A. *Quality of life and satisfaction of patients after nonsurgical primary root canal treatment provided by undergraduate students, graduate students and endodontic specialists*, Int Endod J 2013;46: 11319.
88. Haskell EW, Stanley HR, Chellemi J, et al: *Direct pulp capping treatment: a long-term follow-up*, J Am Dent Assoc 1978;97: 607.
89. Hayashi M, Fujitani M, Yamaki C, et al: *Ways of enhancing pulp preservation by stepwise excavation—a systematic review*, J Dent 2011;39: 95.
90. Hilton TJ, Ferracane JL, Mancl L. *Northwest Practice-based Research Collaborative in Evidence-based Dentistry (NWP): Comparison of CaOH with MTA for direct pulp capping: a PBRN randomized clinical trial*, J Dent Res 2013;92: 16SS.
91. Holroyd J, Gulson A. The radiation protection implications of the use of cone beam computed tomography (CBCT) in dentistry: what you need to know 2009; Health Protection Agency London.
92. Horsted P, Sandergaard B, Thylstrup A, et al: *A retrospective study of direct pulp capping with calcium hydroxide compounds*, Endod Dent Traumatol 1985;1: 29.
93. Hoskinson SE, Ng YL, Hoskinson AE, et al: *A retrospective comparison of outcome of root canal treatment using two different protocols*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 705.
94. Hughes R. *Focal infection revisited*, Rheumatology 1994;33: 370.
95. Hülsmann M, Peters OA, Dummer PM. *Mechanical preparation of root canals: shaping goals, techniques and means*, Endod Topics 2005;10: 30.
96. Hunter W. *The role of sepsis and antisepsis in medicine*,

Telegram: @dental_k

www.konkur.in

Lancet 1911;1: 79.
97. Hunter W. *Chronic sepsis as a cause of mental disorder*, Br J Psychiatr 1927;73: 549.
98. Huumonen S, Lenander-Lumikari M, Sigurdsson A, et al: *Healing of apical periodontitis after endodontic treatment: a comparison between a silicone-based and a zinc oxide-eugenol-based sealer*, Int Endod J 2003;36: 296.
99. Ingle J, Zeldow B. *An evaluation of mechanical instrumentation and the negative culture in endodontic therapy*, J Am Dent Assoc 1958;57: 471.
100. Iqbal MK, Kim S. *For teeth requiring endodontic treatment, what are the differences in outcomes of restored endodontically treated teeth compared to implant-supported restorations?*, Int J Oral Maxillofac Implants 2006;22: 96.
101. Kalichman L, Hunter DJ. *Diagnosis and conservative management of degenerative lumbar spondylolisthesis*, Eur Spine J 2008;17: 327.
102. Kerekes K, Tronstad L. *Long-term results of endodontic treatment performed with a standardized technique*, J Endod 1979;5: 83.
103. Kim SG, Solomon C. *Cost-effectiveness of endodontic molar retreatment compared with fixed partial dentures and single-tooth implant alternatives*, J Endod 2011;37: 321.
104. Koontongkaew S, Silapichit R, Thaweboon B. *Clinical and laboratory assessments of camphorated monochlorophenol in endodontic therapy*, Oral Surg Oral Med Oral Pathol 1988;65: 757.
105. Koppang HS, Koppang R, Stolen SO. *Identification of common foreign material in postendodontic granulomas and cysts*, J Dent Assoc S Afr 1992;47: 210.

Telegram: @dental_k

www.konkur.in

106. Kreisler M, Gockel R, Schmidt I, et al: *Clinical evaluation of a modified marginal sulcular incision technique in endodontic surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: e22.
107. Kuruvilla JR, Kamath MP. *Antimicrobial activity of 2.5% sodium hypochlorite and 0.2% chlorhexidine gluconate separately and combined, as endodontic irrigants*, J Endod 1998;24: 472.
108. Kvist T, Molander A, Dahlen G, et al: *Microbiological evaluation of one- and two-visit endodontic treatment of teeth with apical periodontitis: a randomized, clinical trial*, J Endod 2004;30: 572.
109. Lagouvardos P, Sourai P, Douvitsas G. *Coronal fractures in posterior teeth*, Oper Dent 1989;14: 28.
110. Lana MA, Ribeiro-Sobrinho AP, Stehling R, et al: *Microorganisms isolated from root canals presenting necrotic pulp and their drug susceptibility in vitro*, Oral Microbiol Immunol 2001;16: 100.
111. Lazarski MP, Walker WA III, Flores CM, et al: *Epidemiological evaluation of the outcomes of nonsurgical root canal treatment in a large cohort of insured dental patients*, J Endod 2001;27: 791.
112. Lee SJ, Wu MK, Wesselink PR. *The efficacy of ultrasonic irrigation to remove artificially placed dentine debris from different-sized simulated plastic root canals*, Int Endod J 2004;37: 607.
113. Leksell E, Ridell K, Cvek M, et al: *Pulp exposure after stepwise versus direct complete excavation of deep carious lesions in young posterior permanent teeth*, Endod Dent Traumatol 1996;12: 192.
114. Liang YH, Li G, Wesselink PR, et al: *Endodontic outcome predictors identified with periapical radiographs*

Telegram: @dental_k

www.konkur.in

and cone-beam computed tomography scans, J Endod 2011;37: 326.

115. Liu P, McGrath C, Cheung GS. *Improvement in oral health-related quality of life after endodontic treatment: a prospective longitudinal study*, J Endod 2014;40: 805.
116. Locker D. *Concepts of health, disease and quality of life*, Slade GD Measuring oral health and quality of life 1997; University of North Carolina Chapel Hill, North Carolina 11-23.
117. Loe H. *Does chlorhexidine have a place in the prophylaxis of dental diseases?*, J Periodontal Res Suppl 1973;12: 93.
118. Lumley PJ, Lucarotti PS, Burke FJ. *Ten-year outcome of root fillings in the General Dental Services in England and Wales*, Int Endod J 2008;41: 577.
119. Mah JK, Danforth RA, Bumann A, et al: *Radiation absorbed in maxillofacial imaging with a new dental computed tomography device*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 508.
120. Maltz M. *Does incomplete caries removal increase restoration failure?*, J Dent Res 2011;90: 541- author reply 542.
121. Maltz M, Garcia R, Jardim JJ, et al: *Randomized trial of partial vs. stepwise caries removal: 3-year follow-up*, J Dent Res 2012;91: 10261.
122. Maltz M, Henz SL, de Oliveira EF, et al: *Conventional caries removal and sealed caries in permanent teeth: a microbiological evaluation*, J Dent 2012;40: 776.
123. Marending M, Peters OA, Zehnder M. *Factors affecting the outcome of orthograde root canal therapy in a general dentistry hospital practice*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 119.

Telegram: @dental_k

www.konkur.in

124. Markvart M, Dahlen G, Reit CE, et al: *The antimicrobial effect of apical box versus apical cone preparation using iodine potassium iodide as root canal dressing: a pilot study*, Acta Odontol Scand 2013;71: 786.
125. Mass E, Zilberman U. *Clinical and radiographic evaluation of partial pulpotomy in carious exposure of permanent molars*, Pediatr Dent 1993;15: 257.
126. Masterton JB. *The healing of wounds of the dental pulp: an investigation of the nature of the scar tissue and of the phenomena leading to its formation*, Dent Pract Dent Rec 1966;16: 325.
127. Matsumoto M, Goto T. *Lateral force distribution in partial denture design*, J Dent Res 1970;49: 359.
128. Matsuo T, Nakanishi T, Shimizu H, et al: *A clinical study of direct pulp capping applied to carious-exposed pulps*, J Endod 1996;22: 551.
129. McDonnell G, Russell AD. *Antiseptics and disinfectants: activity, action, and resistance*, Clinical Microbiol Rev 1999;12: 147.
130. Mehta D, Gulabivala K, Ng Y-L: Personal communication. Outcome of periapical surgery, unpublished manuscript, 2014.
131. Mejare I, Cvek M. *Partial pulpotomy in young permanent teeth with deep carious lesions*, Endod Dent Traumatol 1993;9: 238.
132. Mente J, Geletneky B, Ohle M, et al: *Mineral trioxide aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcome*, J Endod 2010;36: 806.
133. Miles JP, Gluskin AH, Chambers D, et al: *Pulp capping with mineral trioxide aggregate (MTA): a retrospective analysis of carious pulp exposures treated by*

Telegram: @dental_k

www.konkur.in

undergraduate dental students, Oper Dent 2010;35: 20.

134. Miller WD. *An introduction to the study of the bacterio-pathology of the dental pulp*, The Dental Cosmos 1894;36: 505-528.
135. Mindiola MJ, Mickel AK, Sami C, et al: *Endodontic treatment in an American Indian population: a 10-year retrospective study*, J Endod 2006;32: 828.
136. Miyashita H, Worthington HV, Qualtrough A, et al: *Pulp management for caries in adults: maintaining pulp vitality (review)*, Cochrane Database Syst Rev 2007.
137. Molander A, Reit C, Dahlen G. *Microbiological evaluation of clindamycin as a root canal dressing in teeth with apical periodontitis*, Int Endod J 1990;23: 113.
138. Molander A, Reit C, Dahlen G. *Microbiological root canal sampling: diffusion of a technology*, Int Endod J 1996;29: 163.
139. Molander A, Reit C, Dahlen G. *Reasons for dentists' acceptance or rejection of microbiological root canal sampling*, Int Endod J 1996;29: 168.
140. Molven O, Halse A, Grung B. *Observer strategy and the radiographic classification of healing after endodontic surgery*, Int J Oral Maxillofac Surg 1987;16: 432.
141. Moore DL. *Conservative treatment of teeth with vital pulps and periapical lesions: a preliminary report*, J Prosthet Dent 1967;18: 476.
142. Morsani JM, Aminoshariae A, Han YW, et al: *Genetic predisposition to persistent apical periodontitis*, J Endod 2011;37: 455.
143. Morse F, Yates M. *Follow up studies of root-filled teeth in relation to bacteriologic findings*, J Am Dent Assoc 1941;28: 956.
144. Nagasiri R, Chitmongkolsuk S. *Long-term survival of*

Telegram: @dental_k

www.konkur.in

endodontically treated molars without crown coverage: a retrospective cohort study, J Prosthet Dent 2005;93: 164.

145. Nair PN. *On the causes of persistent apical periodontitis: a review*, Int Endod J 2006;39: 249.
146. Nair PN, Sjogren U, Krey G, et al: *Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth*, J Endod 1990;16: 589.
147. Nair PNR, Henry S, Cano V, et al: *Microbial status of apical root canal system of human mandibular first molars with primary apical periodontitis after "one-visit" endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 231.
148. Ng YL, Mann V, Gulabivala K. *Outcome of secondary root canal treatment: a systematic review of the literature*, Int Endod J 2008;41: 10266.
149. Ng YL, Mann V, Gulabivala K. *Tooth survival following non-surgical root canal treatment: a systematic review of the literature*, Int Endod J 2010;43: 171.
150. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health*, Int Endod J 2011;44: 583.
151. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2: tooth survival*, Int Endod J 2011;44: 610.
152. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature. Part 1. Effects of study characteristics on probability of success*, Int Endod J 2007;40: 921.
153. Ng YL, Mann V, Rahbaran S, et al: *Outcome of primary root canal treatment: systematic review of the literature.*

Telegram: @dental_k

www.konkur.in

Part 2. Influence of clinical factors, Int Endod J 2008;41: 6.

154. Nicholls E. *The efficacy of cleansing of the root canal*, Br Dent J 1962;112: 167.
155. Niederman R, Theodosopoulou JN. *A systematic review of in vivo retrograde obturation materials*, Int Endod J 2003;36: 577.
156. Orstavik D. *Radiographic evaluation of apical periodontitis and endodontic treatment results: a computer approach*, Int Dent J 1991;41: 89.
157. Orstavik D. *Time-course and risk analyses of the development and healing of chronic apical periodontitis in man*, Int Endod J 1996;29: 150.
158. Orstavik D, Farrants G, Wahl T, et al: *Image analysis of endodontic radiographs: digital subtraction and quantitative densitometry*, Endod Dent Traumatol 1990;6: 6.
159. Orstavik D, Horsted-Bindslev P. *A comparison of endodontic treatment results at two dental schools*, Int Endod J 1993;26: 348.
160. Orstavik D, Kerekes K, Eriksen HM. *Clinical performance of three endodontic sealers*, Endod Dent Traumatol 1987;3: 178.
161. Orstavik D, Kerekes K, Molven O. *Effects of extensive apical reaming and calcium hydroxide dressing on bacterial infection during treatment of apical periodontitis: a pilot study*, Int Endod J 1991;24: 1.
162. Pantchev A, Nohlert E, Tegelberg A. *Endodontic surgery with and without inserts of bioactive glass PerioGlas: a clinical and radiographic follow-up*, Oral Maxillofac Surg 2009;13: 21.
163. Paquette L, Legner M, Fillery ED, et al: *Antibacterial*

Telegram: @dental_k

www.konkur.in

efficacy of chlorhexidine gluconate intracanal medication in vivo, J Endod 2007;33: 788.

164. Parris J, Wilcox L, Walton R. *Effectiveness of apical clearing: histological and radiographical evaluation*, J Endod 1994;20: 219.
165. Patel S, Dawood A, Mannocci F, et al: *Detection of periapical bone defects in human jaws using cone beam computed tomography and intraoral radiography*, Int Endod J 2009;42: 507.
166. Patel S, Rhodes J. *A practical guide to endodontic access cavity preparation in molar teeth*, Br Dent J 2007;203: 133.
167. Patel S, Wilson R, Dawood A, et al: *The detection of periapical pathosis using digital periapical radiography and cone beam computed tomography—part 2: a 1-year post-treatment follow-up*, Int Endod J 2012;45: 711.
168. Peciuliene V, Balciuniene I, Eriksen HM, et al: *Isolation of Enterococcus faecalis in previously root-filled canals in a Lithuanian population*, J Endod 2000;26: 593.
169. Peciuliene V, Reynaud AH, Balciuniene I, et al: *Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis*, Int Endod J 2001;34: 429.
170. Pecora G, De Leonardis D, Ibrahim N, et al: *The use of calcium sulphate in the surgical treatment of a 'through and through' periradicular lesion*, Int Endod J 2001;34: 189.
171. Pennington MW, Vernazza CR, Shackley P, et al: *Evaluation of the cost-effectiveness of root canal treatment using conventional approaches versus replacement with an implant*, Int Endod J 2009;42: 874.
172. Percinoto C, de Castro AM, Pinto LM. *Clinical and radiographic evaluation of pulpotomies employing*

Telegram: @dental_k

www.konkur.in

calcium hydroxide and trioxide mineral aggregate, Gen Dent 2006;54: 258.

173. Peters LB, van Winkelhoff AJ, Buijs JF, et al: *Effects of instrumentation, irrigation and dressing with calcium hydroxide on infection in pulpless teeth with periapical bone lesions*, Int Endod J 2002;35: 13.
174. Petersson A, Axelsson S, Davidson T, et al: *Radiological diagnosis of periapical bone tissue lesions in endodontics: a systematic review*, Int Endod J 2012;45: 783.
175. Pettiette MT, Delano EO, Trope M. *Evaluation of success rate of endodontic treatment performed by students with stainless-steel K-files and nickel-titanium hand files*, J Endod 2001;27: 124.
176. Polycarpou N, Ng YL, Canavan D, et al: *Prevalence of persistent pain after endodontic treatment and factors affecting its occurrence in cases with complete radiographic healing*, Int Endod J 2005;38: 169.
177. Qudeimat MA, Barrieshi-Nusair KM, Owais AI. *Calcium hydroxide vs mineral trioxide aggregates for partial pulpotomy of permanent molars with deep caries*, Eur Arch Paediatr Dent 2007;8: 99.
178. Reeh ES, Messer HH, Douglas WH. *Reduction in tooth stiffness as a result of endodontic and restorative procedures*, J Endod 1989;15: 512.
179. Reit C, Dahlen G. *Decision making analysis of endodontic treatment strategies in teeth with apical periodontitis*, Int Endod J 1988;21: 291.
180. Reit C, Grondahl HG. *Endodontic retreatment decision making among a group of general practitioners*, Scand J Dent Res 1988;96: 112.
181. Reit C, Molander A, Dahlen G. *The diagnostic accuracy of microbiologic root canal sampling and the influence of*

Telegram: @dental_k

www.konkur.in

antimicrobial dressings, Endod Dent Traumatol 1999;15: 278.

182. Ricucci D, Russo J, Rutberg M, et al: *A prospective cohort study of endodontic treatments of 1,369 root canals: results after 5 years*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 825.
183. Rocas IN, Siqueira JF Jr, Del Aguila CA, et al: *Polymorphism of the CD14 and TLR4 genes and post-treatment apical periodontitis*, J Endod 2014;40: 168.
184. Rollison S, Barnett F, Stevens RH. *Efficacy of bacterial removal from instrumented root canals in vitro related to instrumentation technique and size*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 366.
185. Rosenthal S, Spangberg L, Safavi K. *Chlorhexidine substantivity in root canal dentin*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 488.
186. Rud J, Andreasen JO. *A study of failures after endodontic surgery by radiographic, histologic and stereomicroscopic methods*, Int J Oral Surg 1972;1: 311.
187. Russo MC, Holland R, de Souza V. *Radiographic and histological evaluation of the treatment of inflamed dental pulps*, Int Endod J 1982;15: 137.
188. Saini HR, Tewari S, Sangwan P, et al: *Effect of different apical preparation sizes on outcome of primary endodontic treatment: a randomized controlled trial*, J Endod 2012;38: 1309.
189. Salehrabi R, Rotstein I. *Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study*, J Endod 2004;30: 846.
190. Salehrabi R, Rotstein I. *Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment*, J Endod 2010;36: 790.

Telegram: @dental_k

www.konkur.in

191. Salvi GE, Siegrist Guldener BE, Amstad T, et al: *Clinical evaluation of root filled teeth restored with or without post-and-core systems in a specialist practice setting*, Int Endod J 2007;40: 209.
192. Santini A. *Assessment of the pulpotomy technique in human first permanent mandibular molars. Use of two direct inspection criteria*, Br Dent J 1983;155: 151.
193. Santucci PJ. *Dycal versus Nd:YAG laser and Vitrebond for direct pulp capping in permanent teeth*, J Clin Laser Med Surg 1999;17: 69.
194. Sari S, Okte Z. *Success rate of Sealapex in root canal treatment for primary teeth: 3-year follow-up*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: e93.
195. Sathorn C, Parashos P, Messer H. *Antibacterial efficacy of calcium hydroxide intracanal dressing: a systematic review and meta-analysis*, Int Endod J 2007;40: 2.
196. Saunders WP, Saunders EM. *Coronal leakage as a cause of failure in root-canal therapy: a review*, Endod Dent Traumatol 1994;10: 105.
197. Scarfe W. *Use of cone-beam computed tomography in endodontics Joint Position Statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endodl 2011;111: 234.
198. Schafer E, Bossmann K. *Antimicrobial efficacy of chlorhexidine and two calcium hydroxide formulations against Enterococcus faecalis*, J Endod 2005;31: 53.
199. Schilder H. *Cleaning and shaping the root canal*, Dent Clin North Am 1974;18: 269.
200. Schwendicke F, Stolpe M, Meyer-Lueckel H, et al: *Cost-effectiveness of one- and two-step incomplete and complete excavations*, J Dent Res 2013;92: 880.

Telegram: @dental_k

www.konkur.in

201. Seltzer S, Bender IB, Turkenkopf S. *Factors affecting successful repair after root canal therapy*, J Am Dent Assoc 1963;67: 651.
202. Seltzer S, Naidorf IJ. *Flare-ups in endodontics: I. Etiological factors*, J Endod 1985;11: 472.
203. Seltzer S, Naidorf IJ. *Flare-ups in endodontics: II. Therapeutic measures*, J Endod 1985;11: 559.
204. Setzer FC, Kohli MR, Shah SB, et al: *Outcome of endodontic surgery: a meta-analysis of the literature. Part 2: Comparison of endodontic microsurgical techniques with and without the use of higher magnification*, J Endod 2012;38: 1.
205. Setzer FC, Shah SB, Kohli MR, et al: *Outcome of endodontic surgery: a meta-analysis of the literature. Part 1: Comparison of traditional root-end surgery and endodontic microsurgery*, J Endod 2010;36: 17575.
206. Shovelton D. *The presence and distribution of microorganisms within non-vital teeth*, Br Dent J 1964;117: 101.
207. Shovelton DS, Friend LA, Kirk EE, et al: *The efficacy of pulp capping materials: a comparative trial*, Br Dent J 1971;130: 385.
208. Shuping GB, Orstavik D, Sigurdsson A, et al: *Reduction of intracanal bacteria using nickel-titanium rotary instrumentation and various medications*, J Endod 2000;26: 751.
209. Siqueira JF Jr, Guimaraes-Pinto T, Rocas IN. *Effects of chemomechanical preparation with 2.5% sodium hypochlorite and intracanal medication with calcium hydroxide on cultivable bacteria in infected root canals*, J Endod 2007;33: 800.
210. Siqueira JF Jr, Magalhaes KM, Rocas IN. *Bacterial*

Telegram: @dental_k

www.konkur.in

reduction in infected root canals treated with 2.5% NaOCl as an irrigant and calcium hydroxide/camphorated paramonochlorophenol paste as an intracanal dressing, J Endod 2007;33: 667.

211. Siqueira JF Jr, Rocas IN, Paiva SS, et al: *Bacteriologic investigation of the effects of sodium hypochlorite and chlorhexidine during the endodontic treatment of teeth with apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 122.
212. Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Relationship between Fcgamma receptor and interleukin-1 gene polymorphisms and post-treatment apical periodontitis*, J Endod 2009;35: 11862.
213. Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Polymorphism of the FcgammaRIIIa gene and post-treatment apical periodontitis*, J Endod 2011;37: 13458.
214. Sjogren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int Endod J 1997;30: 297.
215. Sjogren U, Figdor D, Spangberg L, et al: *The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing*, Int Endod J 1991;24: 119.
216. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498.
217. Sjogren U, Sundqvist G. *Bacteriologic evaluation of ultrasonic root canal instrumentation*, Oral Surg Oral Med Oral Pathol 1987;63: 366.
218. Sjogren U, Sundqvist G, Nair PN. *Tissue reaction to gutta-percha particles of various sizes when implanted subcutaneously in guinea pigs*, Eur J Oral Sci 1995;103:

Telegram: @dental_k

www.konkur.in

313.
219. Slade GD. *Derivation and validation of a short-form oral health impact profile*, Community Dent Oral Epidemiol 1997;25: 284.
220. Smith CS, Setchell DJ, Harty FJ. *Factors influencing the success of conventional root canal therapy—a five-year retrospective study*, Int Endod J 1993;26: 321.
221. Sogur E, Baksi BG, Grondahl HG, et al: *Detectability of chemically induced periapical lesions by limited cone beam computed tomography, intra-oral digital and conventional film radiography*, Dentomaxillofac Radiol 2009;38: 458.
222. Song M, Kim SG, Lee SJ, et al: *Prognostic factors of clinical outcomes in endodontic microsurgery: a prospective study*, J Endod 2013;39: 14917.
223. Souza RA, Dantas JC, Brandao PM, et al: *Apical third enlargement of the root canal and its relationship with the repair of periapical lesions*, Eur J Dent 2012;6: 385.
224. Spangberg LS. *Evidence-based endodontics: the one-visit treatment idea*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 617.
225. Spili P, Parashos P, Messer HH. *The impact of instrument fracture on outcome of endodontic treatment*, J Endod 2005;31: 845.
226. Stavropoulos A, Wenzel A. *Accuracy of cone beam dental CT, intraoral digital and conventional film radiography for the detection of periapical lesions: an ex vivo study in pig jaws*, Clin Oral Investig 2007;11: 101.
227. Stewart GG, Cobe H, Rappaport H. *Study of new medicament in chemomechanical preparation of infected root canals*, J Am Dent Assoc 1961;63: 33.
228. Stoll R, Betke K, Stachniss V. *The influence of different*

Telegram: @dental_k

www.konkur.in

factors on the survival of root canal fillings: a 10-year retrospective study, J Endod 2005;31: 783.

229. Strindberg LZ. The dependence of the results of pulp therapy on certain factors: an analytic study based on radiographic and clinical follow-up examinations 1956; Mauritzon.
230. Sundqvist G. Bacteriological studies of necrotic dental pulps 1976; University of Umeå Sweden.
231. Sundqvist G, Figdor D, Persson S, et al: *Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 86.
232. Taschieri S, Corbella S, Tsesis I, et al: *Effect of guided tissue regeneration on the outcome of surgical endodontic treatment of through-and-through lesions: a retrospective study at 4-year follow-up*, Oral Maxillofac Surg 2011;15: 153.
233. Taschieri S, Del Fabbro M, Testori T, et al: *Efficacy of guided tissue regeneration in the management of through-and-through lesions following surgical endodontics: a preliminary study*, Int J Periodontics Restorative Dent 2008;28: 265.
234. Taschieri S, Del Fabbro M, Testori T, Weinstein R. *Efficacy of xenogeneic bone grafting with guided tissue regeneration in the management of bone defects after surgical endodontics*, J Oral Maxillofac Surg 2007;65: 11217.
235. Teixeira LS, Demarco FF, Coppola MC, et al: *Clinical and radiographic evaluation of pulpotomies performed under intrapulpal injection of anaesthetic solution*, Int Endod J 2001;34: 440.
236. Tickle M, Milsom K, Qualtrough A, et al: *The failure*

Telegram: @dental_k

www.konkur.in

rate of NHS funded molar endodontic treatment delivered in general dental practice, Br Dent J 2008;204: E8-discussion 254.

237. Tobón SI, Arismendi JA, Marín ML, et al: *Comparison between a conventional technique and two bone regeneration techniques in periradicular surgery*, Int Endod J 2002;35: 635.
238. Torabinejad M, Goodacre CJ. *Endodontic or dental implant therapy*, J Amer Dent Assoc 2006;137: 973.
239. Tsesis I, Rosen E, Tamse A, et al: *Effect of guided tissue regeneration on the outcome of surgical endodontic treatment: a systematic review and meta-analysis*, J Endod 2011;37: 10395.
240. Tsesis I, Rosen E, Taschieri S, et al: *Outcomes of surgical endodontic treatment performed by a modern technique: an updated meta-analysis of the literature*, J Endod 2013;39: 332.
241. Tsesis I, Taivishevsky V, Kfir A, et al: *Outcome of surgical endodontic treatment performed by a modern technique: a meta-analysis of literature*, J Endod 2009;35: 1505.
242. Tuna D, Olmez A. *Clinical long-term evaluation of MTA as a direct pulp capping material in primary teeth*, Int Endod J 2008;41: 273.
243. Tyndall DA, Kapa SF, Bagnell CP. *Digital subtraction radiography for detecting cortical and cancellous bone changes in the periapical region*, J Endod 1990;16: 173.
244. Van Nieuwenhuysen JP, Aouar M, D'Hoore W. *Retreatment or radiographic monitoring in endodontics*, Int Endod J 1994;27: 75.
245. Velvart P. *Papilla base incision: a new approach to recession-free healing of the interdental papilla after*

Telegram: @dental_k

www.konkur.in

endodontic surgery, Int Endod J 2002;35: 453.

246. Velvart P, Ebner-Zimmermann U, Ebner JP. *Comparison of papilla healing following sulcular full-thickness flap and papilla base flap in endodontic surgery*, Int Endod J 2003;36: 653.
247. Velvart P, Ebner-Zimmermann U, Ebner JP. *Comparison of long-term papilla healing following sulcular full thickness flap and papilla base flap in endodontic surgery*, Int Endod J 2004;37: 687.
248. Vianna ME, Horz HP, Conrads G, et al: *Effect of root canal procedures on endotoxins and endodontic pathogens*, Oral Microbiol Immunol 2007;22: 411.
249. Villa-Machado PA, Botero-Ramírez X, Tobón-Arroyave SI. *Retrospective follow-up assessment of prognostic variables associated with the outcome of periradicular surgery*, Int Endod J 2013;46: 1063.
250. Von Arx T, Peñarrocha M, Jensen S. *Prognostic factors in apical surgery with root-end filling: A meta-analysis*, J Endod 2010;36: 957.
251. Waly NG. *A five-year comparative study of calcium hydroxide-glutaraldehyde pulpotomies versus calcium hydroxide pulpotomies in young permanent molars*, Egypt Dent J 1995;41: 993.
252. Wang CS, Arnold RR, Trope M, et al: *Clinical efficiency of 2% chlorhexidine gel in reducing intracanal bacteria*, J Endod 2007;33: 1283.
253. Wang Q, Cheung GS, Ng RP. *Survival of surgical endodontic treatment performed in a dental teaching hospital: a cohort study*, Int Endod J 2004;37: 764.
254. Weiss M. *Pulp capping in older patients*, N Y State Dent J 1966;32: 451.
255. Witherspoon DE, Small JC, Harris GZ. *Mineral trioxide*

Telegram: @dental_k

www.konkur.in

aggregate pulpotomies: a case series outcomes assessment, J Am Dent Assoc 2006;137: 610.

256. Wu MK, Shemesh H, Wesselink PR. *Limitations of previously published systematic reviews evaluating the outcome of endodontic treatment*, Int Endod J 2009;42: 656.

257. Xavier AC, Martinho FC, Chung A, et al: *One-visit versus two-visit root canal treatment: effectiveness in the removal of endotoxins and cultivable bacteria*, J Endod 2013;39: 959.

258. Yared GM, Dagher FE. *Influence of apical enlargement on bacterial infection during treatment of apical periodontitis*, J Endod 1994;20: 535.

259. Yoshioka T, Kobayashi C, Suda H, et al: *An observation of the healing process of periapical lesions by digital subtraction radiography*, J Endod 2002;28: 589.

260. Yusuf H. *The significance of the presence of foreign material periapically as a cause of failure of root treatment*, Oral Surg Oral Med Oral Pathol 1982;54: 566.

261. Zehnder M. *Root canal irrigants*, J Endod 2006;32: 389.

Telegram: @dental_k

www.konkur.in

Figure and Table References

Adenubi JO, Rule DC. *Success rate for root fillings in young patients: a retrospective analysis of treated cases*, Br Dent J 1976;141: 237.

Akerblom A, Hasselgren G. *The prognosis for endodontic treatment of obliterated root canals*, J Endod 1988;14: 565.

Akpata ES. *Effect of endodontic procedures on the population of viable microorganisms in the infected root canal*, J Endod 1976;2: 369.

Allen RK, Newton CW, Brown CE Jr. *A statistical analysis of surgical and nonsurgical endodontic retreatment cases*, J Endod 1989;15: 261.

Alley BS, Kitchens GG, Alley LW, Eleazer PD. *A comparison of survival of teeth following endodontic treatment performed by general dentists or by specialists*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 115.

Amagasa T, Nagase M, Sato T, Shioda S. *Apicoectomy with retrograde gutta-percha root filling*, Oral Surg Oral Med Oral Pathol 1989;68: 339.

American Academy of Pediatric Dentistry 2014 Pulp Therapy Subcommittee, Clinical Affairs Committee: American Academy of Pediatric Dentistry (AAPD) guideline on pulp therapy for primary and immature permanent teeth reference manual, vol 36, no 6, Chicago, 2014, AAPD, p 242, www.aapd.org/media/Policies_Guidelines/G_Pulp.pdf. Accessed April 19, 2015.

Appleton JLT. *A note on the clinical value of*

Telegram: @dental_k

www.konkur.in

bacteriologically controlling the treatment of periapical infection, Dental Cosmos 1932;74: 798.

Aqrabawi J. *Management of endodontic failures: case selection and treatment modalities*, Gen Dent 2005;53: 63.

Aqrabawi JA. *Outcome of endodontic treatment of teeth filled using lateral condensation versus vertical compaction (Schilder's technique)*, J Contemp Dent Pract 2006;7: 17.

Aquilino SA, Caplan DJ. *Relationship between crown placement and the survival of endodontically treated teeth*, J Prosthet Dent 2002;87: 256.

Asgary S, Ehsani S. *Permanent molar pulpotomy with a new endodontic cement: a case series*, J Conserv Dent 2009;12: 31.

Auerbach M. *Antibiotics vs. instrumentation in endodontics*, N Y State Dent J 1953;19: 225.

Auerbach MB. *Clinical approach to the problem of pulp canal therapy*, J Am Dent Assoc 1938;25: 939.

Baratieri LN, Monteiro S Jr, Caldeira de Andrada MA. *Pulp curettage: surgical technique*, Quintessence Int 1989;20: 285.

Barbakow FH, Cleaton-Jones P, Friedman D. *An evaluation of 566 cases of root canal therapy in general dental practice. 2. Postoperative observations*, J Endod 1980;6: 485.

Barone C, Dao TT, Basrani BB, et al: *Treatment outcome in endodontics: the Toronto study—phases 3, 4, and 5: apical surgery*, J Endod 2010;36: 28.

Barrieshi-Nusair KM, Qudeimat MA. *A prospective clinical study of mineral trioxide aggregate for partial pulpotomy in cariously exposed permanent teeth*, J Endod 2006;32: 731.

Telegram: @dental_k

www.konkur.in

Barthel CR, Rosenkranz B, Leuenberg A, et al: *Pulp capping of carious exposures: treatment outcome after 5 and 10 years: a retrospective study*, J Endod 2000;26: 525.

Bence R, Madonia JV, Weine FS, Smulson MH. *A microbiologic evaluation of endodontic instrumentation in pulpless teeth*, Oral Surg Oral Med Oral Pathol 1973;35: 676.

Bender IB, Seltzer S, Soltanof W. *Endodontic success: a reappraisal of criteria 1*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1966;22: 780.

Bender IB, Seltzer S, Soltanof W. *Endodontic success: a reappraisal of criteria 2*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1966;22: 790.

Bender IB, Seltzer S, Turkenkopf S. *To culture or not to culture?*, Oral Surg Oral Med Oral Pathol 1964;18: 527.

Benenati FW, Khajotia SS. *A radiographic recall evaluation of 894 endodontic cases treated in a dental school setting*, J Endod 2002;28: 391.

Bergenholtz G, Lekholm U, Milthon R, et al: *Influence of apical overinstrumentation and overfilling on re-treated root canals*, J Endod 1979;5: 310.

Bjorndal L, Reit C, Bruun G, et al: *Treatment of deep caries lesions in adults: randomized clinical trials comparing stepwise vs. direct complete excavation, and direct pulp capping vs. partial pulpotomy*, Eur J Oral Sci 2010;118: 290.

Bjorndal L, Thylstrup A. *A practice-based study on stepwise excavation of deep carious lesions in permanent teeth: a 1-year follow-up study*, Community Dent Oral Epidemiol 1998;26: 122.

Blayney JR. *The clinical results of pulp treatment*, J Natl Dent Assoc 1922;9: 198.

Telegram: @dental_k

www.konkur.in

Bogen G, Kim JS, Bakland LK. *Direct pulp capping with mineral trioxide aggregate: an observational study*, J Am Dent Assoc 2008;139: 305- quiz 305.

Boggia R. *A single-visit treatment of septic root canals using periapically extruded endomethasone*, Br Dent J 1983;155: 300.

Buchbinder M. *A statistical comparison of cultured and non-cultured root canal cases*, J Dent Res 1941;20: 93.

Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 1985;1: 170.

Bystrom A, Happonen RP, Sjogren U, et al: *Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis*, Endod Dent Traumatol 1987;3: 58.

Bystrom A, Sundqvist G. *Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy*, Scand J Dent Res 1981;89: 321.

Bystrom A, Sundqvist G. *Bacteriologic evaluation of the effect of 0.5 percent sodium hypochlorite in endodontic therapy*, Oral Surg Oral Med Oral Pathol 1983;55: 307.

Bystrom A, Sundqvist G. *The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy*, Int Endod J 1985;18: 35.

Caliskan MK. *Success of pulpotomy in the management of hyperplastic pulpitis*, Int Endod J 1993;26: 142.

Caliskan MK. *Pulpotomy of carious vital teeth with periapical involvement*, Int Endod J 1995;28: 172.

Caliskan MK. *Nonsurgical retreatment of teeth with periapical lesions previously managed by either*

Telegram: @dental_k

www.konkur.in

endodontic or surgical intervention, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 242.

Caliskan MK, Sen BH. *Endodontic treatment of teeth with apical periodontitis using calcium hydroxide: a long-term study*, Endod Dent Traumatol 1996;12: 215.

Caplan DJ, Cai J, Yin G, et al: *Root canal filled versus non-root canal filled teeth: a retrospective comparison of survival times*, J Public Health Dent 2005;65: 90.

Caplan DJ, Kolker J, Rivera EM, et al: *Relationship between number of proximal contacts and survival of root canal treated teeth*, Int Endod J 2002;35: 193.

Card SJ, Sigurdsson A, Orstavik D, et al: *The effectiveness of increased apical enlargement in reducing intracanal bacteria*, J Endod 2002;28: 779.

Castagnola L, Orlay HG. *Treatment of gangrene of the pulp by the Walkhoff method*, Br Dent J 1952;19: 93.

Chen SC, Chueh LH, Hsiao CK, et al: *An epidemiologic study of tooth retention after nonsurgical endodontic treatment in a large population in Taiwan*, J Endod 2007;33: 226.

Chen S-C, Chueb L-H, Hsiao CK, et al: *First untoward events and reasons for tooth extraction after nonsurgical endodontic treatment in Taiwan*, J Endod 2008;34: 671.

Cheung GS. *Survival of first-time nonsurgical root canal treatment performed in a dental teaching hospital*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 596.

Cheung GS, Chan TK. *Long-term survival of primary root canal treatment carried out in a dental teaching hospital*, Int Endod J 2003;36: 117.

Chong BS, Pitt Ford TR, Hudson MB. *A prospective clinical study of Mineral Trioxide Aggregate and IRM when used*

Telegram: @dental_k

www.konkur.in

as root-end filling materials in endodontic surgery, Int Endod J 2003;36: 520.

Christiansen R, Kirkevang LL, Horsted-Bindslev P, et al: *Randomized clinical trial of root-end resection followed by root-end filling with mineral trioxide aggregate or smoothing of the orthograde gutta-percha root filling—1-year follow-up*, Int Endod J 2009;42: 105.

Chu FC, Leung WK, Tsang PC, et al: *Identification of cultivable microorganisms from root canals with apical periodontitis following two-visit endodontic treatment with antibiotics/steroid or calcium hydroxide dressings*, J Endod 2006;32: 17.

Chu FC, Tsang CS, Chow TW, et al: *Identification of cultivable microorganisms from primary endodontic infections with exposed and unexposed pulp space*, J Endod 2005;31: 424.

Chugal NM, Clive JM, Spångberg LSW. *A prognostic model for assessment of the outcome of endodontic treatment: effect of biologic and diagnostic variables*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 342.

Chugal NM, Clive JM, Spångberg LS. *Endodontic infection: some biologic and treatment factors associated with outcome*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 81.

Conner DA, Caplan DJ, Teixeira FB, et al: *Clinical outcome of teeth treated endodontically with a nonstandardized protocol and root filled with resilon*, J Endod 2007;33: 1290.

Cotton TP, Schindler WG, Schwartz SA, et al: *A retrospective study comparing clinical outcomes after obturation with Resilon/Epiphany or Gutta-Percha/Kerr sealer*, J Endod 2008;34: 789.

Telegram: @dental_k

www.konkur.in

Cvek M. *Treatment of non-vital permanent incisors with calcium hydroxide*, Odontol Revy 1972;23: 27.
Cvek M. *A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture*, J Endod 1978;4: 232.
Cvek M. *Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha: a retrospective clinical study*, Endod Dent Traumatol 1992;8: 45.
Cvek M, Granath L, Lundberg M. *Failures and healing in endodontically treated non-vital anterior teeth with posttraumatically reduced pulpal lumen*, Acta Odontol Scand 1982;40: 223.
Cvek M, Hollender L, Nord CE. *Treatment of non-vital permanent incisors with calcium hydroxide. VI: A clinical, microbiological and radiological evaluation of treatment in one sitting of teeth with mature or immature root*, Odontol Revy 1976;27: 93.
Dalton BC, Orstavik D, Phillips C, et al: *Bacterial reduction with nickel-titanium rotary instrumentation*, J Endod 1998;24: 763.
Dammaschke T, Steven D, Kaup M, et al: *Long-term survival of root-canal-treated teeth: a retrospective study over 10 years*, J Endod 2003;29: 638.
Danin J, Strömberg T, Forsgren H, et al: *Clinical management of nonhealing periradicular pathosis: surgery versus endodontic retreatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 213.
de Chevigny C, Dao TT, Basrani BR, et al: *Treatment outcome in endodontics: the Toronto study–phase 4: initial treatment*, J Endod 2008;34: 258.
de Lange J, Putters T, Baas EM, et al: *Ultrasonic root-end*

Telegram: @dental_k

www.konkur.in

preparation in apical surgery: a prospective randomized study, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 841.

DeRosa TA. *A retrospective evaluation of pulpotomy as an alternative to extraction*, Gen Dent 2006;54: 37.

Deutsch AS, Musikant BL, Cohen BI, et al: *A study of one visit treatment using EZ-Fill root canal sealer*, Endod Prac 2001;4: 29.

Dominiak M, Lysiak-Drwal K, Gedrange T, et al: *Efficacy of healing process of bone defects after apicectomy: results after 6 and 12 months*, J Physiol Pharmacol suppl 8, 2009;60: 51.

Dorn SO, Gartner AH. *Retrograde filling materials: a retrospective success-failure study of amalgam, EBA, and IRM*, J Endod 1990;16: 391.

Doyle SL, Hodges JS, Pesun IJ, et al: *Retrospective cross sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants*, J Endod 2006;32: 822.

Dugas NN, Lawrence HP, Teplitsky P, et al: *Quality of life and satisfaction outcomes of endodontic treatment*, J Endod 2002;28: 819.

El-Meligy OA, Avery DR. *Comparison of mineral trioxide aggregate and calcium hydroxide as pulpotomy agents in young permanent teeth (apexogenesis)*, Pediatr Dent 2006;28: 399.

Engström B. *The significance of enterococci in root canal treatment*, Odontol Revy 1964;15: 87.

Engström B, Lundberg M. *The correlation between positive culture and the prognosis of root canal therapy after pulpectomy*, Odontol Revy 1965;16: 193.

Engström B, Segerstad LHA, Ramstrom G, Frostell G.

Telegram: @dental_k

www.konkur.in

Correlation of positive cultures with the prognosis for root canal treatment, Odontol Revy 1964;15: 257.

European Society of Endodontology. *Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology*, Int Endod J 2006;39: 921.

Farsi N, Alamoudi N, Balto K, et al: *Clinical assessment of mineral trioxide aggregate (MTA) as direct pulp capping in young permanent teeth*, J Clin Pediatr Dent 2006;31: 72.

Ferrari M, Vichi A, Fadda GM, et al: *A randomized controlled trial of endodontically treated and restored premolars*, J Dent Res 2012;91: 72S.

Field JW, Gutmann JL, Solomon ES, et al: *A clinical radiographic retrospective assessment of the success rate of single-visit root canal treatment*, Int Endod J 2004;37: 70.

Finne K, Nord PG, Persson G, et al: *Retrograde root filling with amalgam and cavit*, Oral Surg Oral Med Oral Pathol 1977;43: 621.

Fitzgerald M, Heys RJ. *A clinical and histological evaluation of conservative pulpal therapy in human teeth*, Oper Dent 1991;16: 101.

Fleming CH, Litaker MS, Alley LW, et al: *Comparison of classic endodontic techniques versus contemporary techniques on endodontic treatment success*, J Endod 2010;36: 414.

Fonzar F, Fonzar A, Buttolo P, et al: *The prognosis of root canal therapy: a 10-year retrospective cohort study on 411 patients with 1175 endodontically treated teeth*, Eur J Oral Implantol 2009;2: 201.

Friedman S, Lost C, Zarrabian M, et al: *Evaluation of success and failure after endodontic therapy using a glass ionomer cement sealer*, J Endod 1995;21: 384.

Telegram: @dental_k

www.konkur.in

Friedman S, Mor C. *The success of endodontic therapy—healing and functionality*, J Calif Dent Assoc 2004;32: 493.

Frostell G. *Clinical significance of the root canal culture*, Transactions of 3rd Int Conferences of Endodontics 1963; 112.

Fuks AB, Gavra S, Chosack A. *Long-term followup of traumatized incisors treated by partial pulpotomy*, Pediatr Dent 1993;15: 334.

Gagliani MM, Gorni FG, Strohmenger L. *Periapical resurgery versus periapical surgery: a 5-year longitudinal comparison*, Int Endod J 2005;38: 320.

Gallien GS Jr, Schuman NJ. *Local versus general anesthesia: a study of pulpal response in the treatment of cariously exposed teeth*, J Am Dent Assoc 1985;111: 599.

Garcia B, Penarrocha M, Martí E, et al: *Periapical surgery in maxillary premolars and molars: Analysis in terms of the distance between the lesion and the maxillary sinus*, J Oral Maxillofac Surg 2008;66: 1212.

García-Mira B, Ortega-Sánchez B, Peñarrocha-Diago M, et al: *Ostectomy versus osteotomy with repositioning of the vestibular cortical in periapical surgery of mandibular molars: a preliminary study*, Medicina Oral, Patología Oral y Cirugía Bucal 2010;15: e628.

Gesi A, Hakeberg M, Warfvinge J, et al: *Incidence of periapical lesions and clinical symptoms after pulpectomy—a clinical and radiographic evaluation of 1- versus 2-session treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 379.

Gomes BP, Lilley JD, Drucker DB. *Variations in the susceptibilities of components of the endodontic microflora to biomechanical procedures*, Int Endod J 1996;29: 235.

Telegram: @dental_k

www.konkur.in

Gorni FGM, Gagliani MM. *The outcome of endodontic retreatment: a 2-yr follow-up*, J Endod 2004;30: 1.
Goyal B, Tewari S, Duhan J, et al: *Comparative evaluation of platelet-rich plasma and guided tissue regeneration membrane in the healing of apicomarginal defects: a clinical study*, J Endod 2011;37: 773.
Grahnén H, Hansson L. *The prognosis of pulp and root canal therapy*, Odontol Revy 1961;12: 146.
Grahnen H, Krasse B. *The effect of instrumentation and flushing of non-vital teeth in endodontic therapy*, Odontol Rev 1963;14: 167.
Grung B, Molven O, Halse A. *Periapical surgery in a Norwegian county hospital: Follow-up findings of 477 teeth*, J Endod 1990;16: 411.
Gruythuysen RJ, van Strijp AJ, Wu MK. *Long-term survival of indirect pulp treatment performed in primary and permanent teeth with clinically diagnosed deep carious lesions*, J Endod 2010;36: 1490.
Halse A, Molven O. *Overextended gutta-percha and Kloroperka N-Ø root canal fillings: radiographic findings after 10–17 years*, Acta Odontol Scand 1987;45: 171.
Harty FJ, Parkins BJ, Wengraf AM. *Success rate in root canal therapy: a retrospective study on conventional cases*, Br Dent J 1970;128: 65.
Haskell EW, Stanley HR, Chellemi J, et al: *Direct pulp capping treatment: a long-term follow-up*, J Am Dent Assoc 1978;97: 607.
Hayashi M, Fujitani M, Yamaki C, et al: *Ways of enhancing pulp preservation by stepwise excavation—a systematic review*, J Dent 2011;39: 95.
Heling B, Kischinovsky D. *Factors affecting successful endodontic therapy*, J Br Endod Soc 1979;12: 83.

Telegram: @dental_k

www.konkur.in

Heling B, Shapira J. *Roentgenologic and clinical evaluation of endodontically treated teeth, with or without negative culture*, Quintessence Int 1978;11: 79.

Heling B, Tamshe A. *Evaluation of the success of endodontically treated teeth*, Oral Surg Oral Med Oral Pathol 1970;30: 533.

Heling I, Bialla-Shenkman S, Turetzky A, et al: *The outcome of teeth with periapical periodontitis treated with nonsurgical endodontic treatment: a computerized morphometric study*, Quintessence Int 2001;32: 397.

Hilton TJ, Ferracane JL, Mancl L, Northwest Practice-based Research Collaborative in Evidence-based Dentistry (NWP). *Comparison of CaOH with MTA for direct pulp capping: a PBRN randomized clinical trial*, J Dent Res 2013;92: 16S.

Hirsch J-M, Ahlström U, Henrikson P-Å, et al: *Periapical surgery*, Int J Oral Surg 1979;8: 173.

Horsted P, Sandergaard B, Thylstrup A, et al: *A retrospective study of direct pulp capping with calcium hydroxide compounds*, Endod Dent Traumatol 1985;1: 29.

Hoskinson SE, Ng YL, Hoskinson AE, et al: *A retrospective comparison of outcome of root canal treatment using two different protocols*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 705.

Hsiao A, Glickman G, He J. *A retrospective clinical and radiographic study on healing of periradicular lesions in patients taking oral bisphosphonates*, J Endod 2009;35: 1525.

Huumonen S, Lenander-Lumikari M, Sigurdsson A, et al: *Healing of apical periodontitis after endodontic treatment: a comparison between a silicone-based and a zinc oxide-eugenol-based sealer*, Int Endod J 2003;36: 296.

Telegram: @dental_k

www.konkur.in

Ingle J, Zeldow B. *An evaluation of mechanical instrumentation and the negative culture in endodontic therapy*, J Am Dent Assoc 1958;57: 471.

Ioannides C, Borstlap WA. *Apicoectomy on molars: a clinical and radiographical study*, Int J Oral Surg 1983;12: 73.

Jansson L, Sandstedt P, Låftman AC, et al: *Relationship between apical and marginal healing in periradicular surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 596.

Jensen S, Nattestad A, Egdø P, et al: *A prospective, randomized, comparative clinical study of resin composite and glass ionomer cement for retrograde root filling*, Clin Oral Investig 2002;6: 236.

Jesslén P, Zetterqvist L, Heimdahl A. *Long-term results of amalgam versus glass ionomer cement as apical sealant after apicectomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79: 101.

Jokinen MA, Kotilainen R, Poikkeus P, et al: *Clinical and radiographic study of pulpectomy and root canal therapy*, Scand J Dent Res 1978;86: 366.

Jordan RE, Suzuki M. *Conservative treatment of deep carious lesions*, J Can Dent Assoc 1971;37: 337.

Jordan RE, Suzuki M, Skinner DH. *Indirect pulp-capping of carious teeth with periapical lesions*, J Am Dent Assoc 1978;97: 37.

Jurcak JJ, Bellizzi R, Loushine RJ. *Successful single-visit endodontics during Operation Desert Shield*, J Endod 1993;19: 412.

Kerekes K. *Radiographic assessment of an endodontic treatment method*, J Endod 1978;4: 210.

Khedmat S. *Evaluation of endodontic treatment failure of*

Telegram: @dental_k

www.konkur.in

teeth with periapical radiolucent areas and factors affecting it, Front Dent 2004;1: 34.

Kim E, Song JS, Jung IY, et al: *Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal–endodontic origin*, J Endod 2008;34: 546.

Klevant FJH, Eggink CO. *The effect of canal preparation on periapical disease*, Int Endod J 1983;16: 68.

Koontongkaew S, Silapichit R, Thaweboon B. *Clinical and laboratory assessments of camphorated monochlorophenol in endodontic therapy*, Oral Surg Oral Med Oral Pathol 1988;65: 757.

Kreisler M, Gockel R, Aubell-Falkenberg S, et al: *Clinical outcome in periradicular surgery: effect of patient- and tooth-related factors: a multicenter study*, Quintessence Int 2013;44: 53.

Kvist T, Molander A, Dahlen G, et al: *Microbiological evaluation of one- and two-visit endodontic treatment of teeth with apical periodontitis: a randomized, clinical trial*, J Endod 2004;30: 572.

Lana MA, Ribeiro-Sobrinho AP, Stehling R, et al: *Microorganisms isolated from root canals presenting necrotic pulp and their drug susceptibility in vitro*, Oral Microbiol Immunol 2001;16: 100.

Lazarski MP, Walker WA III, Flores CM, et al: *Epidemiological evaluation of the outcomes of nonsurgical root canal treatment in a large cohort of insured dental patients*, J Endod 2001;27: 791.

Leco-Berrocal MI, Martinez Gonzalez JM, Donado Rodriguez M. *Clinical and radiological course in apicoectomies with the Erbium:YAG laser, Medicina Oral*,

Telegram: @dental_k

www.konkur.in

Patología Oral y Cirugía Bucal 2007;12: E65.
Leksell E, Ridell K, Cvek M, et al: *Pulp exposure after stepwise versus direct complete excavation of deep carious lesions in young posterior permanent teeth*, Endod Dent Traumatol 1996;12: 192.
Li H, Zhai F, Zhang R, et al: *Evaluation of microsurgery with SuperEBA as root-end filling material for treating post-treatment endodontic disease: a 2-year retrospective study*, J Endod 2014;40: 345.
Liang YH, Li G, Wesselink PR, et al: *Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans*, J Endod 2011;37: 326.
Liang YH, Yuan M, Li G, et al: *The ability of cone-beam computed tomography to detect simulated buccal and lingual recesses in root canals*, Int Endod J 2012;45: 724.
Lilly JP, Cox D, Arcuri M, et al: *An evaluation of root canal treatment in patients who have received irradiation to the mandible and maxilla*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86: 224.
Lindeboom JAH, Frenken J, Kroon FHM, et al: *A comparative prospective randomized clinical study of MTA and IRM as root-end filling materials in single-rooted teeth in endodontic surgery*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 495.
Lumley PJ, Lucarotti PS, Burke FJ. *Ten-year outcome of root fillings in the General Dental Services in England and Wales*, Int Endod J 2008;41: 577.
Lustmann J, Friedman S, Shaharabany V. *Relation of pre- and intraoperative factors to prognosis of posterior apical surgery*, J Endod 1991;17: 239.
Lynch CD, Burke FM, Ní Ríordáin R, et al: *The influence of*

Telegram: @dental_k

www.konkur.in

coronal restoration type on the survival of endodontically treated teeth, Eur J Prosthodont Restor Dent 2004;12: 171.

Mackie IC, Worthington HV, Hill FJ. *A follow-up study of incisor teeth which had been treated by apical closure and root filling*, Br Dent J 1993;175: 99.

Maddalone M, Gagliani M. *Periapical endodontic surgery: a 3-year follow-up study*, Int Endod J 2003;36: 193.

Maltz M, Alves LS, Jardim JJ, et al: *Incomplete caries removal in deep lesions: a 10-year prospective study*, Am J Dent 2011;24: 211.

Maltz M, Garcia R, Jardim JJ, et al: *Randomized trial of partial vs. stepwise caries removal: 3-year follow-up*, J Dent Res 2012;91: 1026.

Maltz M, Henz SL, de Oliveira EF, et al: *Conventional caries removal and sealed caries in permanent teeth: a microbiological evaluation*, J Dent 2012;40: 776.

Marín-Botero ML, Domínguez-Mejía JS, Arismendi-Echavarría JA, et al: *Healing response of apicomarginal defects to two guided tissue regeneration techniques in periradicular surgery: A double-blind, randomized-clinical trial*, Int Endod J 2006;39: 368.

Markvart M, Dahlen G, Reit CE, et al: *The antimicrobial effect of apical box versus apical cone preparation using iodine potassium iodide as root canal dressing: a pilot study*, Acta Odontol Scand 2013;71: 786.

Marti-Bowen E, Penarrocha-Diago M, Garcia-Mira B. *Periapical surgery using the ultrasound technique and silver amalgam retrograde filling. A study of 71 teeth with 100 canals*, Medicina Oral, Patologia Oral y Cirugia Bucal 2005;10: E67.

Mass E, Zilberman U. *Clinical and radiographic evaluation of partial pulpotomy in carious exposure of permanent*

Telegram: @dental_k

www.konkur.in

molars, Pediatr Dent 1993;15: 257.
Masterton JB. *The healing of wounds of the dental pulp: an investigation of the nature of the scar tissue and of the phenomena leading to its formation*, Dent Pract Dent Rec 1966;16: 325.
Matsumoto T, Nagai T, Ida K, et al: *Factors affecting successful prognosis of root canal treatment*, J Endod 1987;13: 239.
Matsuo T, Nakanishi T, Shimizu H, Ebisu S. *A clinical study of direct pulp capping applied to carious-exposed pulps*, J Endod 1996;22: 551.
Mejare I, Cvek M. *Partial pulpotomy in young permanent teeth with deep carious lesions*, Endod Dent Traumatol 1993;9: 238.
Mente J, Geletneky B, Ohle M, et al: *Mineral trioxide aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcome*, J Endod 2010;36: 806.
Mente J, Hage N, Pfefferle T, et al: *Mineral trioxide aggregate apical plugs in teeth with open apical foramina: a retrospective analysis of treatment outcome*, J Endod 2009;35: 1354.
Miles JP, Gluskin AH, Chambers D, et al: *Pulp capping with mineral trioxide aggregate (MTA): a retrospective analysis of carious pulp exposures treated by undergraduate dental students*, Oper Dent 2010;35: 20.
Molander A, Reit C, Dahlen G. *Microbiological evaluation of clindamycin as a root canal dressing in teeth with apical periodontitis*, Int Endod J 1990;23: 113.
Molander A, Warfvinge J, Reit C, et al: *Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical*

Telegram: @dental_k

www.konkur.in

periodontitis: a randomized clinical trial, J Endod 2007;33: 1145.

Molven O, Halse A. *Success rates for gutta-percha and Kloropercha N-Ø root fillings made by undergraduate students: radiographic findings after 10-17 years*, Int Endod J 1998;21: 243.

Molven O, Halse A, Grung B. *Observer strategy and the radiographic classification of healing after endodontic surgery*, Int J Oral Maxillofac Surg 1987;16: 432.

Moore DL. *Conservative treatment of teeth with vital pulps and periapical lesions: a preliminary report*, J Prosthet Dent 1967;18: 476.

Morsani JM, Aminoshariae A, Han YW, et al: *Genetic predisposition to persistent apical periodontitis*, J Endod 2011;37: 455.

Morse DR, Esposito JV, Pike C, et al: *A radiographic evaluation of the periapical status of teeth treated by the gutta-percha-eucapercha endodontic method: a one-year follow-up study of 458 root canals. Part I*, Oral Surg Oral Med Oral Pathol 1983;55: 607.

Morse DR, Esposito JV, Pike C, et al: *radiographic evaluation of the periapical status of teeth treated by the gutta-percha-eucapercha endodontic method: a one-year follow-up study of 458 root canals. Part II*, Oral Surg Oral Med Oral Pathol 1983;56: 89.

Morse DR, Esposito JV, Pike C, et al: *A radiographic evaluation of the periapical status of teeth treated by the gutta-percha-eucapercha endodontic method: a one-year follow-up study of 458 root canals. Part III*, Oral Surg Oral Med Oral Pathol 1983;56: 190.

Morse F, Yates M. *Follow up studies of root-filled teeth in relation to bacteriologic findings*, J Am Dent Assoc

Telegram: @dental_k

www.konkur.in

1941;28: 956.
Moshonov J, Slutzky-Goldberg I, Gottlieb A, et al: *The effect of the distance between post and residual gutta-percha on the clinical outcome of endodontic treatment*, J Endod 2005;31: 177.
Murphy WK, Kaugars GE, Collett WK, et al: *Healing of periapical radiolucencies after nonsurgical endodontic therapy*, Oral Surg Oral Med Oral Pathol 1991;71: 620.
Nagamine M. *Studies on treatment of deep caries lesions utilizing polycarboxylate cement combined with tanninfluoride preparation*, J Okayama Dent Soc 1993;12: 1- [Japanese].
Nagasiri R, Chitmongkolsuk S. *Long-term survival of endodontically treated molars without crown coverage: a retrospective cohort study*, J Prosthet Dent 2005;93: 164.
Negishi J, Kawanami M, Ogami E. *Risk analysis of failure of root canal treatment for teeth with inaccessible apical constriction*, J Dent 2005;33: 399.
Nelson IA. *Endodontics in general practice: a retrospective study*, Int Endod J 1982;15: 168.
Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health*, Int Endod J 2011;44: 583.
Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2: tooth survival*, Int Endod J 2011;44: 610.
Nicholls E. *The efficacy of cleansing of the root canal*, Br Dent J 1962;112: 167.
Nordenram A. *Biobond for retrograde root filling in apicoectomy*, Eur J Oral Sci 1970;78: 251.

Telegram: @dental_k

www.konkur.in

Olgart LG. *Bacteriological sampling from root canals directly after chemo-mechanical treatment: a clinical and bacteriological study*, Acta Odontol Scand 1969;27: 91.

Oliet S. *Single-visit endodontics: a clinical study*, J Endod 1983;9: 147.

Oliet S, Sorin SM. *Evaluation of clinical results based upon culturing root canals*, J Br Endod Soc 1969;3: 3.

Orstavik D. *Time-course and risk analyses of the development and healing of chronic apical periodontitis in man*, Int Endod J 1996;29: 150.

Orstavik D, Kerekes K, Molven O. *Effects of extensive apical reaming and calcium hydroxide dressing on bacterial infection during treatment of apical periodontitis: a pilot study*, Int Endod J 1991;24: 1.

Ortega-Sánchez B, Peñarrocha-Diago M, Rubio-Martínez LA, Vera-Sempere JF. *Radiographic morphometric study of 37 periapical lesions in 30 patients: validation of success criteria*, J Oral Maxillofac Surg 2009;67: 846.

Pantchev A, Nohlert E, Tegelberg A. *Endodontic surgery with and without inserts of bioactive glass PerioGlas: a clinical and radiographic follow-up*, Oral Maxillofac Surg 2009;13: 21.

Pantschev A, Carlsson AP, Andersson L. *Retrograde root filling with EBA cement or amalgam: a comparative clinical study*, Oral Surg Oral Med Oral Pathol 1994;78: 101.

Paquette L, Legner M, Fillery ED, et al: *Antibacterial efficacy of chlorhexidine gluconate intracanal medication in vivo*, J Endod 2007;33: 788.

Peak JD. *The success of endodontic treatment in general dental practice: a retrospective clinical and radiographic study*, Prim Dent Care 1994;1: 9.

Telegram: @dental_k

www.konkur.in

Peak JD, Hayes SJ, Bryant ST, et al: *The outcome of root canal treatment. A retrospective study within the armed forces (Royal Air Force)*, Br Dent J 2001;190: 140.

Peciuliene V, Balciuniene I, Eriksen HM, et al: *Isolation of Enterococcus faecalis in previously root-filled canals in a Lithuanian population*, J Endod 2000;26: 593.

Peciuliene V, Reynaud AH, Balciuniene I, et al: *Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis*, Int Endod J 2001;34: 429.

Pecora G, De Leonardis D, Ibrahim N, et al: *The use of calcium sulphate in the surgical treatment of a 'through and through' periradicular lesion*, Int Endod J 2001;34: 189.

Pekruhn RB. *The incidence of failure following single-visit endodontic therapy*, J Endod 1986;12: 68.

Peñarrocha M, Carrillo C, Peñarrocha M, et al: *Symptoms before periapical surgery related to histologic diagnosis and postoperative healing at 12 months for 178 periapical lesions*, J Oral Maxillofac Surg 2011;69: e31.

Peñarrocha M, Martí E, García B, et al: *Relationship of periapical lesion radiologic size, apical resection, and retrograde filling with the prognosis of periapical surgery*, J Maxillofac Oral Surg 2007;65: 1526.

Peñarrocha-Diago M, Maestre-Ferrín L, Peñarrocha-Oltra D, et al: *Influence of hemostatic agents upon the outcome of periapical surgery: dressings with anesthetic and vasoconstrictor or aluminum chloride, Medicina Oral*, Patologia Oral y Cirugia Bucal 2013;18: e272.

Peñarrocha Diago M, Ortega Sánchez B, García Mira B, et al: *Evaluation of healing criteria for success after periapical surgery*, Medicina Oral, Patologia Oral y Cirugia Bucal 2008;13: 143.

Telegram: @dental_k

www.konkur.in

Penarrocha-Diago MA, Ortega-Sanchez B, Garcia-Mira B, et al: *A prospective clinical study of polycarboxylate cement in periapical surgery*, Medicina Oral, Patologia Oral y Cirugia Bucal 2012;17: e276.

Peñarrocha Diago M, Sanchis Bielsa JM, Gay Escoda C. *Periapical surgery of 31 lower molars based on the ultrasound technique and retrograde filling with silver amalgam*, Medicina Oral 2001;6: 376.

Penesis VA, Fitzgerald PI, Fayad MI, et al: *Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: a randomized controlled trial with one-year evaluation*, J Endod 2008;34: 251.

Peretz B, Yakir O, Fuks AB. *Follow up after root canal treatment of young permanent molars*, J Clin Pediatr Dent 1997;21: 237.

Persson G. *Periapical surgery of molars*, Int J Oral Surg 1982;11: 96.

Peters LB, van Winkelhoff AJ, Buijs JF, et al: *Effects of instrumentation, irrigation and dressing with calcium hydroxide on infection in pulpless teeth with periapical bone lesions*, Int Endod J 2002;35: 13.

Peters LB, Wesselink PR. *Periapical healing of endodontically treated teeth in one and two visits obturated in the presence or absence of detectable microorganisms*, Int Endod J 2002;35: 660.

Pettiette MT, Delano EO, Trope M. *Evaluation of success rate of endodontic treatment performed by students with stainless-steel K-files and nickel-titanium hand files*, J Endod 2001;27: 124.

Platt AS, Wannfors K. *The effectiveness of compomer as a root-end filling: a clinical investigation*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 508.

Telegram: @dental_k

www.konkur.in

Qudeimat MA, Barrieshi-Nusair KM, Owais AI. *Calcium hydroxide vs mineral trioxide aggregates for partial pulpotomy of permanent molars with deep caries*, Eur Arch Paediatr Dent 2007;8: 99.

Rahbaran S, Gilthorpe MS, Harrison SD, Gulabivala K. *Comparison of clinical outcome of periapical surgery in endodontic and oral surgery units of a teaching dental hospital: a retrospective study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91: 700.

Rapp EL, Brown CE Jr, Newton CW. *An analysis of success and failure of apicoectomies*, J Endod 1991;17: 508.

Reid RJ, Abbott PV, McNamara JR, et al: *A five-year study of hydron root canal fillings*, Int Endod J 1992;25: 213.

Reit C, Dahlen G. *Decision making analysis of endodontic treatment strategies in teeth with apical periodontitis*, Int Endod J 1988;21: 291.

Reit C, Molander A, Dahlen G. *The diagnostic accuracy of microbiologic root canal sampling and the influence of antimicrobial dressings*, Endod Dent Traumatol 1999;15: 278.

Rhein ML, Krasnow F, Gies WJ. *A prolonged study of the electrolytic treatment of dental focal infection: a preliminary report*, Dent Cosmos 1926;68: 971.

Ricucci D, Grondahl K, Bergenholtz G. *Periapical status of root-filled teeth exposed to the oral environment by loss of restoration or caries*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 354.

Ricucci D, Russo J, Rutberg M, et al: *A prospective cohort study of endodontic treatments of 1,369 root canals: results after 5 years*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 825.

Rocas IN, Siqueira JF Jr, Del Aguila CA, et al:

Telegram: @dental_k

www.konkur.in

Polymorphism of the CD14 and TLR4 genes and post-treatment apical periodontitis, J Endod 2014;40: 168.

Rubinstein RA, Kim S. *Long-term follow-up of cases considered healed one year after apical microsurgery*, J Endod 2002;28: 378.

Rud J, Andreasen JO. *A study of failures after endodontic surgery by radiographic, histologic and stereomicroscopic methods*, Int J Oral Surg 1972;1: 311.

Rud J, Munksgaard EC, Andreasen JO, et al: *Retrograde root filling with composite and a dentin-bonding agent. 2*, Endod Dent Traumatol 1991;7: 126.

Rud J, Rud V, Munksgaard EC. *Long-term evaluation of retrograde root filling with dentin-bonded resin composite*, J Endod 1996;22: 90.

Rud J, Rud V, Munksgaard EC. *Periapical healing of mandibular molars after root-end sealing with dentine-bonded composite*, Int Endod J 2001;34: 285.

Russo MC, Holland R, de Souza V. *Radiographic and histological evaluation of the treatment of inflamed dental pulps*, Int Endod J 1982;15: 137.

Safavi KE, Dowden WE, Langeland K. *Influence of delayed coronal permanent restoration on endodontic prognosis*, Endod Dent Traumatol 1987;3: 187.

Salehrabi R, Rotstein I. *Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study*, J Endod 2004;30: 846.

Salehrabi R, Rotstein I. *Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment*, J Endod 2010;36: 790.

Salvi GE, Siegrist Guldener BE, Amstad T, et al: *Clinical evaluation of root filled teeth restored with or without post-and-core systems in a specialist practice setting*, Int

Telegram: @dental_k

www.konkur.in

Endod J 2007;40: 209.
Santini A. *Assessment of the pulpotomy technique in human first permanent mandibular molars: use of two direct inspection criteria*, Br Dent J 1983;155: 151.
Santucci PJ. *Dycal versus Nd:YAG laser and Vitrebond for direct pulp capping in permanent teeth*, J Clin Laser Med Surg 1999;17: 69.
Sari S, Durutűrk L. *Radiographic evaluation of periapical healing of permanent teeth with periapical lesions after extrusion of AH Plus sealer*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: e54.
Sawusch RH. *Direct and indirect pulp capping with two new products*, J Am Dent Assoc 1982;104: 459.
Schwartz-Arad D, Yarom N, Lustig JP, et al: *A retrospective radiographic study of root-end surgery with amalgam and intermediate restorative material*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 472.
Selden HS. *Pulpoperiapical disease: diagnosis and healing: a clinical endodontic study*, Oral Surg 1974;27: 271.
Seltzer S, Bender IB, Turkenkopf S. *Factors affecting successful repair after root canal therapy*, J Am Dent Assoc 1963;67: 651.
Setzer FC, Boyer KR, Jeppson JR, et al: *Long-term prognosis of endodontically treated teeth: a retrospective analysis of preoperative factors in molars*, J Endod 2011;37: 21.
Shah N. *Non-surgical management of periapical lesions: a prospective study*, Oral Surg Oral Med Oral Pathol 1988;66: 365.
Shovelton DS, Friend LA, Kirk EE, et al: *The efficacy of pulp capping materials: a comparative trial*, Br Dent J 1971;130: 385.

Telegram: @dental_k

www.konkur.in

Shuping GB, Orstavik D, Sigurdsson A, Trope M. *Reduction of intracanal bacteria using nickel-titanium rotary instrumentation and various medications*, J Endod 2000;26: 751.

Siqueira JF Jr, Guimaraes-Pinto T, Rocas IN. *Effects of chemomechanical preparation with 2.5% sodium hypochlorite and intracanal medication with calcium hydroxide on cultivable bacteria in infected root canals*, J Endod 2007;33: 800.

Siqueira JF Jr, Magalhaes KM, Rocas IN. *Bacterial reduction in infected root canals treated with 2.5% NaOCl as an irrigant and calcium hydroxide/camphorated paramonochlorophenol paste as an intracanal dressing*, J Endod 2007;33: 667.

Siqueira JF Jr, Rocas IN, Paiva SS, et al: *Bacteriologic investigation of the effects of sodium hypochlorite and chlorhexidine during the endodontic treatment of teeth with apical periodontitis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104: 122.

Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Relationship between Fcgamma receptor and interleukin-1 gene polymorphisms and post-treatment apical periodontitis*, J Endod 2009;35: 1186.

Siqueira JF Jr, Rocas IN, Provenzano JC, et al: *Polymorphism of the FcgammaRIIIa gene and post-treatment apical periodontitis*, J Endod 2011;37: 1345.

Siqueira JF Jr, Rôças IN, Riche FN, et al: *Clinical outcome of the endodontic treatment of teeth with apical periodontitis using an antimicrobial protocol*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 757.

Sjogren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic*

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treatment of teeth with apical periodontitis, Int Endod J 1997;30: 297.

Sjogren U, Figdor D, Spangberg L, et al: *The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing*, Int Endod J 1991;24: 119.

Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498.

Sjogren U, Sundqvist G. *Bacteriologic evaluation of ultrasonic root canal instrumentation*, Oral Surg Oral Med Oral Pathol 1987;63: 366.

Skupien JA, Opdam N, Winnen R, et al: *A practice-based study on the survival of restored endodontically treated teeth*, J Endod 2013;39: 1335.

Smith CS, Setchell DJ, Harty FJ. *Factors influencing the success of conventional root canal therapy—a five-year retrospective study*, Int Endod J 1993;26: 321.

Soltanoff W. *A comparative study of the single-visit and the multiple-visit endodontic procedure*, J Endod 1978;4: 278.

Song M, Jung IY, Lee SJ, et al: *Prognostic factors for clinical outcomes in endodontic microsurgery: a retrospective study*, J Endod 2011;37: 927- Erratum in *J Endod* 37:1595, 2011a.

Song M, Kim E. *A prospective randomized controlled study of mineral trioxide aggregate and super ethoxy–benzoic acid as root-end filling materials in endodontic microsurgery*, J Endod 2012;38: 875.

Song M, Kim SG, Lee SJ, et al: *Prognostic factors of clinical outcomes in endodontic microsurgery: a prospective study*, J Endod 2013;39: 1491.

Song M, Kim SG, Shin SJ, et al: *The influence of bone tissue deficiency on the outcome of endodontic microsurgery: a*

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www.konkur.in

prospective study, J Endod 2013;39: 1341.
Song M, Nam T, Shin SJ, et al: *Comparison of clinical outcomes of endodontic microsurgery: 1 year versus long-term follow-up*, J Endod 2014;40: 490.
Song M, Shin SJ, Kim E. *Outcomes of endodontic micro-resurgery: a prospective clinical study*, J Endod 2011;37: 316.
Stewart GG, Cobe H, Rappaport H. *Study of new medicament in chemomechanical preparation of infected root canals*, J Am Dent Assoc 1961;63: 33.
Stoll R, Betke K, Stachniss V. *The influence of different factors on the survival of root canal fillings: a 10-year retrospective study*, J Endod 2005;31: 783.
Storms JL. *Factors that influence the success of endodontic treatment*, J Can Dent Assoc (Tor) 1969;35: 83.
Strindberg LZ. Long-term followup of traumatized incisors treated by partial pulpotomy 1956; Mauritzon.
Sumi Y, Hattori H, Hayashi K, et al: *Ultrasonic root-end preparation: clinical and radiographic evaluation of results*, J Oral Maxillofac Surg 1996;54: 590.
Sundqvist G, Figdor D, Persson S, et al: *Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 86.
Swartz DB, Skidmore AE, Griffin JA. *Twenty years of endodontic success and failure*, J Endod 1983;9: 198.
Tan L, Chen NN, Poon CY, et al: *Survival of root filled cracked teeth in a tertiary institution*, Int Endod J 2006;39: 886.
Taschieri S, Corbella S, Tsesis I, et al: *Effect of guided tissue regeneration on the outcome of surgical endodontic treatment of through-and-through lesions: a retrospective*

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www.konkur.in

study at 4-year follow-up, Oral Maxillofac Surg 2011;15: 153.

Taschieri S, Del Fabbro M, Testori T, et al: *Endodontic surgery with ultrasonic retrotips: one-year follow-up*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100: 380.

Taschieri S, Del Fabbro M, Testori T, et al: *Endodontic surgery using 2 different magnification devices: preliminary results of a randomized controlled study*, J Oral Maxillofac Surg 2006;64: 235.

Taschieri S, Del Fabbro M, Testori T, et al: *Efficacy of guided tissue regeneration in the management of through-and-through lesions following surgical endodontics: a preliminary study*, Int J Periodontics Restorative Dent 2008;28: 265.

Taschieri S, Del Fabbro M, Testori T, et al: *Endoscopic periradicular surgery: a prospective clinical study*, Br J Oral Maxillofac Surg 2007;45: 242.

Taschieri S, Del Fabbro M, Testori T, et al: *Endodontic reoperation using an endoscope and microsurgical instruments: one year follow-up*, Br J Oral Maxillofac Surg 2007;45: 582.

Taschieri S, Del Fabbro M, Testori T, et al: *Efficacy of xenogeneic bone grafting with guided tissue regeneration in the management of bone defects after surgical endodontics*, J Oral Maxillofac Surg 2007;65: 1121.

Taschieri S, Del Fabbro M, Testori T, et al: *Microscope versus endoscope in root-end management: a randomized controlled study*, Int J Oral Maxillofac Surg 2008;37: 1022- doi:10.1016/j.ijom.2008.07.001.

Taschieri S, Machtou P, Rosano G, et al: *The influence of previous non-surgical re-treatment on the outcome of*

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endodontic surgery, Minerva Stomatol 2010;59: 625.
Taschieri S, Weinstein T, Tsesis I, et al: *Magnifying loupes versus surgical microscope in endodontic surgery: a four-year retrospective study*, Aust Endod J 2013;39: 78.
Teixeira LS, Demarco FF, Coppola MC, et al: *Clinical and radiographic evaluation of pulpotomies performed under intrapulpal injection of anaesthetic solution*, Int Endod J 2001;34: 440.
Tervit C, Paquette L, Torneck CD, et al: *Proportion of healed teeth with apical periodontitis medicated with two percent chlorhexidine gluconate liquid: a case-series study*, J Endod 2009;35: 1182.
Testori T, Capelli M, Milani S, et al: *Success and failure in periradicular surgery: a longitudinal retrospective analysis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 493.
Tilashalski KR, Gilbert GH, Boykin MJ, et al: *Root canal treatment in a population-based adult sample: status of teeth after endodontic treatment*, J Endod 2004;30: 577.
Tobón SI, Arismendi JA, Marín ML, et al: *Comparison between a conventional technique and two bone regeneration techniques in periradicular surgery*, Int Endod J 2002;35: 635.
Trope M, Delano EO, Orstavik D. *Endodontic treatment of teeth with apical periodontitis: single vs. multivisit treatment*, J Endod 1999;25: 345.
Tsesis I, Rosen E, Schwartz-Arad D, et al: *Retrospective evaluation of surgical endodontic treatment: traditional versus modern technique*, J Endod 2006;32: 412.
Vallecillo Capilla M, Muñoz Soto E, Reyes Botella C, et al: *Periapical surgery of 29 teeth: a comparison of conventional technique, microsaw and ultrasound*, Med

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www.konkur.in

Oral 2002;7: 46-50.
Vianna ME, Horz HP, Conrads G, et al: *Effect of root canal procedures on endotoxins and endodontic pathogens*, Oral Microbiol Immunol 2007;22: 411.
Villa-Machado PA, Botero-Ramírez X, Tobón-Arroyave SI. *Retrospective follow-up assessment of prognostic variables associated with the outcome of periradicular surgery*, Int Endod J 2013;46: 1063.
von Arx T, Gerber C, Hardt N. *Periradicular surgery of molars: a prospective clinical study with a one-year follow-up*, Int Endod J 2001;34: 520.
von Arx T, Jensen SS, Hanni S. *Clinical and radiographic assessment of various predictors for healing outcome 1 year after periapical surgery*, J Endod 2007;33: 123.
von Arx T, Jensen SS, Hanni S, et al: *Five-year longitudinal assessment of the prognosis of apical microsurgery*, J Endod 2012;38: 570.
von Arx T, Kurt B. *Root-end cavity preparation after apicoectomy using a new type of sonic and diamond-surfaced retrotip: a 1-year follow-up study*, J Oral Maxillofac Surg 1999;57: 656.
Von Arx T, Peñarrocha M, Jensen S. *Prognostic factors in apical surgery with root-end filling: a meta-analysis*, J Endod 2010;36: 957.
Waikakul A, Punwutikorn J. *Clinical study of retrograde filling with gold leaf: comparison with amalgam*, Oral Surg Oral Med Oral Pathol 1991;71: 228.
Wälivaara DÅ, Abrahamsson P, Fogelin M, Isaksson S. *Super-EBA and IRM as root-end fillings in periapical surgery with ultrasonic preparation: a prospective randomized clinical study of 206 consecutive teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112:

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www.konkur.in

258.
Wälivaara DA, Abrahamsson P, Isaksson S, et al: *Prospective study of periapically infected teeth treated with periapical surgery including ultrasonic preparation and retrograde intermediate restorative material root-end fillings*, J Oral Maxillofac Surg 2007;65: 931.
Wälivaara DA, Abrahamsson P, Sämfors KA, et al: *Periapical surgery using ultrasonic preparation and thermoplasticized gutta-percha with AH Plus sealer or IRM as retrograde root-end fillings in 160 consecutive teeth: a prospective randomized clinical study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: 784.
Waly NG. *A five-year comparative study of calcium hydroxide-glutaraldehyde pulpotomies versus calcium hydroxide pulpotomies in young permanent molars*, Egypt Dent J 1995;41: 993.
Wang CH, Chueh LH, Chen SC, et al: *Impact of diabetes mellitus, hypertension, and coronary artery disease on tooth extraction after nonsurgical endodontic treatment*, J Endod 2011;37: 1.
Wang CS, Arnold RR, Trope M, et al: *Clinical efficiency of 2% chlorhexidine gel in reducing intracanal bacteria*, J Endod 2007;33: 1283.
Weiger R, Rosendahl R, Löst C. *Influence of calcium hydroxide intracanal dressings on the prognosis of teeth with endodontically induced periapical lesions*, Int Endod J 2000;33: 219.
Weiss M. *Pulp capping in older patients*, N Y State Dent J 1966;32: 451.
Werts R. *Endodontic treatment: a five-year follow-up*, Dent Survey 1975;51: 29.
Witherspoon DE, Small JC, Harris GZ. *Mineral trioxide*

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aggregate pulpotomies: a case series outcomes assessment, J Am Dent Assoc 2006;137: 610.

Witherspoon DE, Small JC, Regan JD, et al: *Retrospective analysis of open apex teeth obturated with mineral trioxide aggregate*, J Endod 2008;34: 1171.

Xavier AC, Martinho FC, Chung A., et al: *One-visit versus two-visit root canal treatment: effectiveness in the removal of endotoxins and cultivable bacteria*, J Endod 2013;39: 959.

Yared GM, Dagher FE. *Influence of apical enlargement on bacterial infection during treatment of apical periodontitis*, J Endod 1994;20: 535.

Zeldow BI, Ingle JI. *Correlation of the positive culture to the prognosis of endodontically treated teeth: a clinical study*, J Am Dent Assoc 1963;66: 9.

Zmener O, Pameijer CH. *Clinical and radiographical evaluation of a resin-based root canal sealer: a 5-year follow-up*, J Endod 2007;33: 676.

Zuolo ML, Ferreira MO, Gutmann JL. *Prognosis in periradicular surgery: a clinical prospective study*, Int Endod J 2000;33: 91.

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18: Root resorption

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CHAPTER OUTLINE

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Dental resorption is defined as the loss of dental hard tissues as a result of clastic activities.[84] Root resorption in the primary dentition is indeed a physiologic process except if it occurs prematurely.[21,22] It is not yet completely understood how this process occurs, although it is believed that it is regulated by cytokines and transcription factors, which are also involved in bone remodeling.[54,125] On the other hand, resorption of permanent dentition is a pathologic process that, if left untreated, may result in premature loss of the affected teeth.

Depending on its location in relation to root surface, root resorption may be classified as external and internal.[4,107] Internal root resorption (IRR) was reported as early as 1830.[18] The etiology and pathogenesis of IRR have not been completely demonstrated.[64,88] Diagnosis of IRR is often a concern to the clinician because it is often confused with external cervical resorption (ECR). An inaccurate diagnosis may result in inappropriate treatment in certain cases.[51,52,83,87,88]

General histologic features

Bone resorption is a process regulated by motile, multinucleated giant cells called osteoclasts. They are formed by the fusion of mononuclear precursor

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cells of the monocyte-macrophage lineage derived from the spleen or bone marrow; osteoblasts and osteocytes, on the other hand, are derived from skeletal precursor cells.[72,103] Osteoclasts are recruited to the site of injury or irritation by the release of many proinflammatory cytokines. To perform their function, osteoclasts must attach themselves to the bone surface. On contact with mineralized extracellular matrices, the actin cytoskeleton of an actively resorbing osteoclast is reorganized to produce an organelle-free zone of sealing cytoplasm (clear zone) associated with the osteoclast's cell membrane; this enables the osteoclast to achieve intimate contact with the hard tissue surface.[91] The clear zone surrounds a series of fingerlike projections (podosomes) of cell membrane, known as the *ruffled border*, beneath which bone resorption occurs. The resorptive area within the clear zone therefore is isolated from the extracellular environment, creating an acidic microenvironment for the resorption of hard tissues.

Odontoclast cells are morphologically similar to osteoclasts but they are responsible of resorbing dental hard tissue (Fig. 18.1).[44] Odontoclasts differ from osteoclasts in that they are smaller, have fewer nuclei, and have smaller sealing zones, possibly as result of differences in their respective resorption substrata.[66] Osteoclasts and odontoclasts resorb their target tissues in a similar manner.[91] The two cells have similar enzymatic properties,[79] show similar cytologic features, and create resorption depressions, termed *Howship lacunae*, on the surface of the mineralized tissues (see Fig. 18.1).[91] Odontoclasts are polarized in relation to dental tissue and have a ruffled border, located inside a clear zone, that is in intimate contact with their dental substratum.[60,91]

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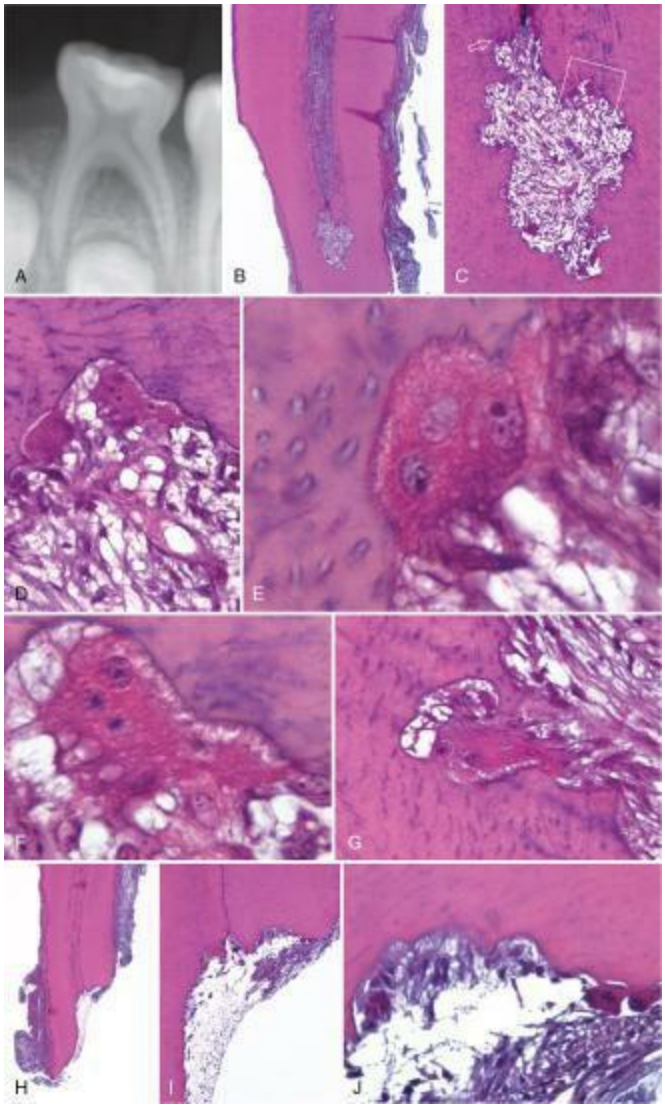

FIG. 18.1 Light microscopy images of a deciduous tooth showing both physiologic and pathologic (inflammatory) resorption. **A,** Tooth 7.5 in a 9-year-old boy. Extensive exposure of a mesial pulp horn occurred during caries excavation, and pulp capping was performed. The patient was brought to the dental office 4 months after the treatment because of severe spontaneous pain. The radiograph shows radiolucencies on both apices and interradicularly. The parents did not accept any treatment and requested extraction. **B,** Longitudinal section taken on a mesiodistal plane, passing approximately at the center of the mesial root. (Hematoxylin-eosin [H&E] stain; original magnification ×25.) **C,** Detail of the resorption area apically in **B** (original magnification ×100). **D,** Magnification of the area demarcated by the rectangle in **C.** Two odontoclasts are present in an area of dentin being resorbed, surrounded by fibroblasts and inflammatory cells (original magnification ×400). **E,** High-power view of the area indicated by the *arrow* in **C.** An odontoclast is in close contact with the dentin in a Howship lacuna. Its cytoplasm appears vacuolated and shows a more intense staining

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reaction than the cytoplasm of the adjacent cells (original magnification ×1000). **F,** High-power view of the upper odontoclast in **D.** The characteristic ruffled border can be distinguished (original magnification ×1000). **G,** Howship lacuna with an odontoclast in another area (original magnification ×400). **H–J,** Progressive magnification of the mesial root apex. The resorption process may be physiologic at this level (original magnification ×16, ×100, and ×400).

Set of radiograph and stained micrographs marked A through J shows the different views of inflammatory cells along with vesicles in connective tissue at the mesial root apex.

At high-power magnification, the cytoplasm of the odontoclasts appears vacuolated and shows a more intense staining reaction than the cytoplasm of the adjacent cells. Where odontoclasts are in contact with the tooth surface, the characteristic brush border can be seen, but the hematoxyphilic zone lining the lacunae is absent.[44] When odontoclasts are observed with an electron microscope, the most striking features are the large number of mitochondria and vacuoles in the cytoplasm and the scarcity of endoplasmic reticulum. An abundance of cytoplasmic ribosomes can be seen. The nucleoli are fairly large and centrally located in the nucleus. Close to the nucleus, the Golgi apparatus appears as a narrow zone of fine canals. Where the odontoclasts are in contact with the tooth surface, the ruffled border (corresponding to the brush border) may be observed with the light microscope. The ruffled border consists of cytoplasmic folds that create a system of canals extending 2 to 3 μm into the cytoplasm. In undecalcified sections, mineral crystals could be observed in these canals.[44]

Although both mononuclear dendritic cells and multinucleated osteoclasts share a common hematopoietic lineage, they have previously been accounted for as immunologic defense cells. Recent studies have indicated that immature dendritic cells also function as osteoclast precursors and have the potential to transdifferentiate into osteoclasts.[104] Because dendritic cells are present in the dental pulp, it is possible they also may function as precursors of odontoclasts.

From a molecular signaling perspective, the osteoprotegerin (OPG)/receptor activator of nuclear factor κB ligand (RANKL)/RANK transcription factor system[23] that controls clastic functions during bone remodeling has also been identified in root resorption.[111] The system is

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responsible for the differentiation of clastic cells from their precursors by means of complex cell-cell interactions with osteoblastic stromal cells. Similar to periodontal ligament (PDL) cells that are responsible for external root resorption,[114] the human dental pulp has recently been shown to express OPG and RANKL messenger ribonucleic acids (mRNAs).[112] OPG, a member of the tumor necrosis factor superfamily, has the ability to inhibit clastic functions by acting as a decoy receptor that binds to RANKL and reduces its affinity for RANK receptors on the surface of clastic precursors. This results in inhibition of the regulation of clastic cell differentiation. Thus, it is possible that the OPG/RANKL/RANK system may be actively involved in the differentiation of odontoclasts during root resorption.

We know that osteoclasts do not adhere to nonmineralized collagen matrices.[108] It has been suggested that the presence of a noncollagenous, organic component in dentin (odontoblast layer and predentin) prevents (internal) resorption of the root canal wall, and precementum prevents (external) resorption of the external root surface.[117,119] Similar to osteoclasts, odontoclasts may bind to extracellular proteins containing the arginine-glycine–aspartic acid (RGD) sequence of amino acids by means of integrins.[100] The latter are specific surface adhesion glycoprotein membrane receptors containing different α and β subunits. In particular, $\alpha_v\beta_3$ integrin plays a key role in the adhesion of clastic cells.[76] Extracellular matrix proteins containing the RGD peptide sequence, particularly osteopontin, present on the surface of mineralized tissues and serve as binding sites of clastic cells.[58] The osteopontin molecule contains different domains, with one domain binding to apatites in the denuded dentin and another domain binding to integrin receptors in the plasma membranes of clastic cells. Thus, osteopontin serves as a linker molecule that optimizes the attachment of a clastic cell to mineralized tissues, mediating the rearrangement of its actin cytoskeleton.[30] It has been speculated that the lack of RGD peptides in predentin reduces the binding of odontoclasts, thereby conferring resistance of the canal walls to IRR.

Once the clastic cells have established contact with the cementum, any subsequent resorption is self-limiting unless the bound cells are subject to continued stimulation.[4] Therefore, in addition to the precipitating events discussed previously, progressive root resorption requires a source of

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stimulation for the resorbing cells. Stimulating factors vary and are related to the site and type of resorption, in addition to the cause of the predisposing damage to the predentin or precementum. Examples include persistent pressure and forces associated with continued orthodontic treatment; persistent impacted teeth; untreated cysts, granulomas, and tumors; endodontic inflammation and/or infection; and periodontal inflammation and/or infection.[45]

External inflammatory resorption

Introduction

External inflammatory resorption (EIR) affects the outer surface of the root and is usually a consequence to dental luxation[2] and avulsion[13] injuries. It is a progressive condition with a potentially precipitous onset, and it is capable of advancing rapidly, such that an entire root surface may be resorbed within a few months if the tooth is left untreated.[5,13] It also affects teeth diagnosed with chronic periapical periodontitis (Figs. 18.2–18.5).[62]

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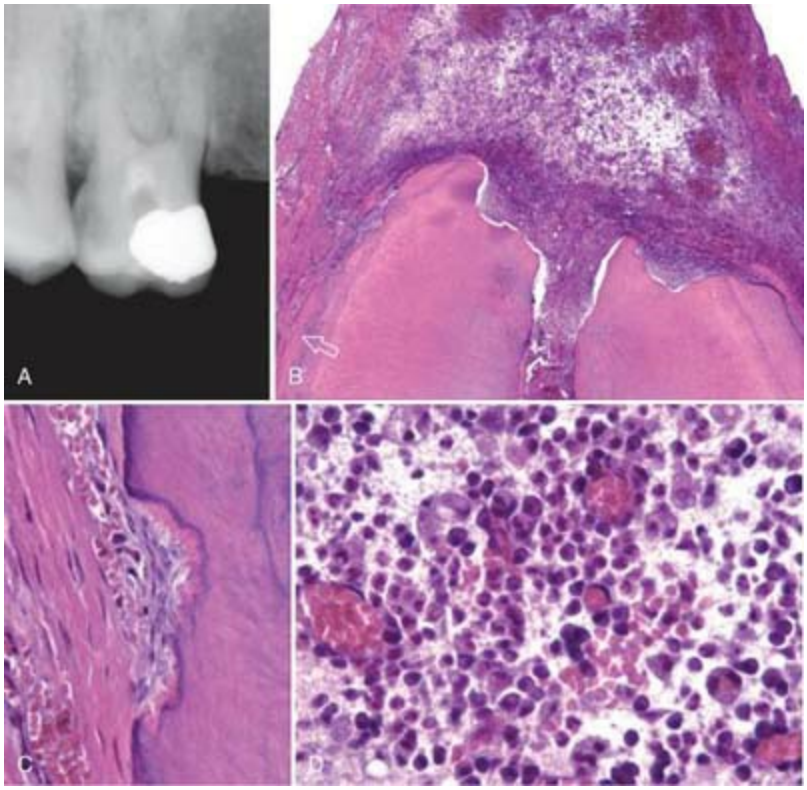

FIG. 18.2 External apical inflammatory resorption. **A,** Maxillary first molar in a 46-year-old female patient that was causing severe pain. The radiographs revealed material in the pulp chamber, a large distal caries, and apical radiolucencies. The tooth was extracted. **B,** Palatal root apex extracted with the pathologic tissue attached. Note the ingrowth of granulation tissue into the foramen and the massive resorption of the apical profile. Also note the resorption of lacunae on the left radicular profile. (H&E stain; original magnification ×25.) **C,** High magnification of the area of the external radicular profile indicated by the *arrow* in **B.** A resorption lacuna can be seen in the cementum. (H&E stain; original magnification ×400.) **D,** View from the center of the apical periodontitis lesion showing severe concentration of chronic inflammatory cells (mostly plasma cells) (original magnification ×400).

A) Radiograph shows a large nearly circular patch on one side of the crown in first molar of the upper jaw. Beside this, the molar has less-density patch on the other side of crown, at the center, and close to the root apices.

B) Stained micrograph shows an arrow pointing toward two tissue protuberances near the root apex on left and inflammation with oval cells at the tip.

C) Stained micrograph shows enlarged view of the protuberance that has connective tissue and different-sized dark cells.

D) Stained micrograph shows tissue having multi-chamber oval and circular

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cells

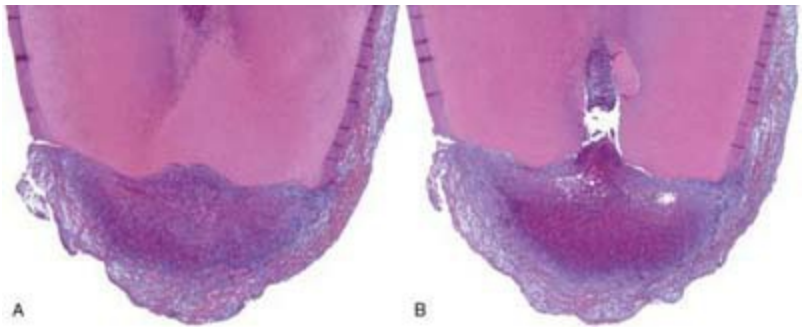

FIG. 18.3 External apical inflammatory resorption. **A,** Mandibular second premolar extracted with the periapical lesion attached. This section, which did not pass through the canal, shows extensive apical resorption. (H&E stain; original magnification ×25.) **B,** Section taken approximately 120 sections away encompasses the apical foramen. In addition to resorption, the opposite phenomenon can be observed; that is, a large calcification partly embedded in the right apical dentin wall (original magnification ×25).

Stained micrograph A shows the inflammation causing depression at root surface.

Stained micrograph B shows the inflammatory cells extended inward of root with white deposition and a nearly oval cyst.

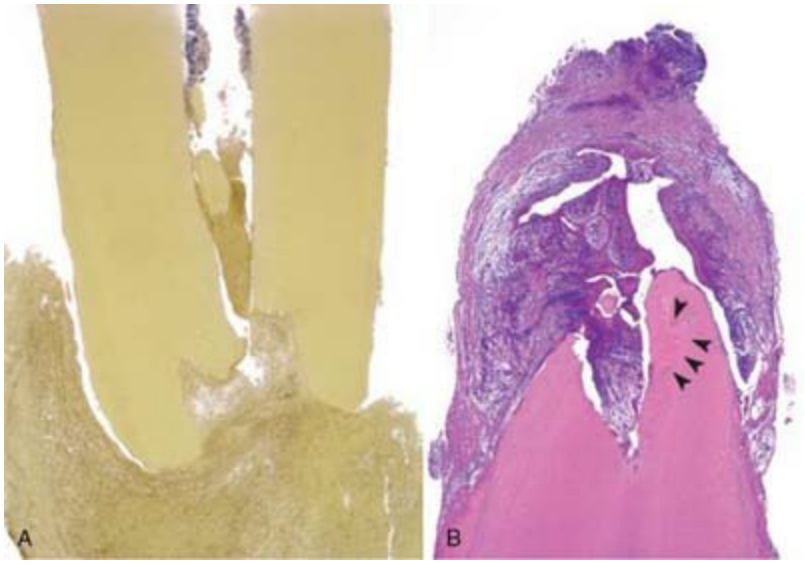

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FIG. 18.4 External apical inflammatory resorption. **A,** Mesial root of a mandibular first molar extracted with a periapical lesion attached. This section passes through the canal and the foramen. Extensive resorption of the foramen has occurred, and a calcification can be seen more coronally. Note that the tissue in the apical canal and in the resorptive defect appears structured and continuous with the apical periodontitis lesion. A thick biofilm can be discerned layering the root canal walls more coronally. (Taylor's modified Brown & Brenn stain; original magnification ×25.) **B,** Palatal root of a maxillary first molar extracted with the periapical lesion attached. A cyst cavity is present in the body of the lesion. Resorption of the foramen is present, and a portion of the right dentin wall has been replaced by bone *(arrowheads).* (H&E stain; original magnification ×25.)

A) Stained micrograph shows root tip of a molar with the absorption of surrounding inflammatory tissue in the root canal. The canal also has white deposition along with granular cells and an oval patch along the canal walls.

B) Stained micrograph shows the resorption of tissue in the root canal along with a lesion near the root apex.

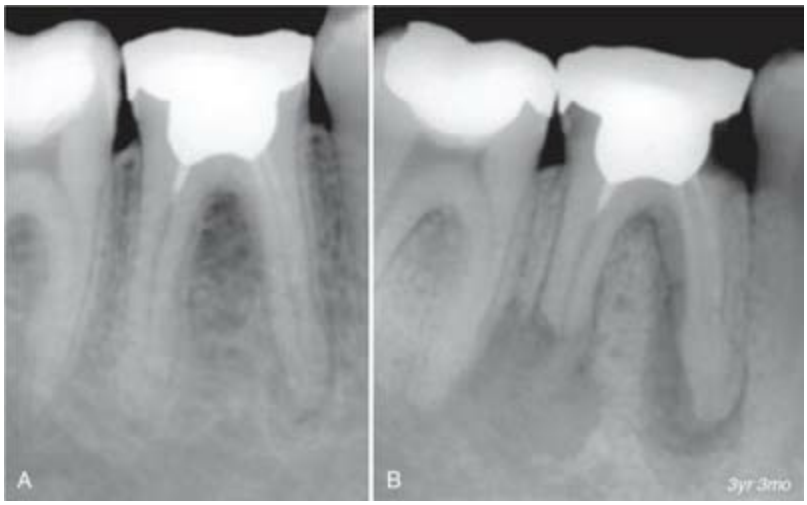

FIG. 18.5 Apical resorption. **A,** 22-year-old male patient complained of pain on chewing. This was caused by a molar that had been treated endodontically 1 year earlier. A radiograph showed obturation material only at the root canal orifices and a periapical radiolucency on the mesial root. Endodontic retreatment was recommended, but the patient declined this treatment. **B,** The patient returned 3 years and 3 months later because of an abscess that was causing severe pain. A radiograph disclosed destructive mesial caries and large periapical radiolucencies on both roots, along with resorption of the whole apical third of the distal root.

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A) Radiograph shows porous tissue at the bifurcation of root system in a tooth.

B) Radiograph taken after 3 years 3 months shows large radiolucent patches extending in and along the root apices.

After luxation injuries, EIR ranges from almost 5%[2] to 18%.[32] It affects 30% of replanted avulsed teeth.[10,11] EIR is the most common form of external resorption root resorption after luxation and avulsion injuries.[32]

Treatment of EIR should be carried out as soon as it is diagnosed, and it is mainly based on effective removal of the infected necrotic pulp.[37] The earlier the diagnosis, management, and treatment of EIR, the better the prognosis of the affected tooth.[35]

The diagnosis of EIR in clinical situations is based solely on the radiographic demonstration of the process.[3] In some cases, the initial radiographic signs of EIR can be visualized as early as 2 weeks after replantation of avulsed teeth.[13] However, the limitations of conventional radiographic imaging in dentistry have been well reported. The diagnostic yield of radiographs is reduced by adjacent anatomic noise,[19,50,61,94] geometric distortion,[50] and compression of three-dimensional (3D) structures onto a two-dimensional (2D) shadowgraph.[31,82,116] These limitations may result in late diagnosis of EIR after dental trauma.

In a prospective study including up to 637 permanent luxated teeth in 400 patients for 10 years, Andreasen and Vestergaard Pedersen[2] reported that no cases of EIR after concussion injuries and only one case after subluxation injuries, which represented only 0.5% of the total number of subluxated teeth. Six percent of extrusively luxated teeth and 3% of laterally luxated teeth developed EIR. However, EIR was a healing complication in 38% of intrusive luxation injuries.[2] Crona-Larsson et al.[32] also reported a higher prevalence of EIR after more severe luxation injuries, with 60% of extrusively luxated teeth and 22% of intruded teeth affected by the complication. They reported EIR to be a much more frequent occurrence after lateral luxation (16.7%) and subluxation injuries (3.8%) relative to the findings of Andreasen and Vestergaard Pedersen.

Etiology and pathogenesis of external inflammatory

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resorption

Normally, permanent teeth are resistant to resorption.[118,119,120] It is generally accepted that odontoclasts do not bind to the nonmineralized layers covering the external root surface and the root canal wall (i.e., the precementum and predentin, respectively).[4,53,118,119,120] Similarly, it has been shown that, between episodes of physiologic resorption, the surface of bone is impervious to osteoclastic activity because it is covered by a layer of nonmineralized collagen fibrils, to which the osteoclast is incapable of binding.[29]

Traumatic dental injuries (e.g., intrusion, lateral luxation, and avulsion) and subsequent replantation often result in contusion injuries to the PDL.[4] Damage to the precementum, with a resultant breach in its integrity, is the precipitating factor in all types of external resorption.[4,6] In the subsequent wound healing process, necrotic PDL tissue remnants are excavated and removed by macrophages and osteoclasts.[39] A critical factor is that the precementum may be stripped from the root surface during the injury and the damaged cementum and bone may also be phagocytosed, resulting in exposure of the underlying dentine to osteoclastic and odontoclastic activity.[39] External root resorption may ensue, but the precise type depends on the severity of the initial injury, the stage of root development, and the pulp status of the affected tooth.[4,6] Once the clastic cells have established contact with the cementum or dentine, any subsequent resorption is self-limiting unless the cells are subject to continued stimulation.[9] As already mentioned, odontoclasts do not adhere to the nonmineralized layers covering the external root surface.[4,53,118,119,120] Therefore, damage to the precementum with a resultant breach in the integrity of these layers is the precipitating factor in all types of ERR.[4,6] Damage to the precementum and underlying tooth structure allow circulating odontoclasts to bind to the underlying mineralized dentine and cementum.[118]

The pathogenesis of EIR can be explained as follows. Contusion injuries to the PDL, after a traumatic dental injury (TDI) involving the periodontal structures, initiate wound healing, during which osteoclasts and macrophages are attracted to the site of the injury to remove the damaged tissue. The initial injury causes a breach in the integrity of the protective precementum. This permits odontoclasts to bind to and resorb the underlying mineralized

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cementum and dentine in a manner similar to the development of surface resorption.[6] However, EIR differs from surface resorption in that it is a progressive event that relies on microbial stimulation from the infected necrotic pulp of the affected tooth for its progression.[9] Therefore, it is more commonly associated with tooth avulsions[10,11] and moderate to severe luxation injuries, which have the potential to compromise pulp vitality.[2] If the strength of the initial osteoclast attack is sufficient to expose patent dentinal tubules beneath the cementum, a communication is created between the pulp space and the external root surface and adjacent periodontal tissues.[9,14] Microbes and/or their toxins (e.g., lipopolysaccharide, muramyl dipeptide, and lipoteichoic acid),[15] located in the root canal and the dentinal tubules, diffuse through the tubules and directly stimulate the resorbing osteoclasts. The resorption process intensifies and accelerates.[7] The resorbed mineralized tissue is replaced by granulation tissue, which ultimately invades the pulp space if the process continues.[15]

Pressure on the root surface during orthodontic treatment[63] and from impacted teeth,[124] cysts,[106] and tumors[65] may also denude the protective precementum from the root surface and therefore initiate ERR.

Histologic appearance

The histologic appearance of EIR is characterized by saucer- or bowl-shaped areas of resorption in both the cementum and dentine, with concomitant inflammation in the adjacent periodontal membrane. Howship lacunae are a common feature of the resorption cavities, and histologic sections show that the lacunae sometimes are occupied by odontoclasts. The inflammatory reaction in the periodontal membrane appears intense and consists of a mixed-cell infiltrate that includes plasma cells, lymphocytes, and polymorphonuclear leucocytes in a granulation tissue matrix. Proliferation of capillaries in the areas of inflammation is also a feature.[14] EIR can be identified histologically 1 week after experimental replantation of teeth.[8]

Clinical features

The tooth in question will not respond to sensibility testing and in advanced cases may be associated with signs of pulpitis or apical periodontitis such as

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discoloration, sinus tract, and/or tenderness to percussion and/or palpation.

Radiographic features

As mentioned, diagnosis of EIR is based solely on radiographic demonstration of the process.[3,13] EIR is characterized radiographically by radiolucent, concave, and sometimes ragged bowl-shaped excavations along the root surface, with corresponding and associated radiolucencies in the adjacent alveolar bone. Complete loss of the lamina dura is seen in the area of the resorption.[14] The initial radiographic signs of EIR can often be seen as early as 3 to 4 weeks after a TDI involving the periodontal tissues,[14] and if it will develop, EIR is always seen within 1 year after the injury.[7]

EIR can have a rapid onset and aggressive progression, such that complete resorption of an entire root can occur within 3 months. The diagnostic potential of a number of radiographic imaging systems has been investigated, with varying degrees of success.

Conventional intraoral radiographic imaging (digital or film based) is currently the clinical reference standard for the detection of ERR after luxation and avulsion injuries.[41,42] However, it has been well documented that this form of imaging is an inadequate method of detecting simulated ERR, especially when the cavity sizes are small.[3] Clinical studies have also demonstrated that conventional radiography grossly underestimates the extent of inflammatory root resorption.[40]

Clinical studies directly comparing the ability of intraoral radiographs and cone beam computed tomography (CBCT) to detect and diagnose EIR are limited. One clinical study reported that CBCT is superior to conventional radiography in diagnosing and determining the extent of nonspecific inflammatory resorption on root surfaces.[40] D'Addazio et al.[36] compared the ability of CBCT and periapical radiography to detect simulated external resorption cavities of about 2 mm in diameter in a human ex vivo model. Although both imaging modalities were 100% sensitive in the detection of the lesions, only CBCT could accurately assess the position of the defects on the root surface and their relationship to the root canal, even though multiple angled periapical radiographs of the test teeth were available to the examiners.

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Management

In cases were the tooth is treatable, root canal treatment will be able to help in arresting the resorption and therefore prevent further progression of the resorption. This would create an environment conducive to hard tissue repair of the damaged root surface.[33,34,37] Therefore, it is essential to initiate root canal treatment as soon as radiographic signs of EIR are identified.[35] An exception to this is replanted teeth with closed apices; in these cases, root canal treatment should be carried out 7 to 10 days after replantation, even if there are no radiographic signs of EIR.[42] The earlier the resorption is diagnosed and treated, the better the prognosis is for the affected tooth. Failure to diagnose and treat the condition may result in tooth loss.

Effective chemomechanical debridement of the root canal space is fundamental to the success of the root canal treatment and the inhibition and cessation of EIR.[34,37] In principle, the specific root canal protocol used is irrelevant, as long as the biologic objectives are met. Long-term dressing of the root canal with calcium hydroxide may be beneficial in the treatment of established EIR; however, this protocol should be used judiciously because of the associated risk of root fracture.[12]

In many cases the EIR is extensive, rendering the tooth unsalvageable and requiring extraction.

Follow-up and prognosis of external inflammatory resorption

Healing of EIR is characterized radiographically by cessation of the resorption process, resolution of the radiolucency in the adjacent bone, and reestablishment of the PDL space.[13] As mentioned previously, in untreated cases, EIR can progress so rapidly that an entire root can be resorbed within 3 months.[13,34] The prognosis is especially poor for untreated immature teeth.[13]

External cervical resorption

Introduction

ECR manifest mainly in the cervical region and it may invade root dentin in

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any direction and to a varying degree. ECR generally develops immediately apical to the epithelial attachment of the tooth. In healthy teeth with a normal periodontal attachment, this is in the tooth's cervical region, a feature that gave rise to the name. However, in teeth that have developed gingival recession and lost periodontal support and/or have developed a long junctional epithelium, the resorptive defect may arise at a more apical location.

ECR has also been referred to as invasive cervical resorption,[56] supraosseous extracanal invasive resorption,[43] peripheral inflammatory root resorption,[48] and subepithelial external root resorption.[109] The authors of this chapter prefer the term ECR because it describes the nature and location of the lesions.

Etiology and pathogenesis

The exact etiology and pathogenesis of ECR have not been fully elucidated. It is widely accepted that the resorptive process is the same for ECR as it is for any other type or resorption: a breach in the protective nonmineralized layers must exist to allow the clastic cells to bind to the underlying dentine, and the same cells must be stimulated to perpetuate the process. However, in ECR only some of the factors that predispose the root surface to clastic activity have been identified.

The anatomic profile of the cementoenamel junction (CEJ) is variable, and the junction between the enamel and the cementum in this region is not contiguous in all teeth. This may lead to exposed areas of unprotected dentin, which are vulnerable to osteoclastic activity, in the cervical region of some teeth.[78]

Mavridou et al.[69] investigated the potential predisposing factors using a two-step approach. The first step was an interview with the patient to obtain a complete information on their dental and medical history, the second step was a thorough clinical investigation of the patient with a combination of periapical radiograph and 3D CBCT scans.

Of the 347 teeth examined, 59% were multifactorial. From these multifactorial cases, 38% had 2 predisposing factors, and 17% and 3% had 3 and 4 factors, respectively.

Orthodontics was the most common appearing factor (45.7%) followed by

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trauma (28.5%), parafunctional habits (23.2%), poor oral hygiene (22.9%), malocclusion (17.5%), and extraction of neighboring tooth (14%).

Combination of orthodontics with previous dental trauma was the most common combination (17.6%); this was followed by orthodontics along with parafunctional habits (13%), orthodontics with extraction of neighboring tooth (12%), extraction of neighboring tooth along with occlusal overloading and poor oral health (5%), malocclusion with poor oral health (3%), and malocclusion along with parafunctional habits (2.7%).

Of the cases examined, ECR was located on 29% of maxillary central incisors, 14% in maxillary canines, 14% in mandibular molars, and 14% maxillary premolars. ECR was less frequently seen in mandibular canines (3%), mandibular centrals (1%), and mandibular lateral (1%).[69]

Histologic appearance

The histologic profile of ECR is similar to that of other forms of resorption, with certain unique features reflecting the invasive nature of the process.

Recently, histologic analysis has revealed that ECR lesion is a mechanism of a three-stage process. Initially, the ECR starts at the cementum apical to the gingival epithelium (i.e., the initiation stage). In the second stage of the resorption (i.e., the resorption phase), the resorptive lesion invades the tooth structure in a 3D manner towards the pulp space. However, the pulp is protected by a pericanalar resorption-resistant sheet which prevents the resorption lesion from penetrating the pulp space. This layer has a fluctuating thickness of 210 μm and consists of predentin, dentin, and occasionally bonelike tissue. At the last advanced stage (i.e., the repair stage), bonelike tissue is deposited into the resorption cavity in which repair takes place (Fig. 18.6).[70]

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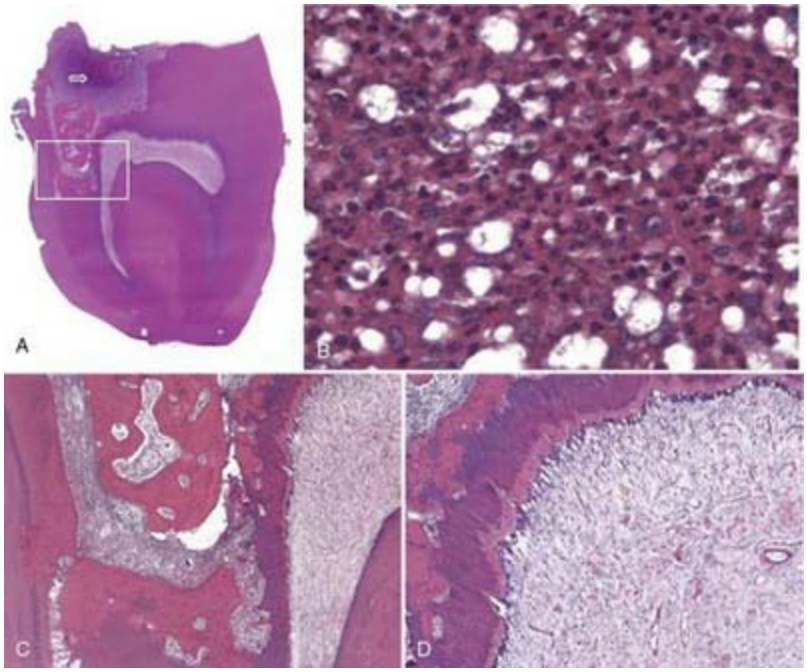

FIG. 18.6 External cervical resorption with replacement. **A,** Asymptomatic mandibular third molar. ECR was diagnosed on a radiograph taken for neighboring teeth. The tooth was extracted. Overview of the tooth (H&E stain; original magnification ×2). **B,** High power view of the resorptive area occupied by granulation tissue indicated by the *arrow* in **A**. Severe concentration of acute and chronic inflammatory cells (original magnification ×400). **C,** Detail of the area demarcated by the *rectangle* in **A**. The large area of resorbed dentin has been partly repaired with bonelike tissue. Note the thin layer of dentin remaining. No inflammatory changes can be observed in the adjacent pulp (original magnification ×25). **D,** Detail from **C**. Normal appearance of dentin, predentin, and odontoblast layer. Absence of inflammation (original magnification ×50).

Set of four stained micrographs marked A through D shows the deposition of hard tissue in place of the resorption cavity of third molar.

In a case study investigating the presence of hypoxia with ECR, Mavridou et al.[71] reported that histologic analysis showed that hypoxia does exist. Hypoxia inducible factor 1a-positive odontoblast was found within the apical area of the resorption (Figs. 18.7 and 18.8).

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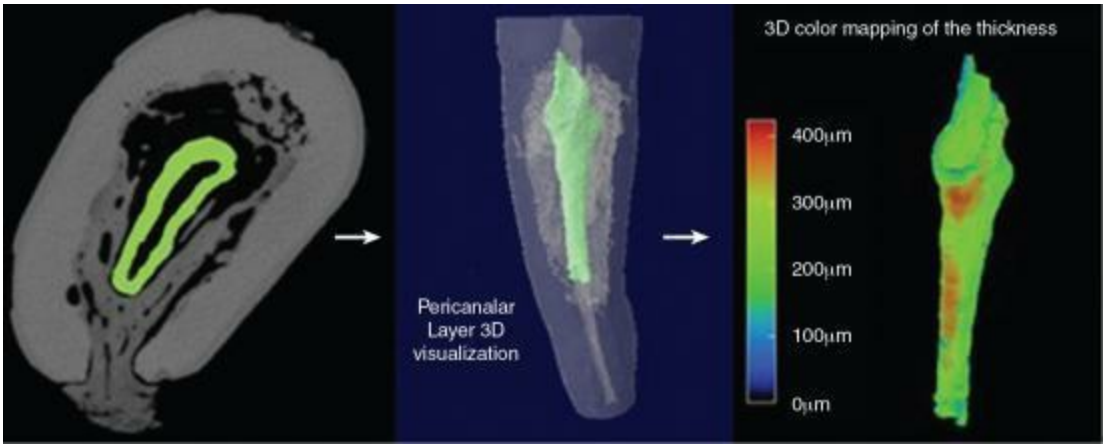

FIG. 18.7 NanoCT imaging of tooth 13 and 3D modeling using CTan, CTvol, and CTvox software, showing the thickness distribution of the pericanalar resorption resistant sheet (PRRS).

3D image shows a green pericanalar layer surrounding the root canal of tooth, followed by its 3D visualization, and color coded map of the thickness. In the color map, pericanalar layer has a thickness of 200 to 300 micrometers. However, two small red patches have thickness of 300 micrometers to 400 micrometers.

Source: (From Patel S, Mavridou A. M, Lambrechts P, Saberi N: External cervical resorption-part 1: histopathology, distribution and presentation. *Int Endod J* 51:1205–1223, 2018.)

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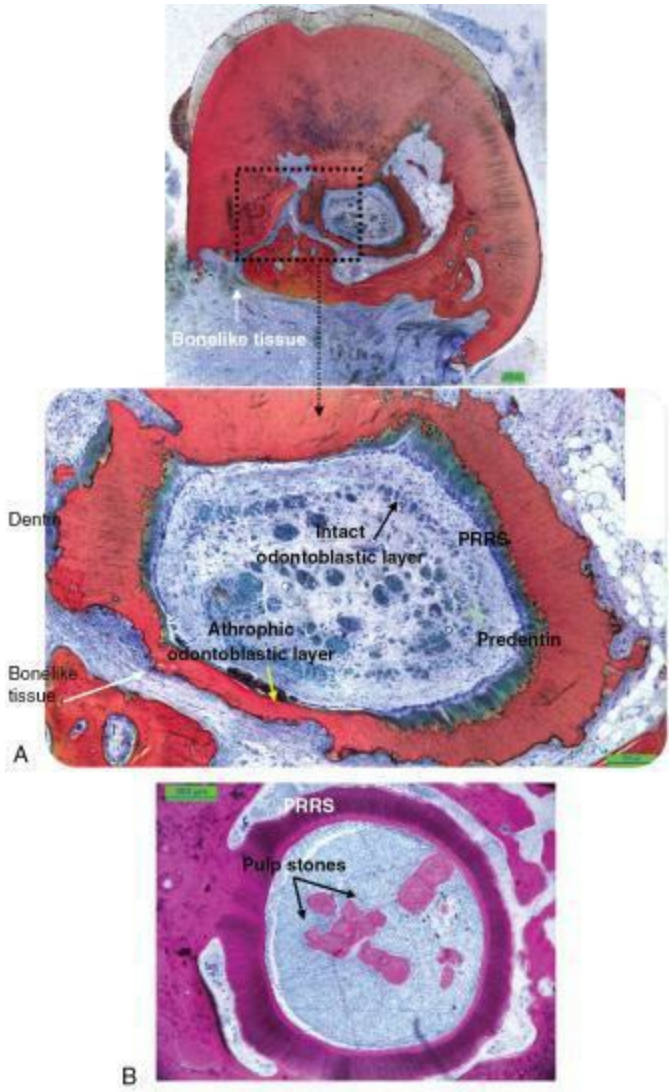

FIG. 18.8 **A,** Structure of pericanalar resorption resistant sheet *(PRRS)* and pulp tissue. PRRS is consisted of predentin, dentin and occasionally bonelike tissue. **B,** Calcification inside the pulp. Histologic staining was performed with a combination of Stevenel's blue and Von Gieson's picrofuchsin, visualizing mineralized tissue *(red)* and nonmineralized tissue *(blue-green)*.

A) Stained micrograph shows structure of PRRS and pulp tissue with labels as follows: bonelike tissue, dentin, predentin, atrophic odontoblastic layer, and intact odontoblastic layer.

B) Stained micrograph shows pulp stones in pulp chamber surrounded by PRRS.

Source: (From Patel S, Mavridou A. M, Lambrechts P, Saberi N: External cervical resorption-part 1: histopathology, distribution and presentation. *Int Endod J* 51:1205–1223, 2018.)

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Clinical features

The clinical features of ECR are variable (Figs. 18.9 and 18.10). The process is very often quiescent and asymptomatic, especially in the earlier stages, and absence of clinical signs and symptoms is very common; the diagnosis is commonly made as a result of a chance radiographic finding. A pink or red discoloration may develop at the cervical region of the tooth; when present, this often is the feature that alerts the patient or clinician to the possible existence of a problem. The discoloration is due to the fibrovascular granulation tissue occupying the resorptive defect, which has a reduced thickness of enamel and dentin at its peripheries because of the loss of hard tissue. The granulation tissue imparts a pink hue to the tooth, through the thinned enamel and dentin, in the region of the resorption.[84] The granulation tissue may perforate the enamel or dentin at the gingival margin, giving the appearance of mild gingival hyperplasia. The discoloration, sometimes referred to as a “pink spot,” can be quite subtle and is often a chance finding by the patient, the dentist or, increasingly, the dental hygienist. However, it is a relatively rare feature of ECR. Furthermore, it must occur at a site where it is readily identifiable (e.g., labial surface of an anterior tooth) to be noticed. Loss of periodontal attachment may occur in the region of the resorption, and probing of the resorptive defect or the associated periodontal pocket causes the granulation tissue to bleed profusely.[87]

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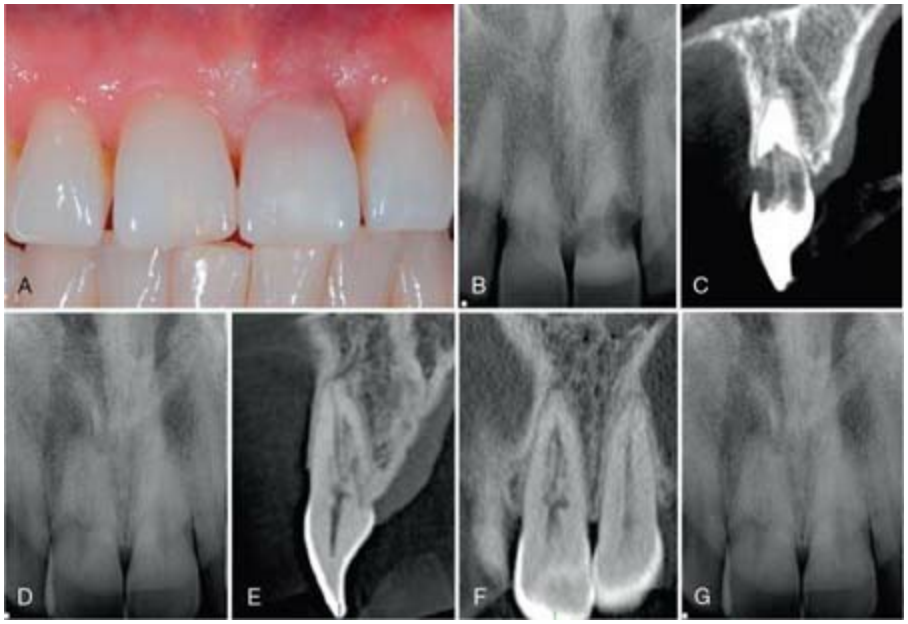

FIG. 18.9 External cervical resorption (ECR). **A,** A 55-year-old female patient presented with an asymptomatic "pink spot." She had no history of any predisposing factors. **B,** A periapical radiograph revealed radiolucent defects on the proximal aspects of the upper left central incisor; note the ragged borders. **C,** A reconstructed coronal cone beam computed tomography (CBCT) slice reveals the true extent of the ECR lesion. Note that the root canal wall appears to be intact. Inhibitory factors in the root canal wall/odontoblastic prevented the ECR lesion from penetrating the root canal. **D,** A 41-year-old male patient presented as a new patient. Routine radiographic examination revealed a poorly defined periapical radiolucency in the root of the upper right central incisor. The appearance is suggestive of ECR; the patient had orthodontic treatment in his early teens and remembered "knocking the tooth" at least twice when he was very young. Reconstructed sagittal **(E)** and coronal **(F)** CBCT slices revealed the true nature of the ECR lesion and showed that the lesion was not amenable to treatment. **G,** The treatment options were discussed with the patient, and it was decided to review the tooth periodically. The 4-year radiograph shows no change in size of this asymptomatic lesion.

Set of photograph and radiographs marked A through G shows pinkish spot toward the gum line and of left incisor in the upper jaw and radiolucent pats at the root tips of the same tooth, respectively.

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FIG. 18.10 **A,** This patient's upper left central incisor had a pink spot and was cavitated. **B,** A radiograph revealed an unusual presentation of ECR; the lesion is circular and has well-defined margins. Note that the outline of the root canal is visible and intact through the radiolucent lesion. **C,** The tooth was unrestorable and was extracted; note the large amount of granulation tissue. **D,** Three-dimensional reconstruction of the extracted tooth from microtomography data revealed bonelike tissue below the overlying granulation tissue. Note the intact root canal wall *(red arrow)*. **E,** Coronal reconstruction from a microtomography scan reveals how the predentin *(red arrow)* prevented the ECR defect from invading the root canal. In addition, bonelike tissue can be seen *(yellow arrow).* Posttreatment view **(F)** and radiograph **(G)** after replacement of the upper left central incisor with an implant-retained crown.

Set of photographs and radiographs marked A through G shows the treatment of pink spot near the gum line of left incisor in the upper jaw by using implanted-retained crown.

Source: (From Patel S, Kanagasingam S, Pitt Ford T: External cervical

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resorption: a review. *J Endod* 35:616–625, 2009.)

As the process progresses, perforation of the root canal wall and bacterial contamination of the pulp may occur. The affected tooth may develop pulpitis and the associated clinical symptoms. Pulp necrosis and chronic periapical periodontitis may eventually develop. Clinical signs and symptoms may be the first indication of a problem with the affected tooth; they may include tooth discoloration, spontaneous localized pain, tenderness on mastication, tenderness to percussion, tenderness to palpation over the apical region of the tooth, a draining sinus, and/or buccal sulcus swelling.

Radiographic features

There is no usual radiographic appearance of ECR. The lesion may have well-defined or irregular margins in the cervical aspect of the tooth.

The radiographic appearance of ECR depends on the location, the extent of invasion, and the relative proportions of fibro-osseous and fibrovascular tissue occupying the resorptive cavity. All ECR defects present as a radiolucency of varying radiodensity, often in but not confined to the cervical region of the affected tooth or teeth (Figs. 18.11 and 18.12; also see Figs. 18.9 and 18.10).

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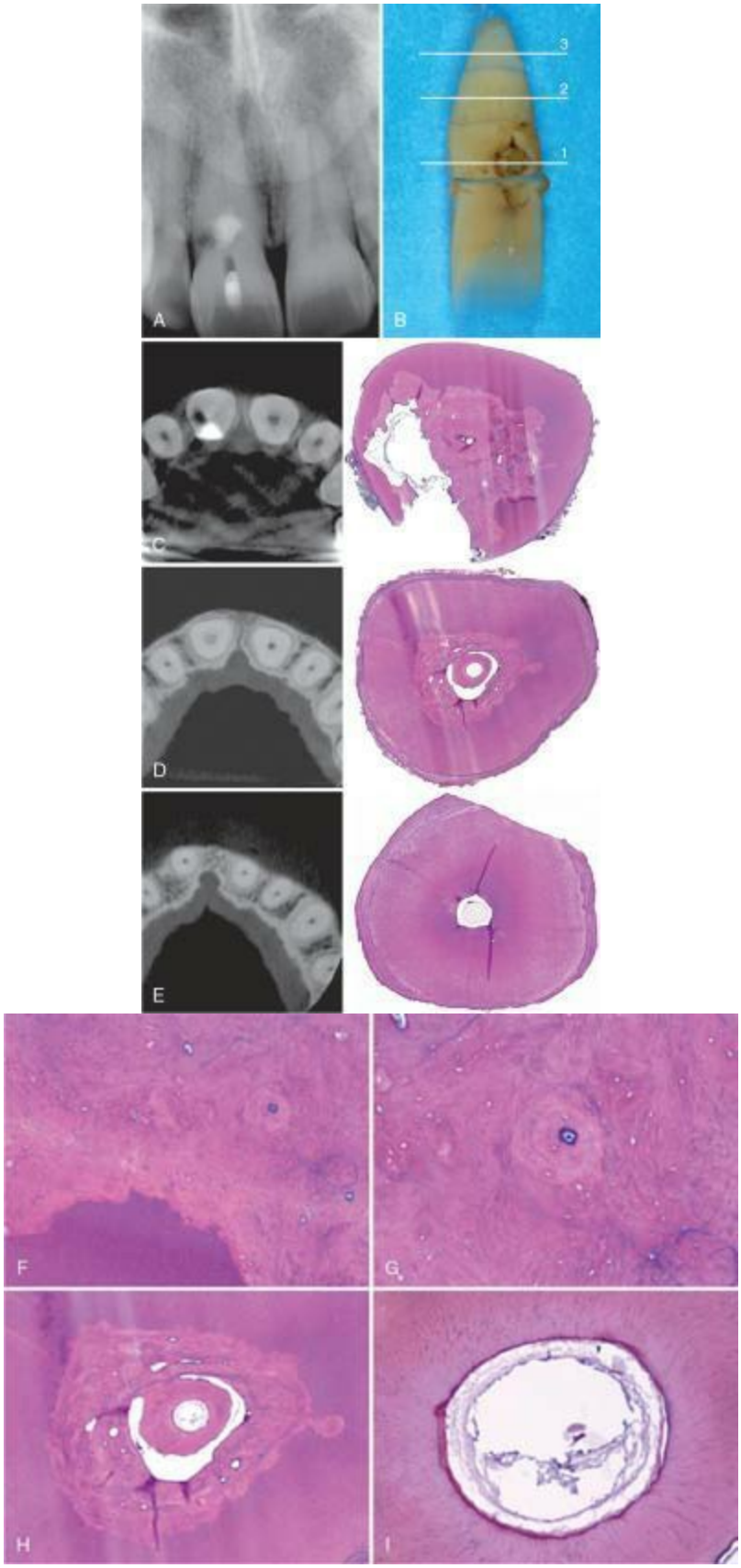

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FIG. 18.11 External cervical resorption (ECR) with replacement. **A,** Radiograph of a maxillary central incisor of a 34-year-old female patient who recalled being struck in the face with a cricket ball when she was 11 years old. The dentist mistakenly diagnosed the resorptive defect as caries and attempted to manage it accordingly. The tooth was asymptomatic; however, a 4-mm periodontal probing depth was identified on the palatal aspect of the tooth. A cone beam computed tomography (CBCT) scan confirmed an ECR defect. The tooth was deemed unrestorable and was extracted with the patient's consent. **B,** Palatal view of the tooth at the end of the demineralization process, while immersed in the clearing agent. The tooth was separated into four portions, which were embedded separately in paraffin blocks. **C,** CBCT axial section passing through the coronal third at the level of *line 1* in **B.** The corresponding histologic section shows that most of the dentin has been replaced by a bonelike tissue. The root canal is no longer present. (H&E stain; original magnification ×8.) **D,** CBCT axial section passing through the middle third at the level of *line 2* in **B.** The root canal can be appreciated at this level, although reduced in size, but it appears to be encircled by bonelike tissue (original magnification ×8). **E,** CBCT axial section taken from the apical third at the level of *line 3* in **B.** At this level, the canal is the same size of that of the contralateral tooth. The histologic section confirmed the absence of bonelike tissue (original magnification ×8). **F,** Detail from **C** showing the transition from dentin to bonelike tissue (original magnification ×100). **G,** Higher magnification of **F.** The metaplastic tissue does not show the lamellar structure typical of bone (original magnification ×400). **H,** Detail of the canal in **D.** The metaplastic bonelike tissue has concentrically replaced a consistent portion of dentin, leaving in place a reduced layer of the original dentin (original magnification ×25). **I,** High-power view confirms that the tissue surrounding the canal is dentin (original magnification ×400). *Considerations:* The resorption process, followed by replacement with a bonelike tissue, started in the cervical area and extended in an apical direction, tunneling dentin circumferentially up to the transition between the middle and apical thirds of the tooth.

Set of radiographs, specimen, and micrograph marked A through D shows the reduction of ECR from the level of line 1 to line 3 associated with the root canal of central incisor in the upper jaw.

Set of four stained micrographs marked F through I shows the conversion of bone-like tissue surrounding the root canal of incisor to dentin appeared as well-margined circular layer with tubules.

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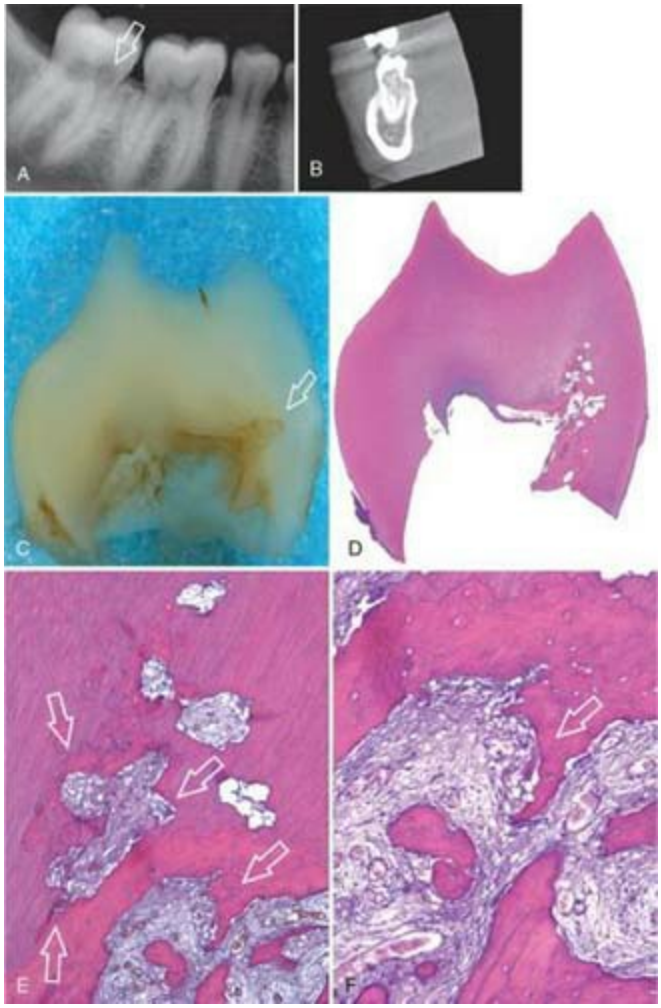

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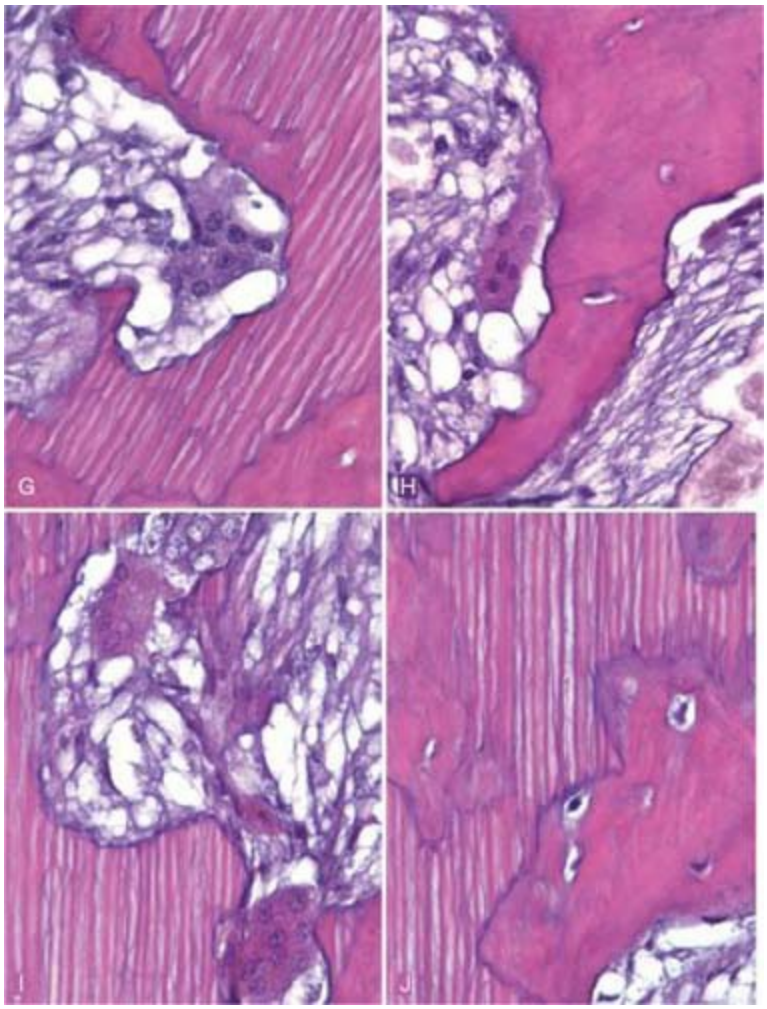

FIG. 18.12 External cervical resorption with replacement. **A,** Mandibular first and second molars in a 27-year-old female patient. The patient had symptoms of irreversible pulpitis associated with the lower right second molar; the lower right first molar was asymptomatic. **B,** CBCT scan taken through the second molar at the level of the area indicated by the *arrow* in **A.** Note massive resorption involving the crown and root. **C,** Mesial portion of the crown of the second molar after clearing. **D,** Section taken on a buccolingual plane. Overview shows resorption and replacement on the lingual side, corresponding to the area indicated by the *arrow* in **C.** (H&E stain; original magnification ×6.) **E,** Detail of the area indicated by the *arrow* in **C.** Several areas of resorption, with replacement by metaplastic tissue, can be seen (original magnification ×100). **F,** Magnification of the area indicated by the right *lower arrow* in **E.** The metaplastic tissue closely resembles bone (original magnification ×400). **G,** High-power view of the area indicated by the right *upper arrow* in **E.** Note the lacuna in dentin occupied by a multinucleated clastic cell (original magnification ×1000). **H,** High-power view of the metaplastic bone tissue in the area indicated by the *arrow* in **F.** A bone trabecula is being resorbed by a typical osteoclast (original magnification ×1000). **I,** High-power view of the

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area indicated by the left *lower arrow* in **E.** A Howship lacuna with odontoclasts can be seen (original magnification ×1000). **J,** High-power view of the area indicated by the left *upper arrow* in **E.** Note the islands of bone tissue, with typical osteocytes, surrounded by dentin (original magnification ×1000). *Considerations:* In this case the metaplastic tissue was similar to normal lamellar bone. It is interesting that the bone tissue was undergoing remodeling, as evidenced by the presence of osteoclasts. Osteoclasts and odontoclasts can be observed in the same area and show morphologic similarity.

Set of radiographs, photograph, and micrographs marked A through F shows the bonelike metaplastic tissue above the pulp chamber of first and second molars in the lower jaw.

Set of four micrographs marked G through J shows the metaplastic tissue in different areas of molar.

If the lesion is predominantly fibrovascular and granulomatous, the lesion tends to be radiolucent. However, in long standing lesion, the radiolucency may appear with a cloudy appearance. In advanced cases with extensive repair of the tissue destruction, significant deposition of fibro-osseous tissue gives the defect a mottled radiographic appearance (Fig. 18.13).

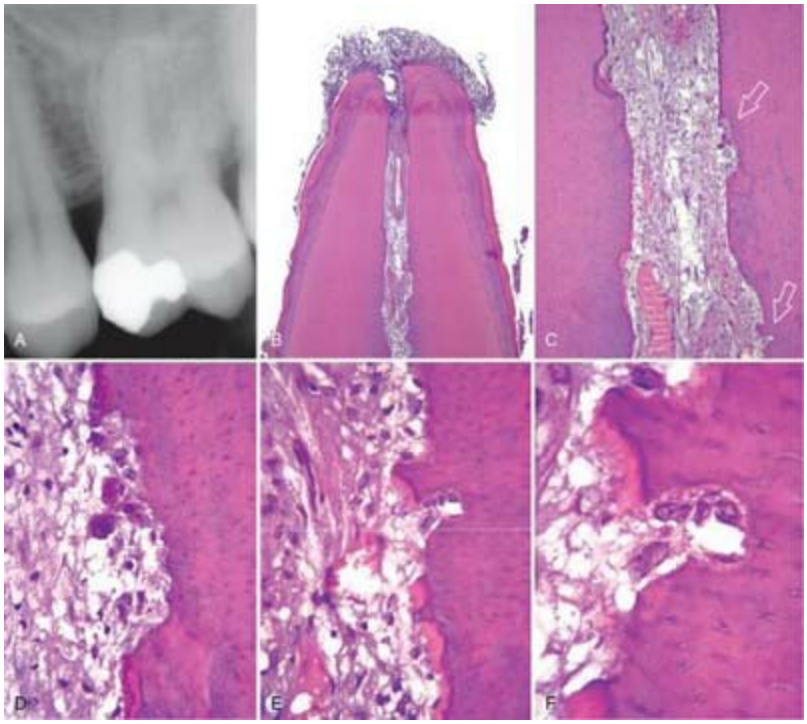

FIG. 18.13 Early internal root resorption. **A,** A 54-year-old male patient

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presented with a long story of pain on chewing and with cold stimuli. Recently the pain had become continuous and severe. The maxillary first molar, which had a mesial amalgam restoration, did not respond to sensitivity tests. A radiograph showed a periapical radiolucency on the palatal root. After removal of the restorative materials, a crack line involving the pulp chamber floor was diagnosed, and the tooth was extracted. **B,** Some pathologic tissue remained attached to the palatal root apex at extraction. A longitudinal section passing at the center of the canal demonstrated vital connective tissue in the apical third. (H&E stain; original magnification ×16.) **C,** Detail of the canal in **B.** Note the connective tissue with vessels and relatively few inflammatory cells, in addition to the resorption lacunae on the root canal walls (original magnification ×1000). **D,** High-power view of the area of the canal wall indicated by the upper *arrow* in **C.** Note the resorption lacuna with odontoclasts (original magnification ×1000). **E** and **F,** Progressive magnification of the area of the canal wall indicated by the lower *arrow* in **C.** Note the Howship lacuna housing clastic cells. Predentin is present in some areas of the wall, but it is absent in the areas with active resorption (original magnification ×400 and ×1000).

Set of radiograph and stained micrographs marked A through F shows the vesicles and inflammatory cells along with predentin on the root canal.

The margins of the lesion may vary from poorly to well defined, depending on the depth of the defect and the proportion and distribution of osseous inclusions in the lesion. Although lesions with irregular margins are more common, some ECR defects may have smooth and/or well-defined margins.

Despite its often cervical location, ECR may commence apical to this region, reflecting the position of the epithelial attachment of the affected tooth. In teeth with a normal periodontal attachment, the invasive nature of the process may result in the lesion extending some distance apical and/or coronal to the cervical location where it started. Furthermore, the tissue destruction at the site of onset may sometimes be minimal and/or not evident on conventional radiographs due to its location on the root surface. In these instances, the lesion may appear to have originated at a location where significant tissue destruction, evident radiographically, has occurred. This may be some distance from the actual point of origin. This feature has come to light only since the advent of assessment of ECR with CBCT.

The radiographic features of ECR are very similar to those of IRR (discussed later), and differentiating between them, especially in the absence of clinical signs, may be challenging. It is useful to trace the outline of the

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root canal walls as they approach and pass through the resorption defect on the radiograph. In cases of ECR, the outline of the canal wall should be visible and intact and should maintain its course as it passes through the defect. This is due to the fact that the resorptive lesion lies on the external surface of the root and is not in communication with the root canal; it is merely superimposed on the defect radiographically. In cases of IRR, it should be possible to trace the outline of the root canal through the resorptive defect because the defect is an extension of the root canal wall and is continuous with it.[46] Although this is a useful diagnostic feature, it does have some shortcomings. First, the outline of the root canal wall may be obscured by calcified tissue in the resorptive defect (ECR or IRR). Second, when ECR has resulted in extensive tissue destruction, perforation of the root canal wall may have allowed communication between the canal wall and the external defect.

Parallax radiographs should always be used to obtain further information about the nature of the resorptive process. In addition to a paralleled periapical radiograph, another radiograph should be taken with a shift (parallax) in the horizontal angulation of the x-ray tube in relation to the image receptor. In cases of ECR, the position of the resorptive defect moves relative to the root canal. If the lesion is located palatally/lingually, the defect moves in the same direction as the x-ray tube shift. If the lesion is located buccally, it moves in the opposite direction. This is sometimes referred to as the “same lingual, opposite buccal” (SLOB) rule. In contrast, internal resorptive defects maintain their position relative to the root canal because the defects are an extension of the root canal system.

CBCT has allowed 3D assessment of the nature, position, and extent of the resorptive defect, eliminating diagnostic confusion and providing essential information about the restorability and subsequent management of the tooth. CBCT is particularly useful if the clinician is not sure whether the ECR cavity has perforated the root canal wall (and thus for determining the need for root canal treatment). A CBCT scan eliminates the need for exploratory treatment. In an ex vivo study, Vaz de Souza et al.[113] compared the diagnostic efficacy of two CBCT scanners with periapical radiograph for the detection and classification of simulated ECR lesions. Examiners were asked to classify and identify the root surface(s) that ECR extended to with

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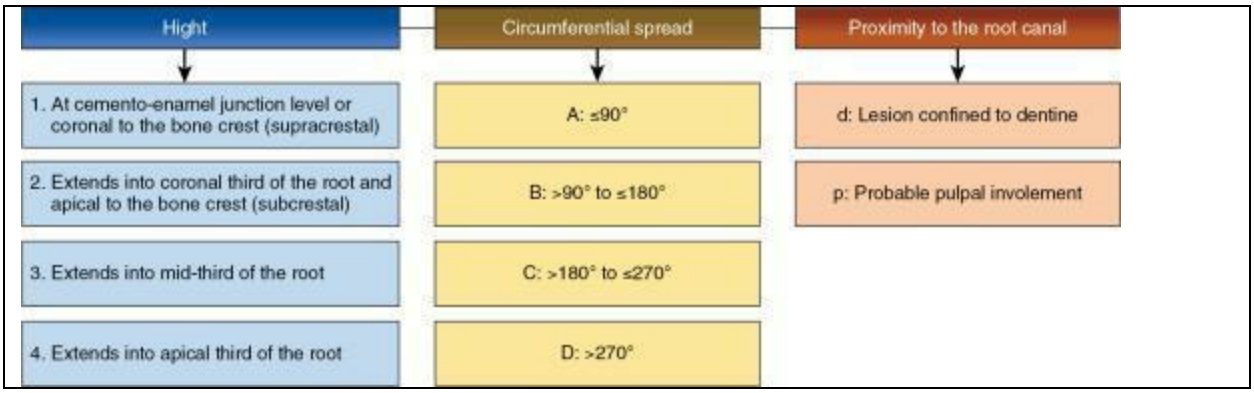
periapical radiograph and CBCT. Identification of the lesion according to the tooth surface was successful in 89.1% Morita CBCT, 87.8% Kodak CBCT, and 49.4% periapical radiograph cases. According to Heithersay classification, ECR was classified correctly in 71.4% Kodak CBCT, 70% Morita CBCT, and 32% periapical radiograph.

A clinical study by Patel et al.[83] comparing the ability of conventional radiographs and CBCT to diagnose and to differentiate accurately between IRR and ECR showed that CBCT was significantly more accurate (100%) than periapical radiographs at diagnosing the presence and nature of the root resorption. These researchers also concluded that the correct treatment plan should incorporate the additional information provided by CBCT.[83]

Recently, Patel et al.[86] suggested a 3D classification that accounts for the lesion height, circumferential spread, and proximity to the root canal. The aim of the classification is to ensure accurate diagnosis and communication of ECR between clinicians (Table 18.1).

Table 18.1

Three-Dimensional Classification of External Cervical Resorption



Management

The fundamental treatment objectives in ECR are to excavate the resorptive defect, halt the resorptive process, restore the hard tissue defect with an esthetic filling material, and prevent and monitor the tooth for recurrence. Effective management of ECR depends on accurate assessment of the true nature and accessibility of the lesion.

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Preoperative CBCT scans can help in assessing the extent of the defect and aid in good treatment planning. Also, treatment options could be confidently discussed with the patient and "exploratory" treatment may be avoided.[97,98]

Small lesions have the most favorable outcome where typically the pulp is not involved. These lesions are considered as Heithersay class 1 and class 2.[55] Surgical access to the site of resorption is gained by raising a mucoperiosteal flap, the dimensions of which should allow visualization of the full extent of the defect. Once access has been achieved, the resorptive cavity is excavated. Fibrovascular granulomatous tissue is readily removed with a hand excavator. However, defects containing significant amounts of fibro-osseous tissue (especially when the latter is contiguous with the adjacent dentin) require discriminate removal of the tissue with ultrasonic instruments. It may be extremely difficult to differentiate between sound dentin and fibro-osseous deposits; therefore, use of the surgical operating microscope is essential. Frequent intraoperative radiographs may be necessary to ensure accurate removal of unwanted, hard resorptive tissue and to prevent unnecessary removal of sound dental tissue.

Once the resorptive tissue has been removed, the cavity may be treated with a 90% aqueous solution of trichloroacetic acid; this causes coagulation necrosis of the resorptive tissue without damaging the periodontal tissue.[55] The acid also penetrates and treats small channels of resorption that are not accessible to mechanical instrumentation.[87] Once the defect has been excavated and treated with trichloroacetic acid, any undermined dentin or enamel at the peripheries of the cavity is removed with a bur in a high-speed turbine, and the cavity is restored with an esthetically acceptable restorative material, such as composite resin or glass ionomer cement. Biodentine may prove to be a particularly suitable material for restoring these defects[28,80] because it may combine acceptable esthetics with the ability to support PDL attachment.

Once the cavity has been restored, the mucoperiosteal flap is replaced and secured in position. If perforation of the root canal wall has occurred, root canal treatment should be carried out. Access to the root canal system should be gained under a rubber dam before the resorptive defect is assessed. The root canal should be prepared in the area of the defect as normal, using saline as an irrigant. A tapered gutta-percha (GP) point then should be placed in the

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canal to maintain its patency during the excavation and restoration of the resorptive defect and to provide a barrier against which the final restoration can be condensed. The rubber dam is removed, and surgical treatment of the resorptive defect can be carried out as described, without any risk of the resorptive debris entering the root canal system. After repositioning of the mucoperiosteal flap, root canal treatment can be completed in the normal manner, without fear of extrusion of infected tissue, irrigants, or medicaments into the periodontal tissues.

One of the treatment modalities of ECR is internal repair of the lesion, which does not require any surgical procedure.[85] This treatment option is indicated when the resorptive defect is close to or has just perforated the pulp chamber, and external surgical flap is not accessible and/or result in excessive removal of sound tooth structure. The internal repair is preferred in cases where the entry point is small, intraosseous, and apical to the epithelial junction.

Root canal treatment in such cases might be indicated if the defect was close to the pulp. Endodontic treatment could be done in one visit. After completion of the endodontic treatment, the access cavity should be extended to encompass the ECR lesion. The ECR and endodontic access cavity may be restored with a direct plastic restoration such as composite resin or glass-ionomer cement. Alternatively, Biodentine could be used as a dentine substitute, over which a thin layer of glass ionomer cement and/or composite resin may be placed.[90,99]

The treatment of ECR depends on the severity, extent, and location of the resorptive defect and the restorability of the tooth. Heithersay[55] developed a four-stage classification system for ECR based on the depth of penetration of the resorption in a buccolingual and apicocoronal direction. He examined the prognosis of treatment in 101 cases of ECR in 94 patients using the nonsurgical protocol referred to previously and related the success rates for treatment to the classification of the lesion.[55] He reported a 100% success rate for class I and class II lesions, a 77.8% success rate for class III lesions, and a 12.5% success rate for class IV lesions. This emphasizes the poorer outcome that can be expected for more advanced cases. A major limitation of the Heithersay classification is that it is valid only if the ECR lesion is confined to the proximal aspect of the tooth, because lesions are assessed on

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2D radiographs. If the ECR lesion is located on and/or extends to the labial and/or buccal (proximal) aspects of the tooth, the true nature of lesion cannot be accurately assessed with radiographs.[81,87]

Internal root resorption

Introduction

IRR is a form of root resorption that originates in and affects the root canal wall.[6] It is further classified as either inflammatory or replacement. The replacement type is associated with the deposition of mineralized tissue in the root canal space after the initial loss of dentin (initial dentin loss is a feature of both types).[6] Because their characteristics are largely similar, the two types of IRR are discussed together.

Etiology and pathogenesis of internal root resorption

Damage to the outermost protective odontoblast layer and the predentin of the canal wall results in exposure of the underlying mineralized dentin to odontoclasts, which subsequently leads to IRR (Fig. 18.14).[108,118]

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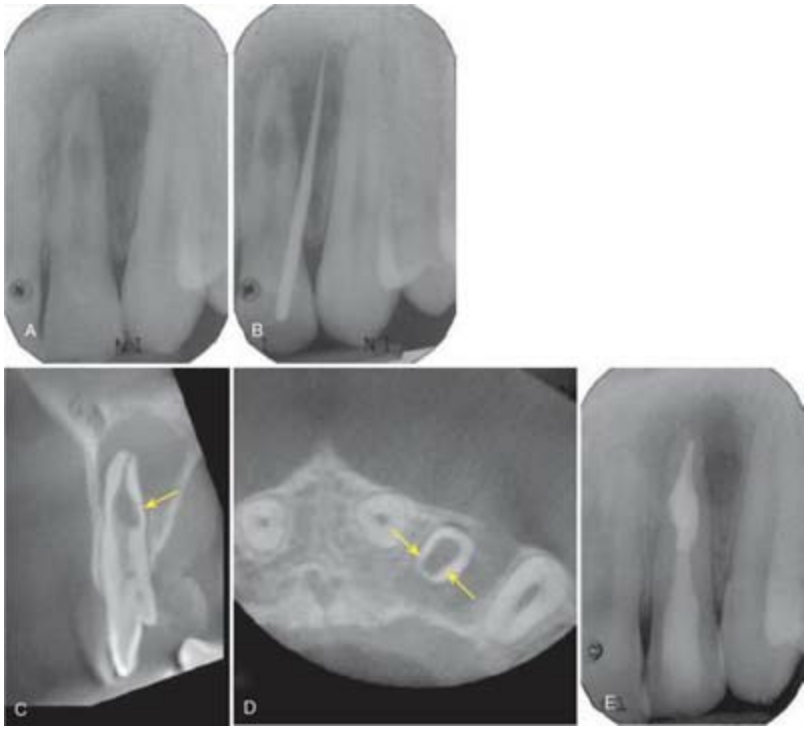

FIG. 18.14 **A** and **B,** Parallax views of the maxillary left lateral incisor showing internal root resorption with necrosis. A gutta-percha point has been used to track the sinus. The reconstructed sagittal **(C)** and axial **(D)** slices from a CBCT scan reveal that the lesion has resorbed the palatal aspect of the root *(arrows)* and has nearly perforated the root wall. **E,** The tooth has been obturated with gutta-percha using a thermoplasticized technique.

Set of five radiographs marked A through E shows the healing of radiolucent root canal and patch near the apex using gutta-percha.

Source: (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review. *J Endod* 36:1107–1121, 2010.)

The exact cause or type of injury that causes such damage is not completely understood. Several etiologic factors have been expressed for the loss of predentin, including trauma, caries and periodontal infections, excessive heat generated during restorative procedures on vital teeth, calcium hydroxide procedures, vital root resections, anachoresis, orthodontic treatment, cracked teeth, or simply idiopathic dystrophic changes in normal pulps.[7,17,26,92,115] In a study of 25 teeth with internal resorption, trauma was found to be the most common predisposing factor, responsible for 45% of the cases examined.[27] The suggested etiologies in the other cases were inflammation as a result of carious lesions (25%) and carious/periodontal

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lesions (14%). The cause of the internal resorption in the remaining teeth was unknown. Other reports in the literature support the view that trauma[7,119,121] and pulpal inflammation/infection[52,121] are the major contributory factors in the initiation of internal resorption.

As previously mentioned, damage to the odontoblast layer and predentin of the canal wall is a prerequisite for initiation of IRR.[119] However, progression of IRR depends on bacterial stimulation of the clastic cells involved in hard tissue resorption. Without this stimulation, the resorption is self-limiting.[119]

For IRR to continue, the pulp tissue apical to the resorptive lesion must have a viable blood supply; this provides clastic cells and their nutrients, and the infected necrotic coronal pulp tissue provides stimulation for those clastic cells (see Fig. 18.14).[107] Bacteria may enter the pulp canal through dentinal tubules, carious cavities, cracks, fractures, and lateral canals. In the absence of a bacterial stimulus, the resorption is transient and may not advance to the stage that can be diagnosed clinically and radiographically. Therefore, the pulp apical to the site of resorption must be vital for the resorptive lesion to progress (see Fig. 18.14). If left untreated, internal resorption may continue until the inflamed connective tissue filling the resorptive defect degenerates, advancing the lesion in an apical direction. Ultimately, if left untreated, the pulp tissue apical to the resorptive lesion undergoes necrosis and the bacteria infect the entire root canal system, resulting in apical periodontitis.[95]

Histologic appearance

Wedenberg and Zetterqvist[121] reported on the histologic nature of IRR. The authors specifically examined the histologic, enzyme histochemical, and scanning electron micrographic (SEM) features of the resorptive process in a small sample of primary and secondary teeth extracted due to progressive IRR. The histologic and enzyme histochemical profiles were identical for the two groups, but the resorptive process appeared to occur more rapidly in the primary teeth. The pulpal tissue was populated to varying degrees in all teeth with an inflammatory infiltrate composed predominantly of lymphocytes and macrophages, with some neutrophils. The connective tissue in the pulp spaces was less vascular than healthy pulpal tissue and resembled periodontal membrane connective tissue with relatively more cells and fibers. The odontoblast layer and predentin were absent from the affected dentinal walls,

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which were populated by large, multinucleated odontoclasts occupying resorption lacunae. The odontoclasts showed evidence of active resorption (see Fig. 18.14). They were accompanied by mononuclear cells, believed to be odontoclast precursors, which populated the connective tissue adjacent to the site of resorption. Both types of cell displayed tartrate-resistant acid phosphatase (TRAP) activity.

Interestingly, the root canal wall was incompletely lined with a mineralized tissue resembling bone or cementum in all of the examined teeth.[121] Furthermore, islands of calcified tissue of a similar nature occupied the pulpal space in three of the cases. Islands of mineralized tissue occupying the root canal space are the defining feature of internal replacement resorption.[6] The authors suggested that deposition of this mineralized tissue is likely to be part of a coupling process, at the end of a period of resorption, in which osteoblasts are attracted to the affected site and participate in bone formation.[121]

Clinical features

The clinical features of IRR largely depend on the histologic status of the affected pulp, the extent of the hard tissue destruction caused by the resorptive process, and the position of the resorptive cavity in the root canal space. In the active stages of resorption, bacterial contamination of vital pulpal tissue may cause an acute inflammatory response, leading to clinical symptoms of pulpitis. With the onset of pulpal necrosis and an established bacterial colonization of the root canal space, clinical signs and symptoms associated with acute or chronic apical periodontitis may develop. Sinus tract(s) may occur and may be associated with suppuration in the periapical tissues or possibly at the site of a perforation of the root canal wall caused by the hard tissue destruction. Extensive resorption of the coronal pulp may result in a pink or red discoloration visible through the crown of the affected tooth; this is caused by granulomatous tissue extending into and occupying the resorptive defect.[67] Although often reported as a common clinical indicator of the process, these pink spots actually are rare in cases of IRR. They may occur relatively more frequently in cases of ECR, but they are not very common with that resorption type, either. Often the affected tooth is asymptomatic, and clinical signs are absent.

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Radiographic features and diagnosis

The diagnosis of any type of root resorption depends on radiographic demonstration of its presence. Two-dimensional radiographs make the detection and differentiation of the various types of resorption challenging. Both IRR and ECR could have similar features which makes differentiation and diagnosis from a two-dimensional shadowgraph difficult.[51,84,88,107] Gartner et al.[46] reported that lesions of IRR present radiographically as radiolucencies of uniform density that have a smooth outline and are symmetrically distributed over the root of the affected tooth (Fig. 18.15). The authors further reported that the outline of the root canal wall should not be traceable through the resorption defect because the root canal wall balloons out. Other authors have described IRR lesions as oval, circumscribed radiolucencies in continuity with the root canal wall.[77] Although certain cases of IRR may have some or all of these radiographic features, many do not; each case should be assessed individually before a diagnosis is made.

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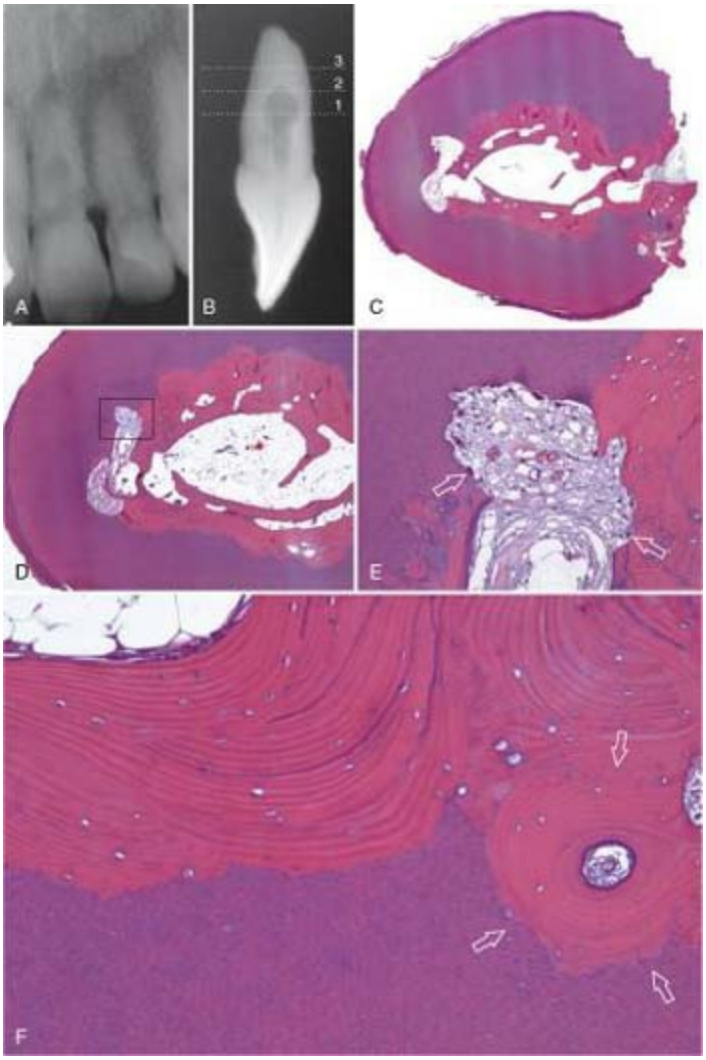

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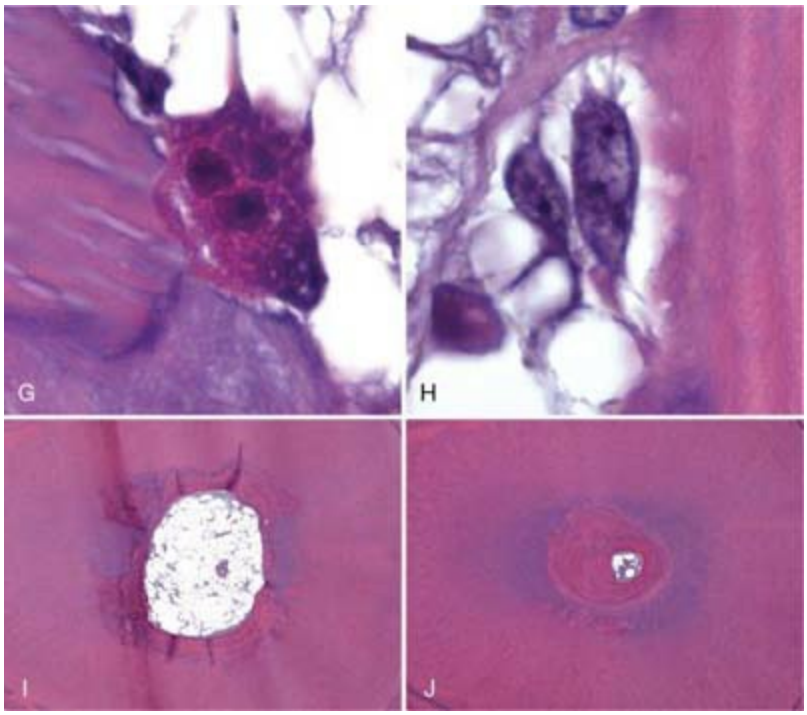

FIG. 18.15 Light microscopy images of a tooth with internal (root canal) replacement resorption. The tooth belonged to a 44-year-old male patient who was referred to the first author for management of a perforated root. The tooth was asymptomatic on examination, but there was a history of previous trauma. **A,** Radiograph of a maxillary central incisor with a radiolucent lesion in the middle third of the root canal. The radiolucent lesion appears to be mottled, which suggests internal root resorption with metaplasia. **B,** Clinical radiograph of the tooth after extraction, taken at a 90-degree angle, showing the continuity of the resorptive lesion with the canal space. **C,** Cross section taken approximately at the level of *line 1* in *B.* The low-magnification overview shows that the dentin around the root canal had been replaced by an ingrowth of bone tissue, and the root appears to have been perforated on the distopalatal aspect. (H&E stain; ×8.) **D,** Higher magnification of *C.* (H&E stain; ×16.) **E,** High magnification of the area demarcated by the rectangle in *D.* The intraradicular dentin has been resorbed. (H&E stain; ×100.) **F,** High-magnification view taken from the right part of *C,* showing that the resorbed dentin has been replaced by lamellar bone. Osteocytes are present in lacunae between the lamellae. A characteristic cross section of an osteon can be seen on the right *(open arrows),* with concentric lamellae surrounding a vascular structure. (H&E stain; ×100.) **G,** High-magnification view of the area indicated by the left open *arrow* in *E.* A multinucleated resorbing cell (odontoclast) can be seen in a dentinal lacuna, indicating active resorption of the dentinal wall. (H&E stain; ×1000.) **H,** High-magnification view of the bone surface indicated by the right *arrow* in *E.* The large cells are osteoblast-like cells. Once they produced mineralized tissue, they were embedded in the bone lacunae, assuming the characteristics of

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osteocytes. (H&E stain; ×1000.) **I,** Cross section taken approximately at the level of *line 2* in *B.* The root canal is still large at this level and is surrounded by a relatively thin layer of newly formed bone. (H&E stain; ×16.) **J,** Cross section taken approximately at the level of *line 3* in *B.* At this level the root canal appears consistently narrowed by a dense layer of newly formed bone. (H&E stain; ×16.)

Set of radiograph and micrographs marked A through F shows the development of concentric circles around the root canal.

Set of four micrographs marked G through J shows the deposition of multinucleated cells resulting in the reduction of root canal.

Source: (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review. *J Endod* 36:1107–1121, 2010.)

IRR can occur at any location in the root canal system and may manifest radiographically as a radiolucency with variable shape, radiodensity, outline, and symmetry in relation to the root canal. Internal inflammatory root resorption lesions are more likely be uniformly radiolucent, whereas in internal replacement (metaplastic) root resorption, the defect has a somewhat mottled or clouded appearance as a result of the radiopaque nature of the calcified material occupying the lesion (Fig. 18.16).[88] ECR lesions may contain predominantly granulomatous tissue, predominantly calcified tissue, or a mixture of the two; therefore, they may have a radiodensity similar to either type of internal resorption, which complicates the clinical differentiation of the disease process. As noted by Gartner et al.,[46] the best practice is to trace the outline of the root canal wall as it approaches and leaves the resorption defect. An IRR cavity is continuous with the normal root canal walls because it is essentially an extension of them. As such, in teeth with single canals affected by IRR, the canal walls should not be traceable through the defect. This is in contrast to ECR, in which the lesion lies buccal or palatal/lingual to the defect and is consequently superimposed on the canal system when viewed on conventional radiographs. In this situation the canal walls should maintain their normal course as they pass through the resorption defect, allowing them to be traced through it. However, it should be noted that in teeth with multiple canals, a canal that has been unaffected by IRR may be superimposed onto the IRR resorption defect on conventional radiographs. When the guidelines outlined by Gartner

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et al.[46] are used, this may lead to misdiagnosis

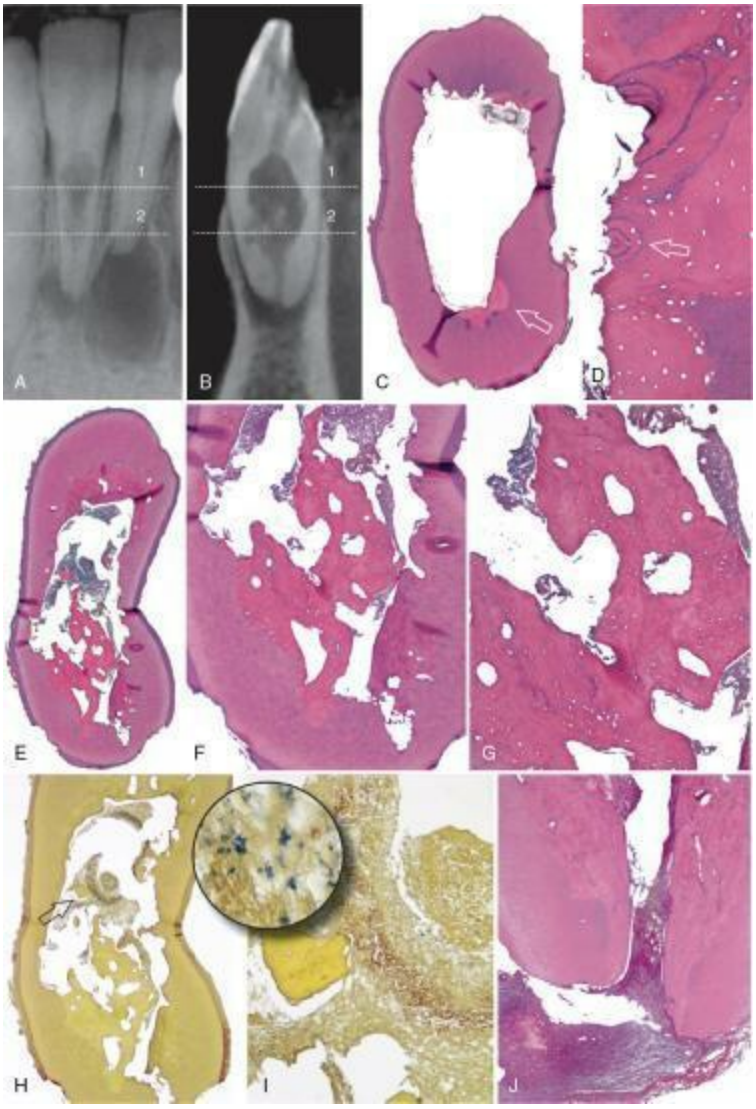

FIG. 18.16 Light microscopy images of a variant of internal (root canal) replacement resorption with tunneling resorption. The lower right lateral incisor belonged to a 39-year-old former boxer who had suffered a jaw fracture in a boxing match in his early twenties and was placed in intermaxillary fixation. The patient developed symptoms 20 years later and complained of pain associated with his lower incisors. **A,** Radiograph of the mandibular right incisors. The lower right central incisor had asymptomatic apical periodontitis associated with a necrotic and infected pulp. The lower right lateral incisor showed a large area of internal root resorption. The tooth did not respond to sensitivity tests. **B,** Sagittal CBCT slice shows some calcified tissue in the resorptive defect. **C,** Cross section taken at the level of *line 1* in *A* and *B.* The overview shows that the canal was apparently empty at this level. (H&E

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stain; ×6.) **D,** High magnification of the area indicated by the *arrow* in *C.* Lamellar bone filling an area of previous resorption. Note the osteon structure *(arrow).* (H&E stain; ×100.) **E,** Cross section taken at the level of line 2 in *A* and *B.* Overview shows that the canal lumen was partly occupied by necrotic remnants, partly by bonelike tissue. (H&E stain; ×8.) **F,** High magnification of the lower part in *E.* (H&E stain; ×50.) **G,** Higher magnification of *F.* Bone trabeculae are surrounded by necrotic debris. (H&E stain; ×100.) **H,** Cross section taken from the same area as that in *E*. (Taylor's modified Brown & Brenn [TBB] stain; ×16.) **I,** High magnification of the area indicated by the *arrow* in *H.* A fragment of bonelike tissue can be seen surrounded by bacteria-colonized necrotic tissues. (TBB stain; ×100; inset ×1000.) **J,** Longitudinal section passing approximately through the center of the root apex. Dentin walls have been resorbed and replaced by a bonelike tissue. (H&E stain; ×16.).

Set of radiographs and stained micrographs marked A through J shows the deposition of bonelike tissue around the root canal at the center of right incisor.

Source: (From Patel S, Ricucci D, Durack C, Tay F: Internal root resorption: a review. *J Endod* 36:1107–1121, 2010.)

Parallax radiographs must always be used to obtain further information about the resorptive process. In addition to a paralleled periapical radiograph, a radiograph should be taken with a shift in the horizontal angulation of the x-ray tube in relation to the image receptor. IRR lesions maintain their position relative to the root canal system on the angled view. ECR lesions move in the same direction as the x-ray tube shift if they are lingually/palatally positioned and move in the opposite direction if they are buccally located.[46] This diagnostic technique, coupled with tracing of the root canal/pulp chamber outline through the lesion, have been the most reliable aids in the differential diagnosis of IRR when conventional radiography is used. However, as discussed previously, the amount of information available from conventional radiographic imaging is limited. This can lead to misdiagnoses and incorrect treatment in the management of IRR and invasive cervical resorption.

The use of CBCT as a diagnostic and treatment planning tool in the management of IRR has been reported in the literature.[20] Information such as the position, extent, and dimensions of an IRR lesion, in addition to the presence of any associated perforation, can be obtained from a CBCT scan.

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The same scan can differentiate between ECR and IRR, removing any doubt about the diagnosis that may have arisen with the conventional radiographic examination.

Management

Once a diagnosis of IRR has been made, the extent of the hard tissue destruction must be assessed, and a clinical decision must be made about the prognosis of the affected tooth. If the affected tooth is salvageable and has a reasonable prognosis, root canal treatment is necessary. As with any infected tooth, the main purpose of the root canal treatment is to remove the intraradicular bacteria and disinfect the root canal space. If the resorptive process is still active, the treatment serves an adjunctive purpose, which is to eliminate the vital apical tissue that is sustaining and stimulating the resorbing cells.

The nature of the resorptive process in cases of IRR presents the endodontist with unique operative challenges. In teeth with active resorption, profuse bleeding from the granulomatous and inflamed pulpal tissues may impair visibility in the initial stages of treatment and may provide a stubborn source of mild hemorrhage when attempts are made to dry the canal after chemomechanical preparation. Furthermore, the irregularly concave nature of resorption defects makes them inaccessible to direct mechanical debridement.

Chemomechanical debridement of the root canal

Teeth usually have unique morphologic features of root canal that harbor microorganisms in infected teeth. Endodontic instruments and passively delivered irrigants fail to penetrate into these secluded spaces and niches.[75,96,101] The use of ultrasonic instruments to aid the penetration of endodontic irrigants has been shown to improve the removal of organic debris and biofilms from the root canal space.[24] Given the inaccessibility of IRR defects to normal instrumentation and passive irrigation, ultrasonic activation of irrigants should be considered an essential step in the treatment of these cases (see Fig. 18.14). However, even when this adjunctive measure is used, microbes may persist in confined areas after chemomechanical debridement.[24] As such, an intracanal antibacterial medicament should be used to further reduce the microbial load and improve the disinfection of the

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root canal space.[101] Calcium hydroxide is an antibacterial, interappointment, endodontic medicament that has been shown to eradicate bacteria persisting in the root canal space after root canal treatment.[25,102] Also, when used in conjunction with sodium hypochlorite, it potentiates the effect of that irrigant in the removal of organic debris from the root canal system.[1,110] Based on this evidence the authors advocate the use of calcium hydroxide as an intracanal, antibacterial medicament to supplement the conventional chemomechanical debridement of the root canal system.[88]

Obturation

One of the fundamental objectives of endodontic treatment is to fill the disinfected root canal space completely with an appropriate material. In IRR, the hard tissue defects caused by the resorptive process are challenging to fill adequately. To obturate the resorptive defect, the root-filling material must be able to flow. GP is widely regarded as the gold standard filling material in endodontics. It can plasticize when pressure is applied, and it becomes flowable with the application of heat. Gencoglu et al.[47] examined the ability of different obturation systems and techniques to fill the defects in artificially created internal resorptive cavities ex vivo. They found that Obtura II (ObII) and Microseal (MS) thermoplastic GP systems produced significantly better fills in simulated resorptive cavities than did Thermafill, soft core systems (SCS), and cold lateral condensation (CLC). In a similar study Goldberg et al.[49] demonstrated that the Obtura II system filled simulated resorptive defects statistically better than CLC, Thermafill, and a hybrid technique. Gencoglu et al.[47] reported that the Obtura II and MS systems produced fills with greater GP–to-sealer (GP/sealer) ratios than did the other techniques examined. These findings were corroborated in the study by Goldberg et al.[49]

Because root canal sealers shrink on setting[122] and dissolve and degrade to varying degrees in the presence of moisture,[73] fillings with higher GP/sealer ratios reduce the risk of void formation and leakage of contaminants into the root canal system, with potentially positive benefits for the treatment outcome.

Stamos and Stamos[105] and Wilson and Barnes[123] reported on cases of internal resorption in which the Obtura system was successfully used to

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obturate the canal. In light of the evidence presented, the Obtura and MS systems apparently can be expected to produce the best technical results for obturating canals with IRR.

When choosing the appropriate materials and methods for filling resorptive defects of IRR, the clinician first must establish the presence and extent of any perforations in the wall of the affected root. This information can be readily obtained from an appropriate CBCT scan. If perforation has occurred, mineral trioxide aggregate (MTA) should be considered the material of choice to repair the root wall. MTA is biocompatible,[74] has superior sealing properties when used as a retrograde filling material,[16] and has proved effective in the repair of lateral and furcal root perforations in animal studies.[68] Furthermore, the material is well tolerated in the periapical tissues, and when used as a root-end filling material in the absence of infection, it supports almost complete regeneration of the adjacent periodontium.[93] These are desirable properties in the context of perforation repair because of the very real possibility of unintentional extrusion of the material when a perforating internal resorptive defect is repaired in an orthograde manner. However, the flow properties of MTA are significantly poorer than those of heated GP. Its use as an effective filling material in IRR depends on adequate ultrasonic activation of the material to disperse it into the recesses of the defect.[20] Use of a dental operating microscope and the correct equipment to deliver the material are essential.

A hybrid technique to obturate canals affected by perforating internal resorption also may be used. In these cases, the canal apical to the resorption defect is filled with GP. The GP can then be used as a barrier against which the MTA can be packed. Hsiang-Chi Hsien et al.[57] successfully used this technique to treat a perforating IRR defect in a maxillary incisor. Jacobovitz and De Lima[59] described a case in which a maxillary central incisor with a large, perforating internal resorption defect had a poor prognosis and initially was designated for extraction. The tooth subsequently was successfully treated in an orthograde manner with white MTA and GP.[59]

Clinical situations arise in which a perforating resorptive defect causes extensive dental hard tissue destruction that fails to respond to or is not amenable to repair with an orthograde approach. Surgical treatment may be needed in these cases. For the reasons discussed already, MTA would be the

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material of choice to repair these perforations. In cases that have not already been treated in a nonsurgical manner, the operator first must access the root canal as for orthograde treatment. A well-fitting, tapered GP point or an appropriately sized finger spreader is then positioned in the canal to occlude it and to provide a barrier against which the MTA can be packed once surgical access to the defect has been gained. The barrier also prevents inadvertent deposition of the MTA into the apical third of the canal. The perforation is then exposed surgically and repaired with the MTA. The canal can be shaped, disinfected, and obturated with thermoplasticized GP once the MTA has set.

If the resorptive process has caused sufficient tissue destruction to render the tooth unrestorable, extraction is the most appropriate treatment option. If the tooth has been weakened by the disease process to the extent that fracture of the tooth root is likely, the patient may elect to have the tooth extracted. The presence of a perforating resorptive defect is certainly not a contraindication to treatment, but a perforation of significant size will have a bearing on the decision to surgically treat or extract the tooth. CBCT is an invaluable component of the clinician's armamentarium in the treatment of IRR.[38] A CBCT scan gives the clinician a 3D view of the tooth, the resorptive defect, and the adjacent anatomy. The clinician thus has the information necessary to determine a prognosis for the tooth and/or its amenability to surgical repair. If extraction of the affected tooth is indicated, the scan may be used as a diagnostic and treatment planning tool for provision of a dental implant–retained prosthesis. Bhuva et al.[20] described a case in which CBCT was used as a treatment planning tool in the successful treatment of a case of IRR. As mentioned previously, CBCT has been found to be highly accurate in revealing the nature of the resorptive lesion, and this leads to the selection of the most suitable treatment plan.

Summary

- More research is needed for understanding of the etiology and pathogenesis of different types of root resorption (Table 18.2).
- New ECR classification provides a better description of the lesion in a three-dimensional manner.

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- The earlier the detection of root resorption, the better the prognosis and the outcome.
- CBCT is an excellent diagnostic tool for the confirmation of the presence of ECR and IRR and also in appreciation of the true nature of these conditions.

Table 18.2

Characteristic Key Features of Root Resorption

	INTERNAL		EXTERNAL			
Resorption Type	**Inflammatory**	**Replacement**	**Inflammatory**	**Replacement**	**Cervical**	**Surface**
Clinical finding	Discoloration, symptoms of irreversible pulpitis and/or apical periodontitis	Discoloration, Symptoms of irreversible pulpitis and/or apical periodontitis	Discoloration, symptoms of apical periodontitis	May exhibit a metallic percussion sound. Mobility not expected	Probable periodontal defect, symptoms of (ir)reversible pulpitis	None
Clinical appearance	Pink spot (rare), or sign of pulpitis/apical periodontitis.	Pink spot (rare), or sign of pulpitis/apical periodontitis.	Healthy, or sign of pulpitis/apical periodontitis.	Healthy	±pink spot, or sign of pulpitis/apical periodontitis.	Healthy
Location on root	Anywhere	Anywhere	Anywhere	Anywhere	Cervical third initially but can be seen in mid and apical third in advanced cases	Adjacent to impacted tooth, cyst or tumour. Apical in orthodontic treated teeth
Pulp sensitivity	Maybe +ve in partially vital cases, or −ve in advance necrotic cases	Maybe +ve in partially vital cases, or −ve in advance necrotic cases	−ve	+ve	Usually +ve, −ve in advanced necrotic cases	+ve
Radiological appearance	Symmetrical oval shaped (ballooning) enlargement of root canal	Oval shaped enlargement of root canal with cloudy and/or mottled canal wall	Asymmetrical bowl shaped periradicular or periapical radiolucencies	Asymmetrical bony replacement of root surface and loss of PDL space	(A) symmetrical radiolucent (early), or radiopaque (advanced) appearance. Lesion moves on parallax views	Blunted root apex. Asymmetrical loss of root
Root canal	Canal expands into lesions	Canal expands into lesions	Intact in early EIR, perforation in advanced cases	Intact	Intact, but perforation in advanced cases	Intact

EIR, External inflammatory resorption; *PDL*, periodontal ligament.

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References

1. Andersen M, Lund A, Andreasen JO, et al: *In vitro solubility of human pulp tissue in calcium hydroxide and sodium hypochlorite*, Endod Dent Traumatol 3, 1992;8: 104-108.
2. Andreasen FM, Pedersen BV. *Prognosis of luxated permanent teeth—the development of pulp necrosis*, Endod Dent Traumatol 6, 1985;1: 207-220.
3. Andreasen FM, Sewerin I, Mandel U, et al: *Radiographic assessment of simulated root resorption cavities*, Endod Dent Traumatol 1, 1987;3: 21-27.
4. Andreasen JO. *Review of root resorption systems and models. Etiology of root resorption and the homeostatic mechanisms of the periodontal ligament*, Davidovitch Z The biological mechanisms of tooth eruption and root resorption 1988; EBSCO Media Birmingham 9-21.
5. Andreasen J. *Avulsions*, Andreasen JO Andreasen FM Andersson L Textbook and color atlas of traumatic injuries to the teeth ed 4 2007; Blackwell Munksgaard Copenhagen.
6. Andreasen J, Andreasen F. *Root resorption following traumatic dental injuries. Proceedings of the Finnish Dental Society*, Suomen Hammaslaakariseuran Toimituksia 1992;88: 95-114.
7. Andreasen JO. *Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth*, Scand J Dent Res 3, 1970;78: 273-286.
8. Andreasen JO. *A time-related study of periodontal healing and root resorption activity after replantation of mature permanent incisors in monkeys*, Swed Dent J 3,

Telegram: @dental_k

www.konkur.in

1980;4: 101-110.

9. Andreasen JO. *Relationship between surface and inflammatory resorption and changes in the pulp after replantation of permanent incisors in monkeys*, J Endod 7, 1981;7: 294-301.
10. Andreasen JO, Borum MK, Andreasen FM. *Replantation of 400 avulsed permanent incisors. 3. Factors related to root growth*, Endod Dent Traumatol 2, 1995;11: 69-75.
11. Andreasen JO, Borum MK, Jacobsen HL, et al: *Replantation of 400 avulsed permanent incisors. 2. Factors related to pulpal healing*, Endod Dent Traumatol 2, 1995;11: 59-68.
12. Andreasen JO, Farik B, Munksgaard EC. *Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture*, Dent Traumatol 3, 2002;18: 134-137.
13. Andreasen JO, Hjorting-Hansen E. *Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss*, Acta Odontol Scand 3, 1966;24: 263-286.
14. Andreasen JO, Hjorting-Hansen E. *Replantation of teeth. II. Histological study of 22 replanted anterior teeth in humans*, Acta Odontol Scand 3, 1966;24: 287-306.
15. Andreasen JO, Lovschall H. *Response of oral tissues to trauma*, Traumatic injuries to the teeth 2007; Blackwell Pub. Professional Oxford 62-113.
16. Aqrabawi J. *Sealing ability of amalgam, super EBA cement, and MTA when used as retrograde filling materials*, Br Dent J 5, 2000;188: 266-268.
17. Ashrafi MH, Sadeghi EM. *Idiopathic multiple internal resorption: report of case*, ASDC J Dent Child 3, 1980;47: 196-199.
18. Bell T. *The anatomy, physiology, and diseases of the*

Telegram: @dental_k

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teeth, The North American Medical and Surgical Journal (1826-1831) 19, 1830;10: 63- ARTICLE V.

19. Bender IB, Seltzer S. *Roentgenographic and direct observation of experimental lesions in bone: I. 1961*, J Endod 11, 2003;29: 702-706; discussion 701.
20. Bhuva B, Barnes JJ, Patel S. *The use of limited cone beam computed tomography in the diagnosis and management of a case of perforating internal root resorption*, Int Endod J 8, 2011;44: 777-786.
21. Bille ML, Kvetny MJ, Kjaer I. *A possible association between early apical resorption of primary teeth and ectodermal characteristics of the permanent dentition*, Eur J Orthod 4, 2008;30: 346-351.
22. Bille ML, Nolting D, Kvetny MJ, et al: *Unexpected early apical resorption of primary molars and canines*, Eur Arch Paediatr Dent 3, 2007;8: 144-149.
23. Boyce BF, Xing L. *Functions of RANKL/RANK/OPG in bone modeling and remodeling*, Arch Biochem Biophys 2, 2008;473: 139-146.
24. Burleson A, Nusstein J, Reader A, et al: *The in vivo evaluation of hand/rotary/ultrasound instrumentation in necrotic, human mandibular molars*, J Endod 7, 2007;33: 782-787.
25. Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 5, 1985;1: 170-175.
26. Cabrini RL, Manfredi EE. *Internal resorption of dentine; histopathologic control of eight cases after pulp amputation and capping with calcium hydroxide*, Oral Surg Oral Med Oral Pathol 1, 1957;10: 90-96.

Telegram: @dental_k

www.konkur.in

27. Caliskan MK, Turkun M. *Prognosis of permanent teeth with internal resorption: a clinical review*, Endod Dent Traumatol 2, 1997;13: 75-81.
28. Camilleri J. *Investigation of Biodentine as dentine replacement material*, J Dent 7, 2013;41: 600-610.
29. Chow J, Chambers TJ. *An assessment of the prevalence of organic material on bone surfaces*, Calcif Tissue Int 2, 1992;50: 118-122.
30. Chung CJ, Soma K, Rittling SR, et al: *OPN deficiency suppresses appearance of odontoclastic cells and resorption of the tooth root induced by experimental force application*, J Cell Physiol 3, 2008;214: 614-620.
31. Cohenca N, Simon JH, Mathur A, et al: *Clinical indications for digital imaging in dento-alveolar trauma. Part 2: root resorption*, Dent Traumatol 2, 2007;23: 105-113.
32. Crona-Larsson G, Bjarnason S, Noren JG. *Effect of luxation injuries on permanent teeth*, Endod Dent Traumatol 5, 1991;7: 199-206.
33. Cvek M. *Treatment of non-vital peranent incisors with calcium hydroxide, II. Effect on external root resorption in luxated teeth compared with effect of root filling with guttapercha. A foolow-up*, Odont Revy 1973;24: 343-354.
34. Cvek M. *Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. A retrospective clinical study*, Endod Dent Traumatol 2, 1992;8: 45-55.
35. Cvek M. *Endodontic management and the use of calcium hydroxide in traumatized permanent teeth*, Textbook and color atlas of traumatic injuries to the teeth ed 4 2007; Blackwell Munksgaard Ames, IA 598-657.
36. D'Addazio PS, Campos CN, Özcan M, et al: *A*

Telegram: @dental_k

www.konkur.in

comparative study between cone-beam computed tomography and periapical radiographs in the diagnosis of simulated endodontic complications, Int Endod J 3, 2011;44: 218-224.

37. Dumsha T, Hovland EJ. *Evaluation of long-term calcium hydroxide treatment in avulsed teeth—an in vivo study*, Int Endod J 1, 1995;28: 7-11.
38. Durack C, Patel S. *Cone beam computed tomography in endodontics*, Braz Dent J 3, 2012;23: 179-191.
39. Ehnevid H, Lindskog S, Jansson L, et al: *Tissue formation on cementum surfaces in vivo*, Swed Dent J 1-2, 1993;17: 1-8.
40. Estrela C, Bueno MR, De Alencar AH, et al: *Method to evaluate inflammatory root resorption by using cone beam computed tomography*, J Endod 11, 2009;35: 1491-1497.
41. Diangelis AJ, Andreasen JO, Ebeleseder KA, et al: *Guidelines for the management of traumatic dental injuries. I. Fractures and luxations of permanent teeth*, Dent Traumatol 2, 2007;23: 66-71.
42. Flores MT, Andersson L, Andreasen JO, et al: *Guidelines for the management of traumatic dental injuries. II. Avulsion of permanent teeth*, Dent Traumatol 3, 2007;23: 130-136.
43. Frank AL, Bakland LK. *Nonendodontic therapy for supraosseous extracanal invasive resorption*, J Endod 7, 1987;13: 348-355.
44. Furseth R. *The resorption processes of human deciduous teeth studied by light microscopy, microradiography and electron microscopy*, Arch Oral Biol 4, 1968;13: 417-431.
45. Fuss Z, Tsesis I, Lin S. *Root resorption—diagnosis, classification and treatment choices based on stimulation factors*, Dent Traumatol 4, 2003;19: 175-182.

Telegram: @dental_k

www.konkur.in

46. Gartner AH, Mack T, Somerlott RG, et al: *Differential diagnosis of internal and external root resorption*, J Endod 11, 1976;2: 329-334.
47. Gencoglu N, Yildirim T, Garip Y, et al: *Effectiveness of different gutta-percha techniques when filling experimental internal resorptive cavities*, Int Endod J 10, 2008;41: 836-842.
48. Gold SI, Hasselgren G. *Peripheral inflammatory root resorption: a review of the literature with case reports*, J Clin Periodontol 8, 1992;19: 523-534.
49. Goldberg F, Massone EJ, Esmoris M, et al: *Comparison of different techniques for obturating experimental internal resorptive cavities*, Dent Traumatol 3, 2000;16: 116-121.
50. Gröndahl HG, Huumonen S. *Radiographic manifestations of periapical inflammatory lesions: how new radiological techniques may improve endodontic diagnosis and treatment planning*, Endod Topics 1, 2004;8: 55-67.
51. Gulabivala K, Searson LJ. *Clinical diagnosis of internal resorption: an exception to the rule*, Int Endod J 5, 1995;28: 255-260.
52. Haapasalo M, Endal U. *Internal inflammatory root resorption: the unknown resorption of the tooth*, Endod Topics 1, 2006;14: 60-79.
53. Hammarström L, Lindskog S. *General morphological aspects of resorption of teeth and alveolar bone*, Int Endod J 2, 1985;18: 93-108.
54. Harokopakis-Hajishengallis E. *Physiologic root resorption in primary teeth: molecular and histological events*, J Oral Sci 1, 2007;49: 1-12.
55. Heithersay GS. *Invasive cervical resorption: an analysis of potential predisposing factors*, Quintessence Int 2, 1999;30: 83-95.

Telegram: @dental_k

www.konkur.in

56. Heithersay GS. *Invasive cervical resorption*, Endod Topics 1, 2004;7: 73-92.
57. Hsien HC, Cheng YA, Lee YL, et al: *Repair of perforating internal resorption with mineral trioxide aggregate: a case report*, J Endod 8, 2003;29: 538-539.
58. Ishijima M, Rittling SR, Yamashita T, et al: *Enhancement of osteoclastic bone resorption and suppression of osteoblastic bone formation in response to reduced mechanical stress do not occur in the absence of osteopontin*, J Exp Med 3, 2001;193: 399-404.
59. Jacobovitz M, De Lima R. *Treatment of inflammatory internal root resorption with mineral trioxide aggregate: a case report*, Int Endod J 10, 2008;41: 905-912.
60. Jones SJ, Boyd A. *The resorption of dentine and cementum in vivo and in vitro*, Davidotch Z Biological mechanisms of tooth eruption and root resorption 1988; EBSCO Media Birmingham, AL.
61. Kundel HL, Revesz G. *Lesion conspicuity, structured noise, and film reader error*, AJR Am J Roentgenol 6, 1976;126: 1233-1238.
62. Laux M, Abbott PV, Pajarola G, et al: *Apical inflammatory root resorption: a correlative radiographic and histological assessment*, Int Endod J 6, 2000;33: 483-493.
63. Levander E, Malmgren O. *Evaluation of the risk of root resorption during orthodontic treatment: a study of upper incisors*, Eur J Orthod 1, 1988;10: 30-38.
64. Levin L, Trope M. *Root resorption*, Hargreaves KM Goodis HE Seltzer and Bender's dental pulp 2002; Quintessence Publishing Chicago, IL 425-448.
65. Li B, Long X, Wang S, et al: *Clinical and radiologic features of desmoplastic ameloblastoma*, J Oral Maxillofac

Telegram: @dental_k

www.konkur.in

Surg 8, 2011;69: 2173-2185.
66. Lindskog S, Blomlof L, Hammarstrom L. *Repair of periodontal tissues in vivo and in vitro*, J Clin Periodontol 2, 1983;10: 188-205.
67. Lyroudia KM, Dourou VI, Pantelidou OC, et al: *Internal root resorption studied by radiography, stereomicroscope, scanning electron microscope and computerized 3D reconstructive method*, Dent Traumatol 3, 2002;18: 148-152.
68. Main C, Mirzayan N, Shabahang S, et al: *Repair of root perforations using mineral trioxide aggregate: a long-term study*, J Endod 2, 2004;30: 80-83.
69. Mavridou AM, Bergmans L, Barendregt D, et al: *Descriptive analysis of factors associated with external cervical resorption*, J Endod 10, 2017;43: 1602-1610.
70. Mavridou AM, Hauben E, Wevers M, et al: *Understanding external cervical resorption in vital teeth*, J Endod 12, 2016;42: 1737-1751.
71. Mavridou AM, Hilkens P, Lambrichts I, et al: *Is hypoxia related to external cervical resorption? a case report*, J Endod 4, 2019;45: 459-470.
72. McHugh KP, Shen Z, Crotti TN, et al: *Role of cell-matrix interactions in osteoclast differentiation*, Adv Exp Med Biol 2007;602: 107-111.
73. McMichen FR, Pearson G, Rahbaran S, et al: *A comparative study of selected physical properties of five root-canal sealers*, Int Endod J 9, 2003;36: 629-635.
74. Mitchell PJ, Pitt Ford TR, Torabinejad M, et al: *Osteoblast biocompatibility of mineral trioxide aggregate*, Biomaterials 2, 1999;20: 167-173.
75. Nair PN, Henry S, Cano V, et al: *Microbial status of apical root canal system of human mandibular first molars*

Telegram: @dental_k

www.konkur.in

with primary apical periodontitis after "one-visit" endodontic treatment, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2, 2005;99: 231-252.

76. Nakamura I, Duong LT, Rodan SB, et al: *Involvement of a v b 3 integrins in osteoclast function*, J Bone Miner Metab 6, 2007;25: 337-344.
77. Ne RF, Witherspoon DE, Gutmann JL. *Tooth resorption*, Quintessence Int 1, 1999;30: 9-25.
78. Neuvald L, Consolaro A. *Cementoenamel junction: microscopic analysis and external cervical resorption*, J Endod 9, 2000;26: 503-508.
79. Nilsen R, Magnusson BC. *Enzyme histochemistry of induced heterotropic bone formation in guinea-pigs*, Arch Oral Biol 10-11, 1979;24: 833-841.
80. Nowicka A, Lipski M, Parafiniuk M, et al: *Response of human dental pulp capped with biodentine and mineral trioxide aggregate*, J Endod 6, 2013;39: 743-747.
81. Patel S, Dawood A. *The use of cone beam computed tomography in the management of external cervical resorption lesions*, Int Endod J 9, 2007;40: 730-737.
82. Patel S, Dawood A, Whaites E, et al: *New dimensions in endodontic imaging: part 1. Conventional and alternative radiographic systems*, Int Endod J 6, 2009;42: 447-462.
83. Patel S, Dawood A, Wilson R, et al: *The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography - an in vivo investigation*, Int Endod J 9, 2009;42: 831-838.
84. Patel S, Ford TP. *Is the resorption external or internal?*, Dent Update 4, 2007;34: 218-220 222, 224-6, 229.
85. Patel S, Foschi F, Condon R, et al: *External cervical resorption: part 2 - management*, Int Endod J 11, 2018;51: 1224-1238.

Telegram: @dental_k

www.konkur.in

86. Patel S, Foschi F, Mannocci F, et al: *External cervical resorption: a three-dimensional classification*, Int Endod J 2, 2018;51: 206-214.
87. Patel S, Kanagasingam S, Pitt Ford T. *External cervical resorption: a review*, J Endod 5, 2009;35: 616-625.
88. Patel S, Ricucci D, Durak C, et al: *Internal root resorption: a review*, J Endod 7, 2010;36: 1107-1121.
89. Patel S, Saberi N. *The ins and outs of root resorption*, Br Dent J 9, 2018;224: 691-699.
90. Patel S, Vincer L. *Case report: single visit indirect pulp cap using biodentine*, Dental Update 2, 2017;44: 141-145.
91. Pierce AM. *Experimental basis for the management of dental resorption*, Endod Dent Traumatol 6, 1989;5: 255-265.
92. Rabinowitch BZ. *Internal resorption*, Oral Surg Oral Med Oral Pathol 2, 1972;33: 263-282.
93. Regan JD, Gutmann JL, Witherspoon DE. *Comparison of Diaket and MTA when used as root-end filling materials to support regeneration of the periradicular tissues*, Int Endod J 10, 2002;35: 840-847.
94. Revesz G, Kundel HL, Graber MA. *The influence of structured noise on the detection of radiologic abnormalities*, Invest Radiol 6, 1974;9: 479-486.
95. Ricucci D. *Apical limit of root canal instrumentation and obturation, part 1. Literature review*, Int Endod J 6, 1998;31: 384-393.
96. Ricucci D, Langeland K. *Apical limit of root canal instrumentation and obturation, part 2. A histological study*, Int Endod J 6, 1998;31: 394-409.
97. Rodríguez G, Abella F, Durán-Sindreu F, et al: *Influence of cone-beam computed tomography in clinical decision making among specialists*, J Endod 2, 2017;43: 194-199.

Telegram: @dental_k

www.konkur.in

98. Rodríguez G, Patel S, Durán-Sindreu F, et al: *Influence of cone-beam computed tomography on endodontic retreatment strategies among general dental practitioners and endodontists*, J Endod 9, 2017;43: 1433-1437.
99. Salzano S, Tirone F. *Conservative nonsurgical treatment of class 4 invasive cervical resorption: a case series*, J Endod 11, 2015;41: 1907-1912.
100. Schaffner P, Dard M. *Structure and function of RGD peptides involved in bone biology*, Cell Mol Life Sci 1, 2003;60: 119-132.
101. Siqueira JF Jr, Rôças IN, Santos SR, et al: *Efficacy of instrumentation techniques and irrigation regimens in reducing the bacterial population within root canals*, J Endod 3, 2002;28: 181-184.
102. Sjögren U, Figdor D, Spångberg L, et al: *The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing*, Int Endod J 3, 1991;24: 119-125.
103. Soltanoff CS, Yang S, Chen W, et al: *Signaling networks that control the lineage commitment and differentiation of bone cells*, Crit Rev Eukaryot Gene Expr 1, 2009;19: 1-46.
104. Speziani C, Rivollier A, Gallois A, et al: *Murine dendritic cell transdifferentiation into osteoclasts is differentially regulated by innate and adaptive cytokines*, Eur J Immunol 3, 2007;37: 747-757.
105. Stamos DE, Stamos DG. *A new treatment modality for internal resorption*, J Endod 7, 1986;12: 315-319.
106. Suei Y, Taguchi A, Nagasaki T, et al: *Radiographic findings and prognosis of simple bone cysts of the jaws*, Dentomaxillofac Radiol 2, 2010;39: 65-71.
107. Tronstad L. *Root resorption—etiology, terminology and clinical manifestations*, Endod Dent Traumatol 6, 1988;4: 241-252.

Telegram: @dental_k

www.konkur.in

108. Trope M. *Root resorption of dental and traumatic origin: classification based on etiology*, Pract Periodontics Aesthet Dent 4, 1998;10: 515-522.
109. Trope M. *Root resorption due to dental trauma*, Endod Topics 1, 2002;1: 79-100.
110. Turkun M, Cengiz T. *The effects of sodium hypochlorite and calcium hydroxide on tissue dissolution and root canal cleanliness*, Int Endod J 5, 1997;30: 335-342.
111. Tyrovola JB, Spyropoulos MN, Makou M, et al: *Root resorption and the OPG/RANKL/RANK system: a mini review*, J Oral Sci 4, 2008;50: 367-376.
112. Uchiyama M, Nakamichi Y, Nakamura M, et al: *Dental pulp and periodontal ligament cells support osteoclastic differentiation*, J Dent Res 7, 2009;88: 609-614.
113. Vaz de Souza D, Schirru E, Mannocci F, et al: *External cervical resorption: a comparison of the diagnostic efficacy using 2 different cone-beam computed tomographic units and periapical radiographs*, J Endod 1, 2017;43: 121-125.
114. Wada N, Maeda H, Tanabe K, et al: *Periodontal ligament cells secrete the factor that inhibits osteoclastic differentiation and function: the factor is osteoprotegerin/osteoclastogenesis inhibitory factor*, J Periodontal Res 1, 2001;36: 56-63.
115. Walton RE, Leonard LA. *Cracked tooth: an etiology for "idiopathic" internal resorption?*, J Endod 4, 1986;12: 167-169.
116. Webber RL, Messura JK. *An in vivo comparison of diagnostic information obtained from tuned-aperture computed tomography and conventional dental radiographic imaging modalities*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2, 1999;88: 239-247.

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www.konkur.in

117. Wedenberg C. *Evidence for a dentin-derived inhibitor of macrophage spreading,* Scand J Dent Res 5, 1987;95: 381-388.
118. Wedenberg C, Lindskog S. *Experimental internal resorption in monkey teeth*, Endod Dent Traumatol 6, 1985;1: 221-227.
119. Wedenberg C, Lindskog S. *Evidence for a resorption inhibitor in dentin*, Scand J Dent Res 3, 1987;95: 205-211.
120. Wedenberg C, Yumita S. *Evidence for an inhibitor of osteoclast attachment in dentinal matrix*, Endod Dent Traumatol 6, 1990;6: 255-259.
121. Wedenberg C, Zetterqvist L. *Internal resorption in human teeth—a histological, scanning electron microscopic, and enzyme histochemical study*, J Endod 6, 1987;13: 255-259.
122. Wiener BH, Schilder H. *A comparative study of important physical properties of various root canal sealers. II. Evaluation of dimensional changes,* Oral Surg Oral Med Oral Pathol 6, 1971;32: 928-937.
123. Wilson P, Barnes I. *Treatment of internal root resorption with thermoplasticized gutta-percha. A case report*, Int Endod J 2, 1987;20: 94-97.
124. Yamaoka M, Furusawa K, Ikeda M, et al: *Root resorption of mandibular second molar teeth associated with the presence of the third molars*, Aust Dent J 2, 1999;44: 112-116.
125. Yildirim S, Yapar M, Sermet U, et al: *The role of dental pulp cells in resorption of deciduous teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1, 2008;105: 113-120.

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19: Management of endodontic emergencies

Fabricio B. Teixeira, Gary S.P. Cheung*

CHAPTER OUTLINE

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Emergency classifications

The proper diagnosis and effective management of acute dental pain are possibly the most rewarding and satisfying aspects of providing dental care. An endodontic emergency is defined as pain or swelling caused by various stages of inflammation or infection of the pulpal or periradicular tissues. The cause of dental pain originates typically from caries, deep or defective restorations, or traumatic injuries. Sometimes occlusion-related issues such as cracks can also mimic acute dental pain (Fig. 19.1). Bender[9] stated that patients who manifest severe or referred pain almost always had a previous history of pain with the offending tooth. Approximately 85% of all dental emergencies arise as a result of pulpal or periapical disease, which would necessitate either extraction or endodontic treatment to relieve the symptoms.[43,75] It has also been estimated that about 12% of the United States population experienced a toothache in the preceding 6 months.[71]

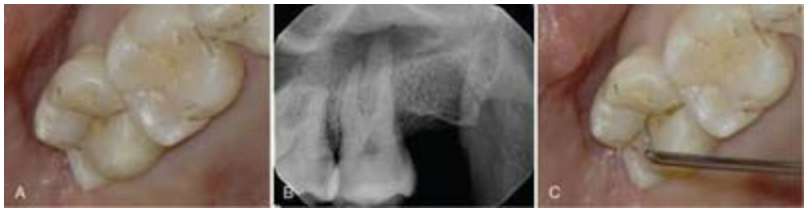

FIG. 19.1 **A,** Patient complained of acute pain on biting at the upper left molar (tooth #15). **B,** Clinical thermal tests revealed pulpal necrosis and a preoperative radiograph taken (periapical lesions noted). **C,** The tooth was split in half.

A) Close-up view shows occlusal view of two teeth with cracks.

B) Radiograph shows two teeth. The tooth on left has radiolucent spot in the pulp chamber. The gum line has porous tissue.

C) Close-up view shows a probe being pointed out at the fracture in tooth that

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has cracks on it.

Determining a definitive diagnosis can sometimes be challenging and even frustrating for clinicians, but a methodical, objective, and subjective evaluation is imperative before developing a proper treatment plan. Unfortunately, on the basis of the diagnosis, there are conflicting opinions on how to best clinically manage various endodontic emergencies. Surveys obtained from board-certified endodontists in 1977,[25,26] 1990,[34] and 2009[69] proposed to define seven conditions as endodontic emergencies. However, due to terminology changes, five clinical presentations are presently considered legitimate emergencies:

1. Symptomatic irreversible pulpitis with normal apical tissues
2. Symptomatic irreversible pulpitis with symptomatic apical periodontitis
3. Necrotic pulp with symptomatic apical periodontitis
4. Necrotic pulp with fluctuant intraoral swelling
5. Necrotic pulp with diffuse facial swelling

There are other endodontic emergencies such as traumatic dental injuries, previously endodontic treated teeth with either symptomatic apical periodontitis or acute apical abscess, and endodontic flare-ups that may occur between treatment sessions. Of course, there are also many types of facial pain that have a nonodontogenic origin; these are described in detail in Chapter 4.

There have been several changes in the determined clinical management of endodontic emergencies over the years. Many of these treatment modifications have occurred because of the more contemporary armamentarium and materials as well as new evidence-based research and the presumption of empirical clinical success.

Emergency endodontic management

Because pain is both a psychological and biologic entity, the management of acute dental pain must take into consideration both the physical symptoms and the emotional status of the patient. The patient's needs, fears, and coping

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mechanisms must be compassionately understood. This assessment and the clinician's ability to build rapport with the patient are critical factors in the comprehensive success of the patient's management.[9,33,54,99]

The methodical steps for determining an accurate diagnosis, based on evaluation of the patient's chief complaint, review of the medical history, and the protocols used for an objective and subjective diagnosis, are described in detail in Chapter 1. Once it has been determined that endodontic treatment is necessary, it is incumbent on the clinician to take the proper steps necessary to manage the acute dental emergency.

The clinician has a responsibility to inform the patient of the recommended treatment plan and to advise the patient of the treatment alternatives, the risks, and benefits that pertain, and the expected prognosis under the present circumstances. Given this information, the patient may elect extraction over endodontics or possibly request a second opinion. The treatment plan should never be forced on a patient. The informed course of treatment is made jointly between the patient and the clinician.

In the event of an endodontic emergency, the clinician must determine the optimal mode of treatment according to the diagnosis. Treatment may vary depending on the pulpal or periapical status, the intensity and duration of pain, and whether there is diffuse or fluctuant swelling. Paradoxically, as discussed later, the mode of therapy that we tend to choose has been directed more from surveys of practicing endodontists rather than from controlled clinical studies or research investigations.

Teeth with vital pulps

Teeth with vital pulps can have one of the following presentations:

- *Normal.* The teeth are asymptomatic with no objective pathoses.
- *Reversible pulpitis.* There is a reversible sensitivity to cold or osmotic changes (i.e., sweet, salty, and sour).
- *Irreversible pulpitis.* The sensitivity to temperature changes is more intense and with a longer duration.

Reversible pulpitis

Reversible pulpitis can be induced by caries, exposed dentin, recent dental

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treatment, and defective restorations. Conservative removal of caries, protection of dentin, and a proper restoration will typically resolve the symptoms. However, the symptoms from exposed dentin, specifically from gingival recession and cervically exposed roots, can often be difficult to alleviate. Topical applications of desensitizing agents and the use of certain dentifrices have been helpful in the management of dentin hypersensitivity; the etiology, physiology, and management of this are discussed in Chapter 13.

Irreversible pulpitis

The diagnosis of irreversible pulpitis can be subcategorized as asymptomatic or symptomatic. *Asymptomatic* irreversible pulpitis pertains to a tooth that has no symptoms, but has deep caries or tooth structure loss that, if left untreated, will cause the tooth to become symptomatic or nonvital. On the other hand, the pain from *symptomatic* irreversible pulpitis is often an emergency condition that requires immediate treatment. These teeth exhibit intermittent or spontaneous pain and are sensitive to thermal triggers. Exposure to extreme temperatures, especially cold, will elicit intense and prolonged episodes of pain, even after the source of the stimulus is removed.

In 1977,[25,26] 187 board-certified endodontists responded to a survey to determine how they would manage various endodontic emergencies. Ten years later, 314 board-certified endodontists responded to the same questionnaire in order to determine whether there had been any changes in how these emergencies were managed.[34] The clinical management of emergency treatment of a tooth with symptomatic irreversible pulpitis with or without normal periapical tissues seemed to be reasonably similar by removing the inflamed pulp tissue either by pulpotomy or complete instrumentation.[83] In a similar survey conducted in 2009,[69] most respondents stated that they would clean to the level of the “apex,” as confirmed with an electronic apex locator; this suggests a change in the management of endodontic cases based on the advent of a more contemporary armamentarium. In general, there is a trend toward more cleaning and shaping of the canal when symptomatic irreversible pulpitis presents with a normal periapex, compared with performing just pulpectomies as described in the 1977 survey. None of the individuals surveyed in the 1990 or 2009 poll

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stated that they would manage these emergencies by establishing any type of drainage by trephining the apex, making an incision, or leaving the tooth open for an extended period of time.

In addition, for vital teeth, the 1977 survey did not even broach the concept of completing the endodontics in one visit, whereas in the 1988 study about one third of the respondents indicated that they would complete these vital cases in a single visit and the response rose to 79% in the most recent survey. Since the early 1980s, there seems to have been an increase in the acceptability of providing endodontic therapy in one visit, especially in cases of vital pulps, with most studies revealing an equal number, or fewer, flare-ups after single-visit endodontic treatment.[27,86,91,96,98,106] However, this has not come without controversy, with some studies showing otherwise,[118] contending that there is more posttreatment pain after single-visit endodontics, and possibly a lower long-term success rate. Unfortunately, time constraints at the emergency visit often make the single-visit treatment option not practical.[4]

If root canal therapy is completed at a later date, medicating the canal with calcium hydroxide has been suggested to reduce the chances of bacterial growth in the canal between appointments in most studies.[20] One randomized clinical study showed that a dry cotton pellet was as effective in relieving pain as a pellet moistened with camphorated monochlorophenol (CMCP), metacresylacetate (cresatin), eugenol, or saline.[45] Sources of infection, such as caries and defective restorations, should be completely removed to prevent recontamination of the root canal system between appointments.[45] The concept of single- versus multiple-visit endodontics is described in greater detail in Chapter 17.

For emergency management of vital teeth that are not initially sensitive to percussion, occlusal reduction has not been shown to be beneficial.[22,34] However, the clinician should be cognizant of the possibility of occlusal interferences and prematurities that might cause tooth fracture under heavy mastication. In vital teeth in which the inflammation has extended periapically, which will present with pretreatment pain to percussion, occlusal reduction has been reported to reduce posttreatment pain.[34,82,97]

Antibiotics are not recommended for the emergency management of irreversible pulpitis (see Chapters 15 and 17),[59,107] as placebo-controlled

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clinical trials have demonstrated that antibiotics have no effect on pain levels in patients with irreversible pulpitis.[80]

Most endodontists and endodontic textbooks recommend the emergency management of *symptomatic* irreversible pulpitis to involve the initiation of root canal treatment,[20,34,44,69,111] with complete pulp removal and total debridement of the root canal system. Unfortunately, in an emergency situation, the allotted time necessary for this treatment is often an issue. Given the potential time constraints and inevitable differences in skill level between clinicians, it may not be feasible to complete the total canal cleaning at the initial emergency visit. Subsequently, especially with multirooted teeth, a pulpotomy (removal of the coronal pulp) has been advocated for emergency treatment of irreversible pulpitis.[17,44,103] In a clinical study of various emergency procedures, it has been demonstrated that this treatment is highly effective for alleviating acute dental pain due to irreversible pulpitis.[17] A randomized clinical trial showed that pulpotomy was an excellent alternative for emergency relief of pain.[32] These findings were similar to the results seen in a recent study,[5] which reported more pain relief after pulpotomy as compared to the root canals treatment.

To assist the clinician in assessing the level of difficulty of a given endodontic case, the American Association of Endodontists (Chicago, IL) has developed the “AAE Endodontic Case Difficulty Assessment Form and Guidelines” (Fig. 19.2). This form is intended to make case selection more efficient, more consistent, and easier to document, as well as to provide a more objective ability to determine when it may be necessary to refer the patient to another clinician who may be better able to manage the complexities of the case. Haug et al. evaluated the impact of the case difficulty on endodontic mishaps in an undergraduate student clinic. They emphasized the importance and relevance of case difficulty assessment. Their results showed that the American Association of Endodontists (AAE) case difficulty assessment form is a valuable tool to predict potential mishaps and the number of treatment visits.[46]

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AAE Endodontic Case Difficulty Assessment Form and Guidelines

PATIENT INFORMATION

Name_____

Address_____

City/State/Zip_____

Phone_____

DISPOSITION

Treat in Office: Yes ☐ No ☐

Refer Patient to:

_____

Date:_____

Guidelines for Using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

LEVELS OF DIFFICULTY

MINIMAL DIFFICULTY	Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the MINIMAL DIFFICULTY category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.
MODERATE DIFFICULTY	Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the MODERATE DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.
HIGH DIFFICULTY	Preoperative condition is exceptionally complicated, exhibiting several factors listed in the MODERATE DIFFICULTY category or at least one in the HIGH DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way.

© American Association of Endodontists, 211 E. Chicago Ave., Suite 1100, Chicago, IL 60611-2691; Phone: 800/872-3636 or 312/266-7255; Fax: 866/451-9020 or 312/266-9867; E-mail: info@aae.org; Web site: www.aae.org

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AAE Endodontic Case Difficulty Assessment Form

Criteria and Subcriteria	Minimal Difficulty	Moderate Difficulty	High Difficulty
A. PATIENT CONSIDERATIONS			
Medical history	☐ No medical problem (ASA Class 1*)	☐ One or more medical problems (ASA Class 2*)	☐ Complex medical history/serious illness/disability (ASA Classes 3-5*)
Anesthesia	☐ No history of anesthesia problems	☐ Vasoconstrictor intolerance	☐ Difficulty achieving anesthesia
Patient disposition	☐ Cooperative and compliant	☐ Anxious but cooperative	☐ Uncooperative
Ability to open mouth	☐ No limitation	☐ Slight limitation in opening	☐ Significant limitation in opening
Gag reflex	☐ None	☐ Gags occasionally with radiographs/treatment	☐ Extreme gag reflex which has compromised past dental care
Emergency condition	☐ Minimum pain or swelling	☐ Moderate pain or swelling	☐ Severe pain or swelling
B. DIAGNOSTIC AND TREATMENT CONSIDERATIONS			
Diagnosis	☐ Signs and symptoms consistent with recognized pulpal and periapical conditions	☐ Extensive differential diagnosis of usual signs and symptoms required	☐ Confusing and complex signs and symptoms; difficult diagnosis ☐ History of chronic oral/facial pain
Radiographic difficulties	☐ Minimal difficulty obtaining/interpreting radiographs	☐ Moderate difficulty obtaining/interpreting radiographs (e.g., high floor of mouth, narrow or low palatal vault, presence of tori)	☐ Extreme difficulty obtaining/interpreting radiographs (e.g., superimposed anatomical structures)
Position in the arch	☐ Anterior/premolar ☐ Slight inclination (<10°) ☐ Slight rotation (<10°)	☐ 1st molar ☐ Moderate inclination (10-30°) ☐ Moderate rotation (10-30°)	☐ 2nd or 3rd molar ☐ Extreme inclination (>30°) ☐ Extreme rotation (>30°)
Tooth isolation	☐ Routine rubber dam placement	☐ Simple pretreatment modification required for rubber dam isolation	☐ Extensive pretreatment modification required for rubber dam isolation
Morphologic aberrations of crown	☐ Normal original crown morphology	☐ Full coverage restoration ☐ Porcelain restoration ☐ Bridge abutment ☐ Moderate deviation from normal tooth/root form (e.g., taurodontism, microdens) ☐ Teeth with extensive coronal destruction	☐ Restoration does not reflect original anatomy/alignment ☐ Significant deviation from normal tooth/root form (e.g., fusion, dens in dente)
Canal and root morphology	☐ Slight or no curvature (<10°) ☐ Closed apex <1 mm diameter	☐ Moderate curvature (10-30°) ☐ Crown axis differs moderately from root axis. Apical opening 1-1.5 mm in diameter	☐ Extreme curvature (>30°) or S-shaped curve ☐ Mandibular premolar or anterior with 2 roots ☐ Maxillary premolar with 3 roots ☐ Canal divides in the middle or apical third ☐ Very long tooth (>25 mm) ☐ Open apex (>1.5 mm in diameter)
Radiographic appearance of canal(s)	☐ Canal(s) visible and not reduced in size	☐ Canal(s) and chamber visible but reduced in size ☐ Pulp stones	☐ Indistinct canal path ☐ Canal(s) not visible
Resorption	☐ No resorption evident	☐ Minimal apical resorption	☐ Extensive apical resorption ☐ Internal resorption ☐ External resorption
C. ADDITIONAL CONSIDERATIONS			
Trauma history	☐ Uncomplicated crown fracture of mature or immature teeth	☐ Complicated crown fracture of mature teeth ☐ Subluxation	☐ Complicated crown fracture of immature teeth ☐ Horizontal root fracture ☐ Alveolar fracture ☐ Intrusive, extrusive or lateral luxation ☐ Avulsion
Endodontic treatment history	☐ No previous treatment	☐ Previous access without complications	☐ Previous access with complications (e.g., perforation, non-negotiated canal, ledge, separated instrument) ☐ Previous surgical or nonsurgical endodontic treatment completed
Periodontal-endodontic condition	☐ None or mild periodontal disease	☐ Concurrent moderate periodontal disease	☐ Concurrent severe periodontal disease ☐ Cracked teeth with periodontal complications ☐ Combined endodontic/periodontic lesion ☐ Root amputation prior to endodontic treatment

*American Society of Anesthesiologists (ASA) Classification System:
Class 1: No systemic illness. Patient healthy.
Class 2: Patient with mild degree of systemic illness, but without functional restrictions, e.g., well-controlled hypertension.
Class 3: Patient with severe degree of systemic illness which limits activities, but does not immobilize the patient.
Class 4: Patient with severe systemic illness that immobilizes and is sometimes life threatening.
Class 5: Patient will not survive more than 24 hours whether or not surgical intervention takes place.
www.asahq.org/clinical/physicalstatus.htm

FIG. 19.2 The American Association of Endodontists *(AAE)* Endodontic Case Difficulty Assessment Form and Guidelines, developed to assist the clinician in assessing the level of difficulty of a given endodontic case and to help determine when referral may be necessary.

AAE Endodontic Case Difficulty Assessment Form and Guidelines are shown along with AAE logo at the upper left corner.

The first panel shows patient information including its name, address, city/state/zip, and phone. To the right, the information under the heading disposition is as follows: treat in office (Yes or no each with blank check boxes), refer patient number, date.

The second panel shows guidelines for using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use

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in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

Levels of difficulty are as follows:

Minimal difficulty: Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the minimal difficulty category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.

Moderate difficulty: Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the moderate difficulty category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.

High difficulty: Preoperative condition is exceptionally complicated, exhibiting several factors listed in the moderate difficulty category or at least one in the high difficulty category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

Text at the bottom reads, The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way.

The continuation of AAE Endodontic Case Difficulty Assessment Form shows three sections as follows: A) Patient considerations, B) Diagnostic and treatment considerations, C) Additional considerations. Each section has list of queries under criteria and subcriteria, minimal difficulty, moderate difficulty, and high difficulty columns.

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Pulpal necrosis with symptomatic apical periodontitis

Over the years, the proper methodology for the emergency endodontic management of necrotic teeth has been controversial. In a 1977 survey of board-certified endodontists,[25,26] it was reported that, in the absence of swelling, most respondents would completely instrument the canals, keeping the file short of the radiographic apex. However, when swelling was present, the majority of those polled in 1977 preferred to leave the tooth open, with instrumentation extending beyond the apex to help facilitate drainage through the canals. Years later and again validated in a 2009 study, most respondents favored complete instrumentation regardless of the presence of swelling. Also, it was the decision of 25.2% to 38.5% of the clinicians to leave these teeth open in the event of diffuse swelling; 17.5% to 31.5% left the teeth open in the presence of a fluctuant swelling. However, as discussed later, there is currently a trend toward not leaving teeth open for drainage. There is also another trend: when treatment is done in more than one visit, most endodontists will use calcium hydroxide as an intracanal medicament.[69]

Care should be taken not to push necrotic debris beyond the apex during root canal instrumentation, as this has been shown to promote more posttreatment discomfort.[15,34,95,104] Crown-down instrumentation techniques have been shown to remove most of the debris coronally rather than pushing it out the apex. The use of positive-pressure irrigation methods, such as needle-and-syringe irrigation, also poses a risk of expressing debris or solution out of the apex.[12,23] Improvements in technology, such as electronic apex locators and use of the limited cone-beam computed tomography (CBCT) have facilitated increased accuracy in determining working length measurements.[74] An increased number of clinicians now use these devices.[62,69] Moreover, new negative-pressure irrigation systems may allow for a more thorough canal debridement with less apical extrusion of debris.[18,75]

The attenuation of postoperative pain in patients with this specific condition has continuously been evaluated in the literature. An outcome study indicated that the use of a negative apical pressure irrigation device significantly reduces the postoperative pain levels in comparison to conventional needle irrigation.[3] A randomized clinical trial demonstrated

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final irrigation with 20 mL sterile cold (2.5°C) saline solution delivered to the working length with a sterile, cold (2.5°C) EndoVac microcannula (Kerr Endo, Orange Country, CA) for 5 minutes.[116] They concluded that cryotherapy reduced the incidence of postoperative pain and the need for medication in patients presenting with a diagnosis of necrotic pulp and symptomatic apical periodontitis.

Trephination

In the absence of swelling, trephination is the surgical perforation of the alveolar cortical plate to release, from between the cortical plates, the accumulated inflammatory and infective tissue exudate that causes pain. Its use has been historically advocated to provide pain relief in patients with severe and recalcitrant periradicular pain.[25,26] The technique involves an engine-driven perforator entering through the cortical bone and into the cancellous bone, often without the need for an incision,[19] in order to provide a pathway for drainage from the periradicular tissues. Although more recent studies have failed to show the benefit of trephination in patients with irreversible pulpitis with symptomatic apical periodontitis[77] or necrotic teeth with symptomatic apical periodontitis,[82] there remain some advocates who recommend trephination for managing acute and intractable periapical pain.[51] The clinician should understand that local anesthesia may be difficult for cases with acute inflammation or infection.[55] Extreme care must be taken when carrying out a trephination procedure to guard against inadvertent and possibly irreversible injury to the tooth root or surrounding structures, such as the mental foramen, intra-alveolar nerve, or maxillary sinus.

Necrosis and single-visit endodontics

Although single-visit endodontic treatment for teeth diagnosed with irreversible pulpitis is not contraindicated,[2,91,93,98,119] performing single-visit endodontics on necrotic and previously treated teeth is not without controversy. In cases of necrotic teeth, although research[27] has indicated that there may be no difference in posttreatment pain if the canals are filled at the time of the emergency versus a later date, some studies[105,112] have questioned the long-term prognosis of such treatment, especially in cases of symptomatic apical periodontitis. Several studies,[28,66] including a CONSORT

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(Consolidated Standards of Reporting Trials) meta-analysis,[92] have shown no difference in outcome between single-visit and two-visit treatments. The concept of single- versus multi-visit endodontics is further discussed in Chapters 5 and 17.

Pulpal necrosis with acute apical abscess

Swelling

Tissue swelling may be associated with an acute apical abscess at the time of the initial emergency visit, or it may occur as an inter-appointment flare-up or as a postendodontic complication. Swellings may be localized or diffuse, fluctuant or firm. Localized swellings are confined within the oral cavity, whereas a diffuse swelling, or cellulitis, is more extensive, spreading through adjacent soft tissues and dissecting tissue spaces along fascial planes.[100]

Swelling may be controlled by establishing drainage through the root canal or by incising the fluctuant swelling. As discussed later and in Chapter 15, antibiotics may be recruited when there are systemic manifestations of the infection, such as fever and malaise. The principal modality for managing swelling secondary to endodontic infections is to achieve drainage and remove the source of the infection.[41] When the swelling is localized, the preferred avenue is drainage through the root canal (Fig. 19.3). Complete canal debridement and disinfection[42,114] are paramount for success regardless of observable drainage, because the presence of any bacteria remaining within the root canal system will compromise the resolution of the acute infection.[73] In the presence of persistent swelling, gentle finger pressure to the mucosa overlying the swelling may help facilitate drainage through the canal. Once the canals have been cleaned and allowed to dry, calcium hydroxide as the intracanal medicament[69] should be placed and the access properly sealed.[20,34,44]

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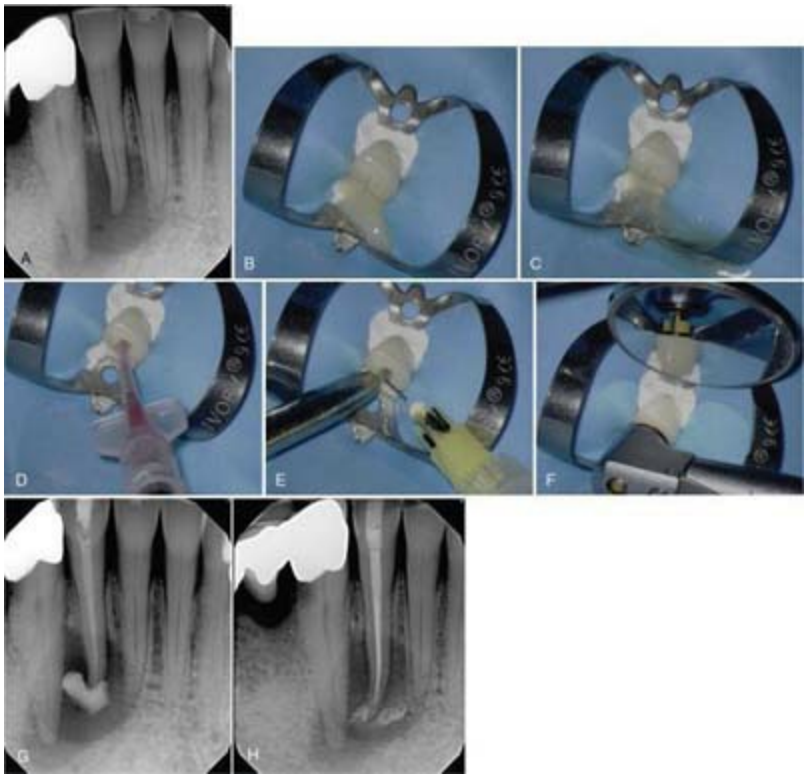

FIG. 19.3 Drainage of pus through the root canal. **A,** Acute apical abscess arising from the lower right lateral incisor with radiographic radiolucency. **B,** Initial drainage through the canal. **C,** Persistent drainage through the canal. **D,** Aspiration of the content with a plastic suction tip. **E,** Irrigation with NaOCl. **F,** Mechanical debridement. **G,** Placement of calcium hydroxide. **H,** Obturation of root canals during second visit.

Set of eight illustrations depicts steps involved in the removal of pus from tooth through root canal.

Fascial space infections

If bacteria from the infected root canal gain entry into the periradicular tissues and the immune system is unable to suppress the invasion, an otherwise healthy patient eventually shows signs and symptoms of an acute apical abscess, which can, in turn, evolve to cellulitis. Clinically, the patient experiences swelling and mild to severe pain. Depending on the relationship of the apices of the affected tooth to the muscular attachments, the swelling may be localized to the vestibule or extend into a fascial space. The patient may also have systemic manifestations, such as fever, chills,

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lymphadenopathy, headache, and nausea. Because the reaction to the infection may occur quickly, the involved tooth may or may not show radiographic evidence of a widened periodontal ligament space. In most cases, the tooth elicits a positive response to percussion, and the periradicular area is tender to palpation. The tooth now becomes a *focus of infection* because it leads to periradicular infection and secondary spread to the fascial spaces of the head and neck, resulting in cellulitis and systemic signs and symptoms of infection.

In such cases, treatment may involve incision for drainage, root canal treatment, or extraction to remove the source of the infection. Antibiotic therapy may be indicated in patients with compromised host resistance, the presence of systemic symptoms, or fascial space involvement. Fascial space infections of odontogenic origin are infections that have spread into the fascial spaces from the periradicular area of a tooth, the focus of infection. They are *not* examples of the theory of focal infection, which describes the dissemination of bacteria or their products from a distant focus of infection. Preferably, this is an example of the local spread of infection from an odontogenic source.

Fascial spaces are potential anatomic areas that exist between the fascia and underlying organs and other tissues. During infection, these spaces are formed as a result of the spread of purulent exudate. The spread of infections of odontogenic origin into the fascial spaces of the head and neck is determined by the location of the root end of affected tooth to its overlying buccal or lingual cortical plate and the relationship of the apex to the attachment of a muscle (Fig. 19.4, *A*). For example, if the source of the infection is a mandibular molar whose apices lie closer to the lingual cortical plate and *above* the attachment of the mylohyoid muscle of the floor of the mouth, the purulent exudate may break through the lingual cortical plate and into the *sublingual* space. However, if the apices lie *below* the attachment of the mylohyoid muscle, the infection may spread into the *submandibular* space.

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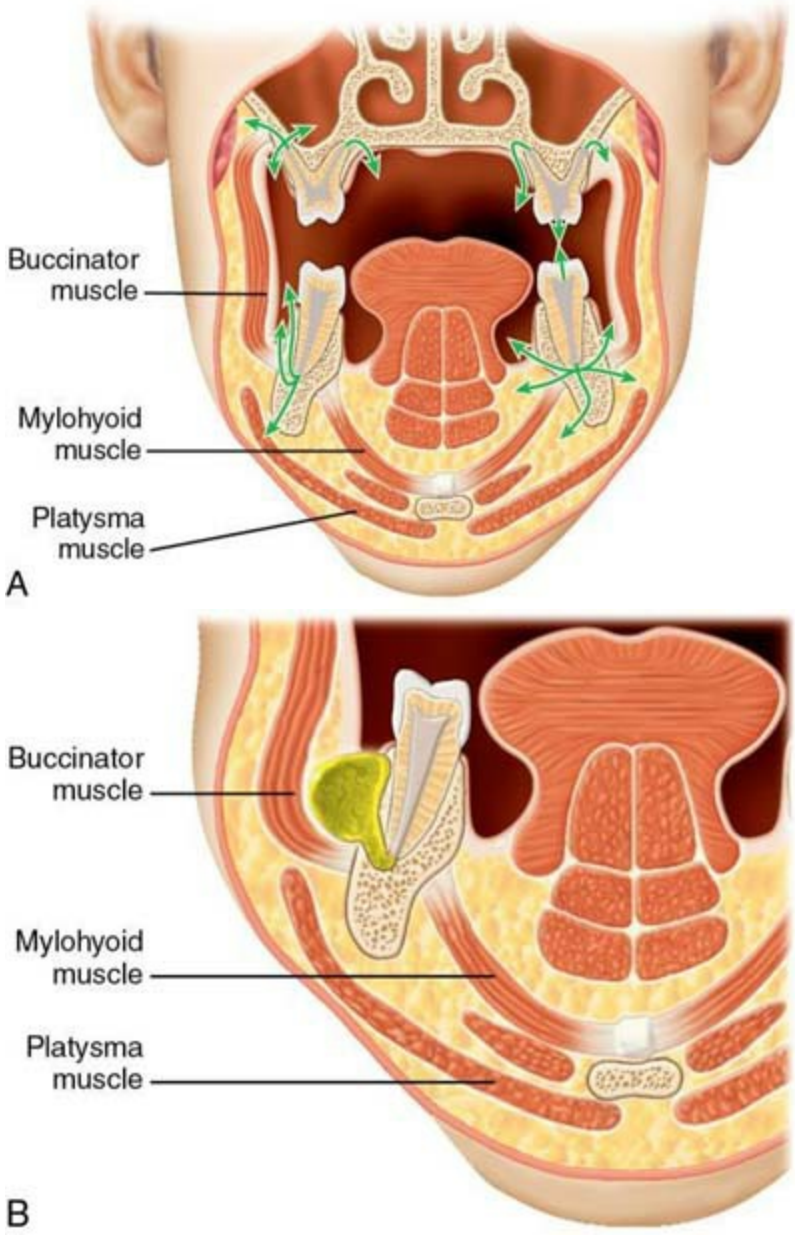

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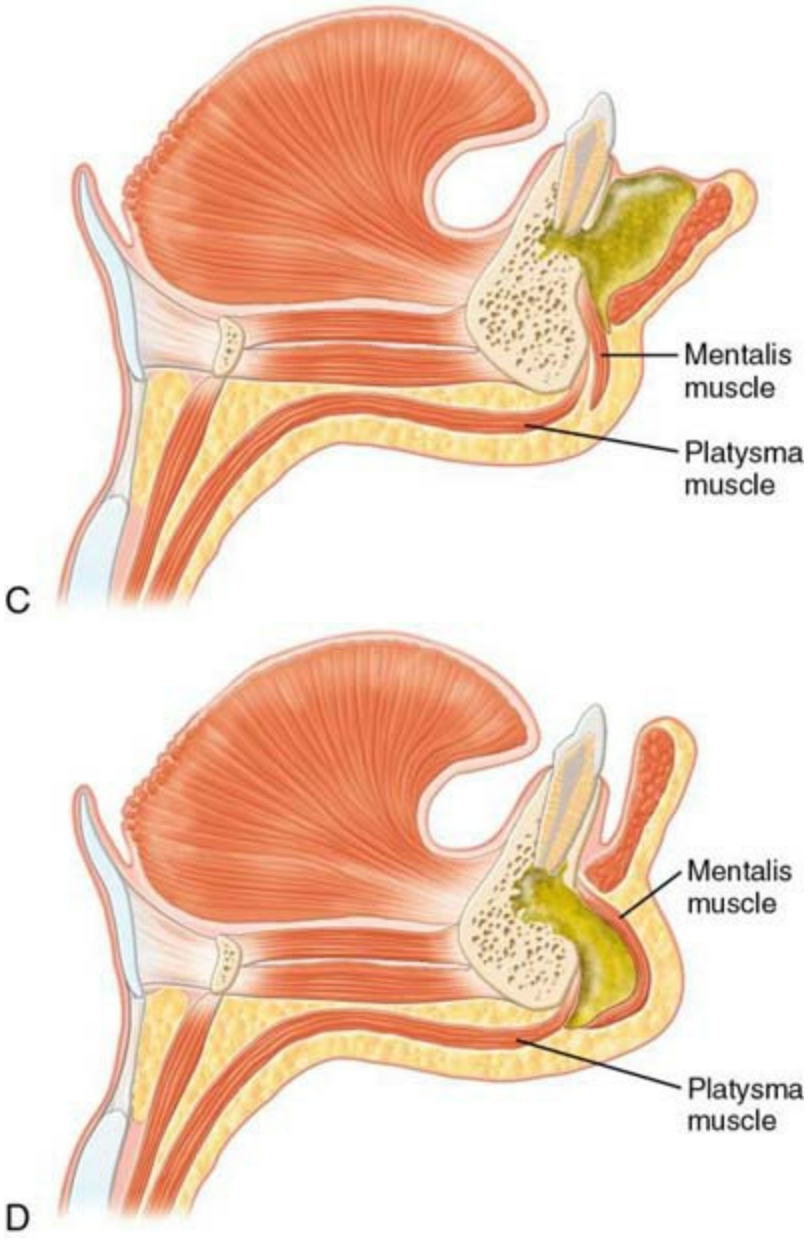

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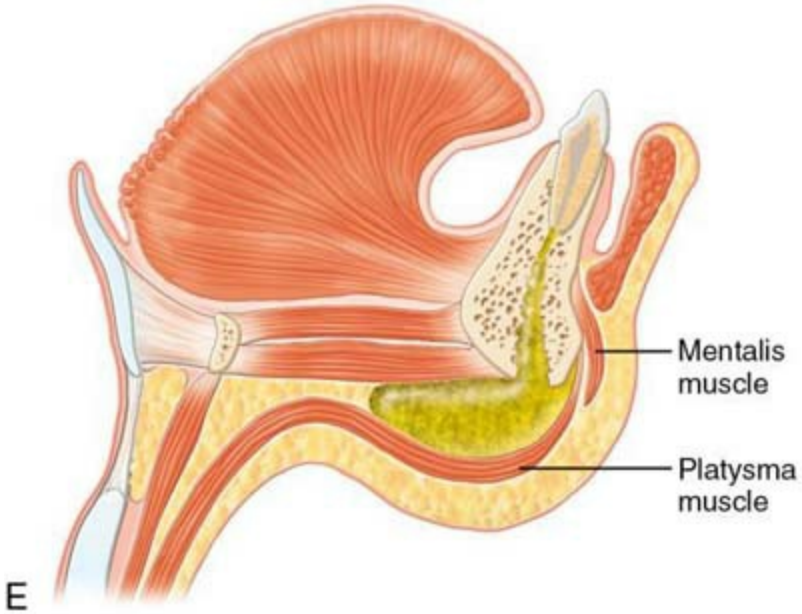

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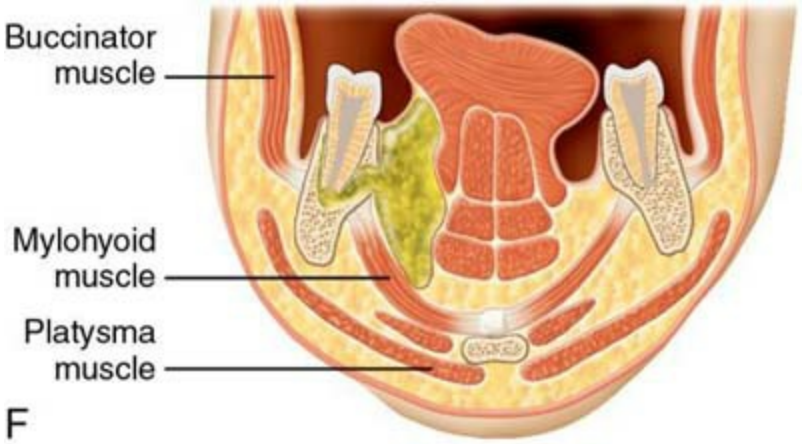

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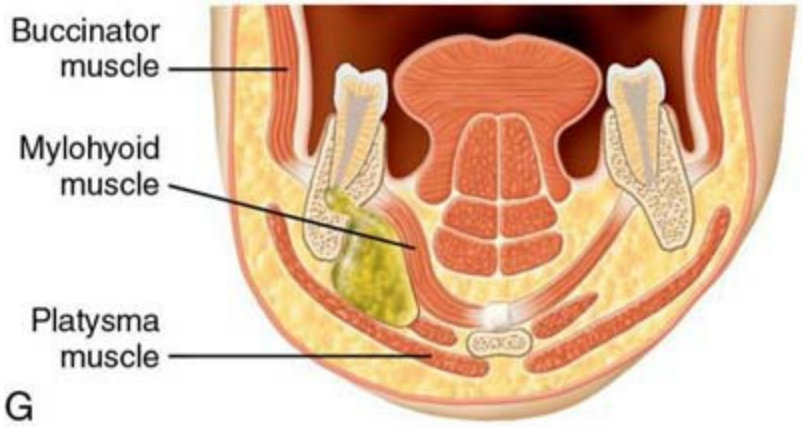

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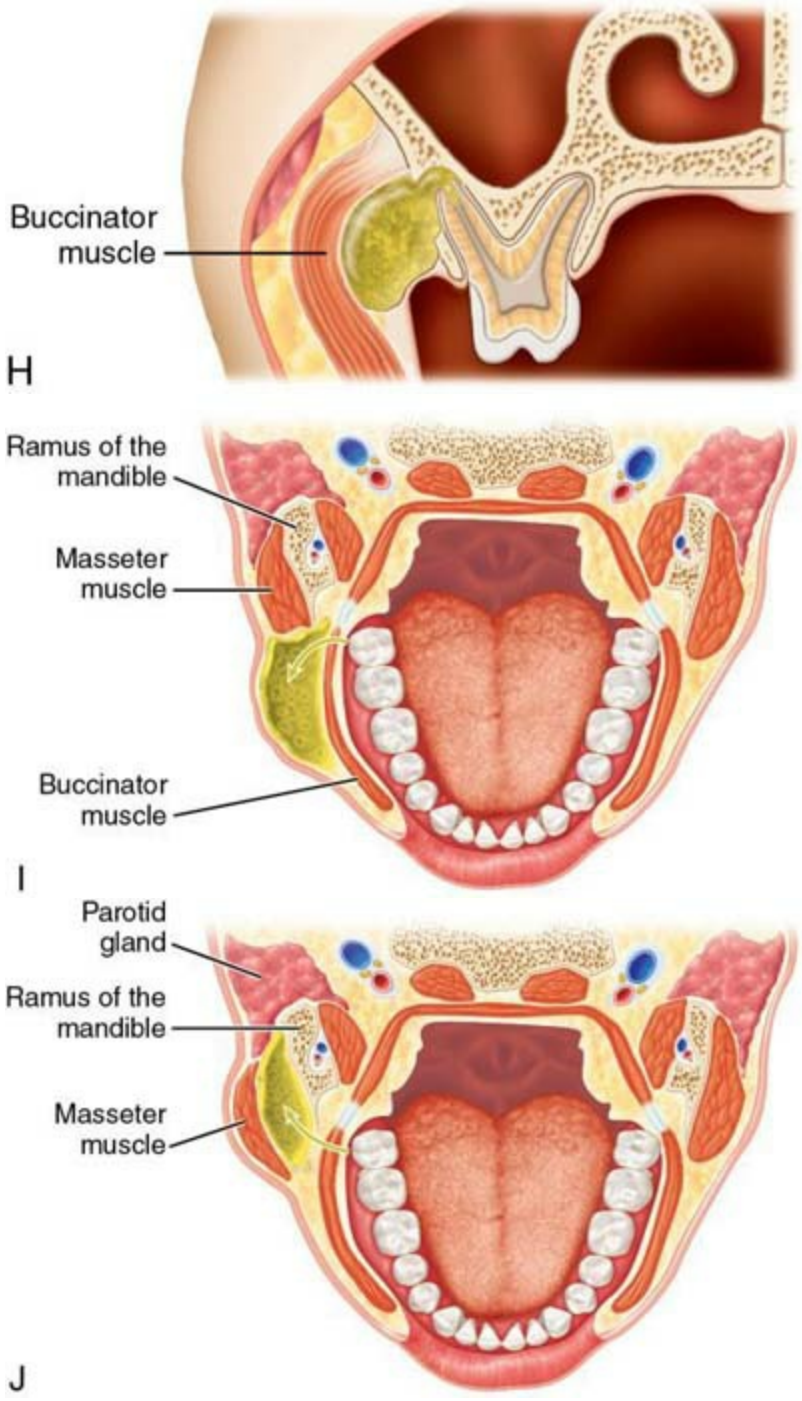

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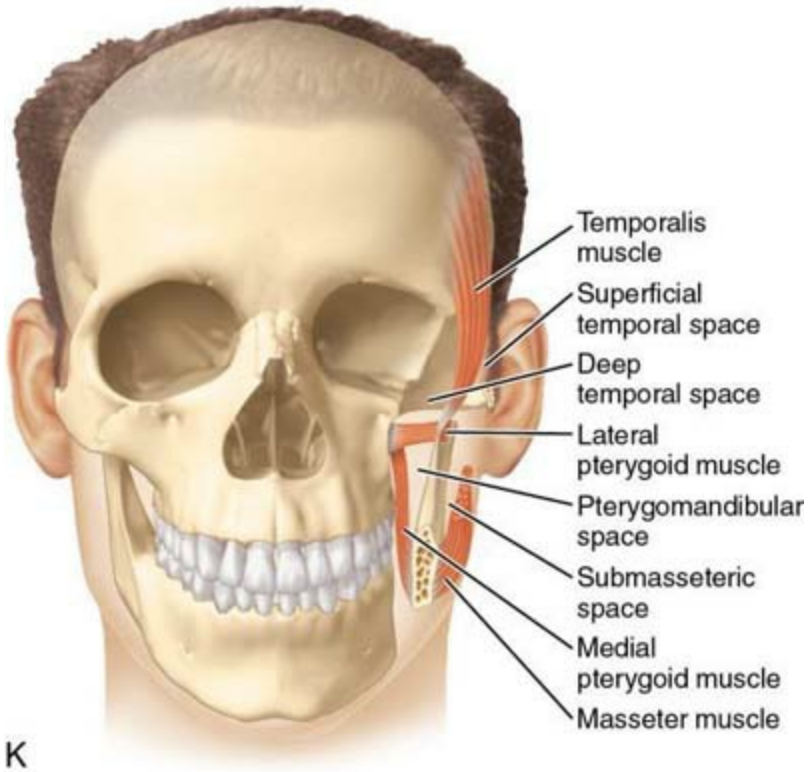

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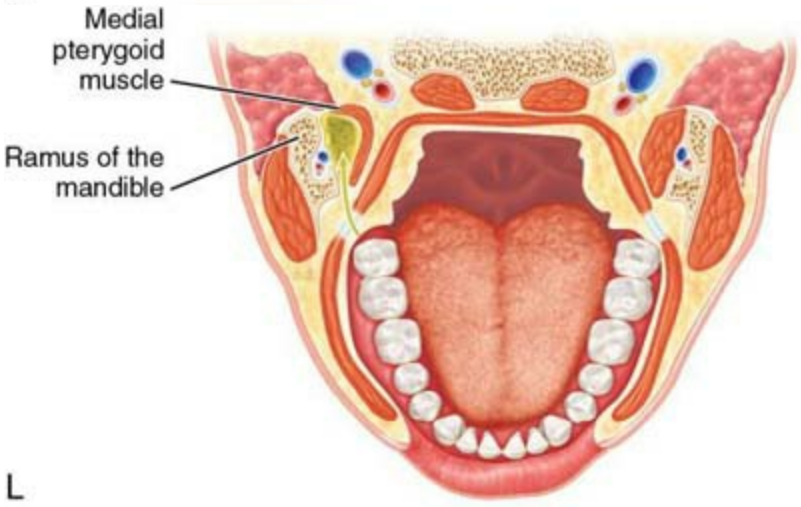

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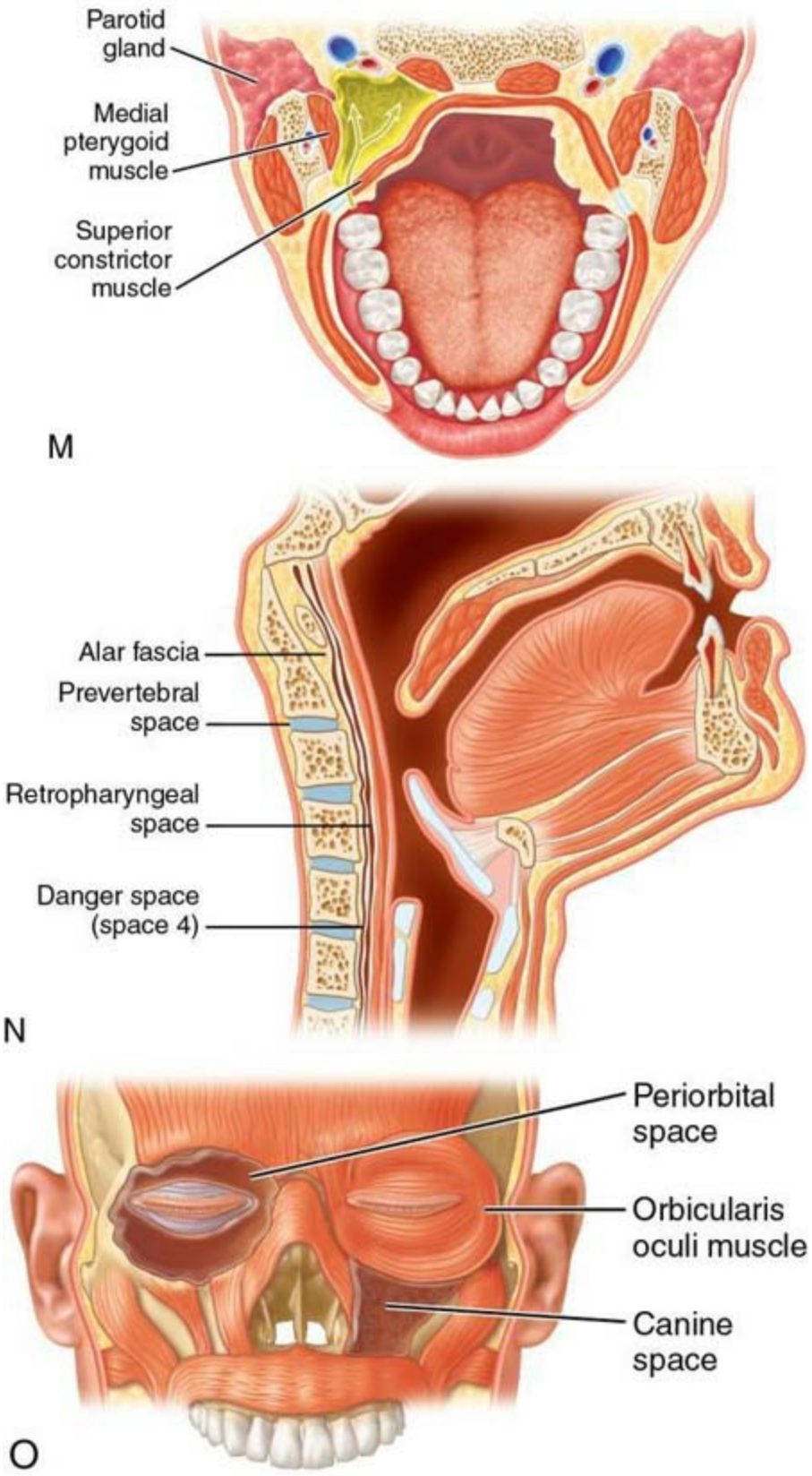

FIG. 19.4 **A,** Spread of odontogenic infections. **B,** Mandibular buccal

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vestibule (posterior tooth). **C,** Mandibular buccal vestibule (anterior tooth). **D,** Mental space. **E,** Submental space. **F,** Sublingual space. **G,** Submandibular space. **H,** Maxillary buccal vestibule. **I,** Buccal space. **J,** Submasseteric space. **K,** Temporal space. **L,** Pterygomandibular space. **M,** Parapharyngeal spaces. **N,** Cervical spaces. **O,** Canine (infraorbital) and periorbital space.

A) Open mouth with labels as follows: Buccinator muscle, mylohyoid muscle, and platysma muscle.

B) Tooth near buccinator muscle has a cloud of unusual cells on the left extending from the root canal.

C) Tooth shows the unusual growth of cells above mentalis muscle.

D) The cloud changes its shape and dislocates next to mentalis muscle.

E) The cloud further moves above platysma muscle.

F) The infectious tissue move on the right side of tooth.

G) Front view shows infectious tissue extending from the root canal above platysma muscle.

H) Enlarged view shows the infectious tissue close to buccinators muscle.

I) An arrow from the molar tooth of mandible leads to infectious tissue along buccinator muscle below masseter muscle and Ramus of the mandible.

J) The infectious tissue is present in-between masseter muscle and Ramus of the mandible below the parotid gland.

K) Front view of face with labels as follows: Temporalis muscle, superficial temporal space, deep temporal space, lateral pterygoid muscle, pterygomandibular space, submasseteric space, medial pterygoid muscle, and masseter muscle.

L) The infectious tissue from tooth moves below medial pterygoid muscle.

M) The infectious tissue lies in between superior constrictor muscle and medial pterygoid muscle.

N) Side view of neck and lower face with labels as follows: Alar fascia, prevertebral space, retropharyngeal space, and danger space (space 4).

O) Front view upper face with labels as follows: Periorbital space, orbicularis oculi muscle, and canine space.

As described by Hohl and colleagues,[53] the fascial spaces of the head and

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neck can be categorized into four anatomic groups:

- The mandible and below
- The cheek and lateral face
- The pharyngeal and cervical areas
- The midface

Swellings of and below the mandible include six anatomic areas or fascial spaces:

- The buccal vestibule
- The body of the mandible
- The mental space
- The submental space
- The sublingual space
- The submandibular space

The *mandibular buccal vestibule* is the anatomic area amid the buccal cortical plate, the overlying alveolar mucosa, and the buccinator muscle (posterior) or the mentalis muscle (anterior) (see Fig. 19.4, *B* and *C*). In this case, the source of the infection is a mandibular posterior or anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the involved tooth lie above the attachment of the buccinator or mentalis muscle, respectively.

The *space of the body of the mandible* is the potential anatomic area between the buccal or lingual cortical plate and its overlying periosteum. The source of infection is a mandibular tooth in which the purulent exudate has broken through the overlying cortical plate but not yet perforated the overlying periosteum. Involvement of this space can also occur as a result of a postsurgical infection.

The *mental space* (see Fig. 19.4, *D*) is the potential bilateral anatomic area of the chin that lies between the mentalis muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the mentalis muscle.

The *submental space* (see Fig. 19.4, *E*) is the potential anatomic area

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between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex of the tooth lies below the attachment of the mylohyoid muscle.

The *sublingual space* (see Fig. 19.4, *F*) is the potential anatomic area between the oral mucosa of the floor of the mouth superiorly and the mylohyoid muscle inferiorly. The lateral boundaries of the space are the lingual surfaces of the mandible. The source of infection is any mandibular tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex or apices of the tooth lie above the attachment of the mylohyoid muscle.

The *submandibular space* (see Fig. 19.4, *G*) is the potential space between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of infection is a posterior tooth, usually a molar, in which the purulent exudate breaks through the lingual cortical plate and the apices of the tooth lie below the attachment of the mylohyoid muscle. If the submental, sublingual, and submandibular spaces are involved at the same time, a diagnosis of Ludwig angina is made. This life-threatening cellulitis can advance into the pharyngeal and cervical spaces, resulting in airway obstruction.

Swellings of the lateral face and cheek include four anatomic areas or fascial spaces:

- The buccal vestibule of the maxilla
- The buccal space
- The submasseteric space
- The temporal space

Anatomically, the *buccal vestibular space* (see Fig. 19.4, *H*) is the area between the buccal cortical plate, the overlying mucosa, and the buccinator muscle. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic process. The source of infection is a posterior maxillary tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the buccinator muscle.

The *buccal space* (see Fig. 19.4, *I*) is the potential space between the

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lateral surface of the buccinator muscle and the medial surface of the skin of the cheek. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic arch, whereas the inferior and posterior boundaries are the attachment of the buccinator to the inferior border of the mandible and the anterior margin of the masseter muscle, respectively. The source of the infection can be either a posterior mandibular or maxillary tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the tooth lie above the attachment of the buccinator muscle (i.e., maxilla) or below the attachment of the buccinator muscle (i.e., mandible).

As the name implies, the *submasseteric space* (see Fig. 19.4, *J*) is the potential space between the lateral surface of the ramus of the mandible and the medial surface of the masseter muscle. The source of the infection is usually an impacted third molar in which the purulent exudate breaks through the lingual cortical plate, and the apices of the tooth lie very close to or within the space.

The *temporal space* (see Fig. 19.4, *K*) is divided into two compartments by the temporalis muscle. The *deep temporal space* is the potential space between the lateral surface of the skull and the medial surface of the temporalis muscle; the *superficial temporal space* lies between the temporalis muscle and its overlying fascia. The deep or superficial temporal spaces are involved indirectly if an infection spreads superiorly from the inferior pterygomandibular or submasseteric spaces, respectively.

Swellings of the pharyngeal and cervical areas include the following fascial spaces:

- The pterygomandibular space
- The parapharyngeal spaces
- The cervical spaces

The *pterygomandibular space* (see Fig. 19.4, *L*) is the potential space between the lateral surface of the medial pterygoid muscle and the medial surface of the ramus of the mandible. The superior extent of the space is the lateral pterygoid muscle. The source of the infection is mandibular second or third molars in which the purulent exudate drains directly into the space. In addition, contaminated inferior alveolar nerve injections can lead to infection of the space.

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The *parapharyngeal spaces* comprise the lateral pharyngeal and retropharyngeal spaces (see Fig. 19.4, *M*). The *lateral pharyngeal space* is bilateral and lies between the lateral surface of the medial pterygoid muscle and the posterior surface of the superior constrictor muscle. The superior and inferior margins of the space are the base of the skull and the hyoid bone, respectively, and the posterior margin is the *carotid space*, or sheath, which contains the common carotid artery, internal jugular vein, and the vagus nerve. Anatomically, the *retropharyngeal space* lies between the anterior surface of the prevertebral fascia and the posterior surface of the superior constrictor muscle and extends inferiorly into the retroesophageal space, which extends into the posterior compartment of the mediastinum. The pharyngeal spaces usually become involved as a result of the secondary spread of infection from other fascial spaces or directly from a peritonsillar abscess.

The cervical spaces comprise the pretracheal, retrovisceral, danger, and prevertebral spaces (see Fig. 19.4, *N*). The *pretracheal space* is the potential space surrounding the trachea. It extends from the thyroid cartilage inferiorly into the superior portion of the anterior compartment of the mediastinum to the level of the aortic arch. Because of its anatomic location, odontogenic infections do not spread to the pretracheal space. The *retrovisceral space* comprises the retropharyngeal space superiorly and the retroesophageal space inferiorly. The space extends from the base of the skull into the posterior compartment of the mediastinum to a level between vertebrae C6 and T4. The *danger space* (i.e., space 4)[29,38] is the potential space between the alar and prevertebral fascia. Because this space is composed of loose connective tissue, it is considered an actual anatomic space extending from the base of the skull into the posterior compartment of the mediastinum to a level corresponding to the diaphragm. It is not unknown for odontogenic infection to spread into this space, if left untreated and undiagnosed.[36] The consequence can be fatal. The *prevertebral space* is the potential space surrounding the vertebral column. As such, it extends from vertebra C1 to the coccyx. A retrospective study showed that 71% of cases in which the mediastinum was involved arose from the spread of infection from the retrovisceral space (21% from the carotid space and 8% from the pretracheal space).[70]

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Swellings of the midface consist of four anatomic areas and spaces:

- The palate
- The base of the upper lip
- The canine spaces
- The periorbital spaces

Odontogenic infections can spread into the areas between the palate and its overlying periosteum and mucosa and the base of the upper lip, which lies superior to the orbicularis oris muscle, even though these areas are not considered actual fascial spaces. The source of infection of the palate is any of the maxillary teeth in which the apex of the involved tooth lies close to the palate. The source of infection of the base of the upper lip is a maxillary central incisor in which the apex lies close to the buccal cortical plate and above the attachment of the orbicularis oris muscle.

The *canine*, or *infraorbital, space* (see Fig. 19.4, *O*) is the potential space between the levator anguli oris muscle inferiorly and the levator labii superioris muscle superiorly. The source of infection is the maxillary canine or first premolar in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies above the attachment of the levator anguli oris muscle. There is a chance for the infection to spread from the infraorbital space into the cavernous sinus in the cranium via the valveless veins of the face and anterior skull base.[29]

The *periorbital space* (see Fig. 19.4, *O*) is the potential space that lies deep to the orbicularis oculi muscle. This space becomes involved through the spread of infection from the canine or buccal spaces. Infections of the midface can be very dangerous because they can result in *cavernous sinus thrombosis*, a life-threatening infection in which a thrombus formed in the cavernous sinus breaks free, resulting in blockage of an artery or the spread of infection. Under normal conditions, the angular and ophthalmic veins and the pterygoid plexus of veins flow into the facial and external jugular veins. If an infection has spread into the midfacial area, however, edema and the resultant increased pressure from the inflammatory response cause the blood to back up into the cavernous sinus. Once in the sinus, the blood stagnates and clots. The resultant infected thrombi remain in the cavernous sinus or

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escape into the circulation.[85,122]

Management of abscesses and cellulitis

The two most essential elements of effective patient management for the resolution of an odontogenic infection are correct diagnosis and removal of the cause. When endodontic treatment is possible and preferred, in an otherwise healthy patient, chemomechanical preparation of the infected root canals and incision for drainage of any fluctuant periradicular swelling usually provide immediate improvement of the clinical signs and symptoms. The majority of cases of endodontic infections can be treated effectively without the use of systemic antibiotics. The appropriate treatment is the removal of the cause of the inflammatory condition.

Antibiotics are still prescribed in clinical situations for which they are typically not indicated.[35] The evidence clearly showed that they are *not* recommended for irreversible pulpitis, symptomatic apical periodontitis, draining sinus tracts, after endodontic surgery, to prevent flare-ups, or after incision for drainage of a localized swelling (without cellulitis, fever, or lymphadenopathy).[31,52,80,94,118] When the ratio of risk to benefit is considered in these situations, antibiotic use may put the patient at risk for side effects of the antimicrobial agent and select for resistant microorganisms. Analgesics (not antibiotics) are indicated for controlling the pain.

Antibiotics in conjunction with appropriate endodontic treatment are recommended for progressive or persistent infections with systemic signs and symptoms such as fever (over 100°F or 37°C), malaise, cellulitis, unexplained trismus, and progressive or persistent swelling (or both) (Fig. 19.5). In such cases, antibiotic therapy is indicated as an adjunct to debridement of the root canal system, which is a reservoir of microorganisms. In addition, aggressive incision for drainage is indicated for any infection marked by cellulitis. The incision for drainage is indicated whether the cellulitis is indurate or fluctuant. It is essential to provide a pathway of drainage to prevent further spread of the abscess or cellulitis. An incision for drainage allows decompression of the increased tissue pressure associated with edema and provides significant pain relief. Furthermore, the incision provides a draining pathway not only for bacteria and their products but also

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for the inflammatory mediators associated with the spread of cellulitis.

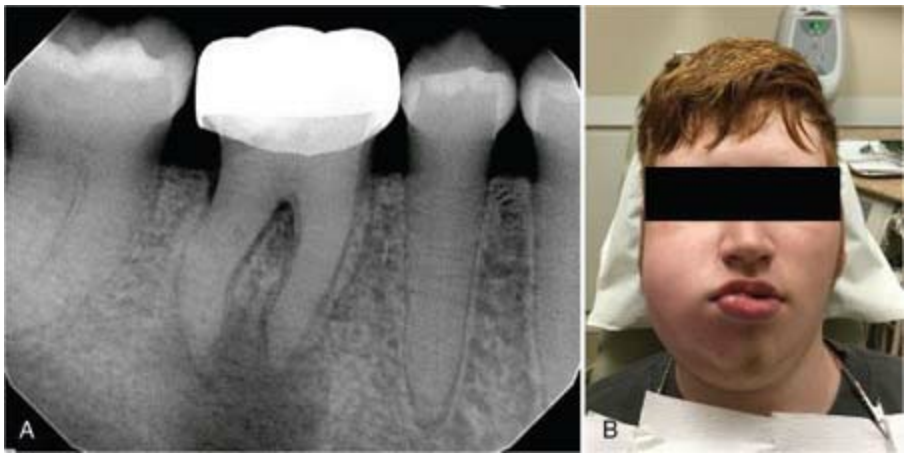

FIG. 19.5 **A,** Preoperative radiograph. **B,** Cellulitis.

A) Radiograph shows circular dark patch at the root tips of first molar. The crown is radiopaque with radiolucency near the neck.

B) Close-up view shows the front view of a patient's face with swelling on right buccal region.

A minimum inhibitory concentration of antibiotic may not reach the source of the infection because of decreased blood flow and because the antibiotic must diffuse through the edematous fluid and pus. Drainage of edematous fluid and purulent exudate improves circulation to the tissues associated with an abscess or cellulitis, providing better delivery of the antibiotic to the area. Placement of a drain may not be indicated for localized fluctuant swellings if complete evacuation of the purulent exudate is believed to have occurred.

For effective drainage, a vertically oriented stab incision is made through the mucoperiosteum in the most dependent site of the swelling. The incision must be long enough to allow blunt dissection using a curved hemostat or periosteal elevator under the periosteum for drainage of pockets of inflammatory exudate (Fig 19.6). A rubber dam drain or a Penrose drain is indicated for any patient with a progressive abscess or cellulitis to maintain an open pathway for drainage. A more detailed description is given later.

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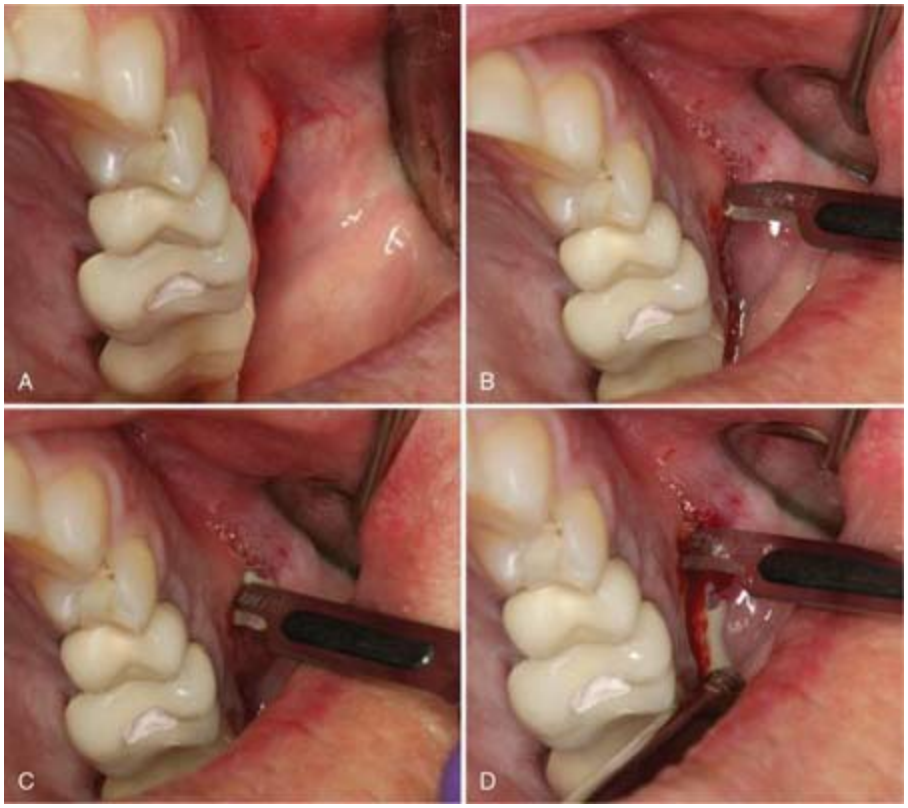

FIG. 19.6 **A,** Intraoral localized swelling. **B,** Preparation for incision. **C,** Incision. **D,** Purulence discharge.

A) Close-up view shows a teeth quadrant with inflamed mass near premolars. The first molar has temporary filling.

B) A dental instrument is placed on the inflamed mass.

C) A dental instrument is pressed into the inflamed mass.

D) Milky fluid along with blood is being released from the inflamed mass.

Patients with cellulitis should be followed daily to ensure that the infection is resolving. The best practical guide for determining the duration of antibiotic therapy is the clinical improvement of the patient. When clinical evidence indicates that the infection is certain to resolve or is resolved, antibiotics should be administered for no longer than 1 or 2 days more.

Endodontic treatment should be completed as soon as possible after the incision for drainage. The drain can usually be removed 1 or 2 days after improvement. If no significant clinical improvement occurs, the diagnosis and treatment must be reviewed carefully. Consultation with a specialist and referral may be indicated for severe or persistent infections. Likewise,

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patients requiring extraoral drainage should be referred to a clinician trained in the technique.

Incision for drainage

Although a recent study concluded that establishing drainage from a localized soft-tissue swelling may not result in the immediate relief of pain,[11] this procedure is sometimes necessary. This can be accomplished through the *incision for drainage* of the area.[81] Incision for drainage is indicated whether the cellulitis is indurated or fluctuant,[100] and it is used when a pathway for drainage is needed to prevent further spread of infection. The incision for drainage allows decompression from the increased tissue pressure associated with edema and can provide significant pain relief for the patient; noteworthy is that often pain decreases when the soft tissues swell due to the relief of pressure within the bone. The incision also provides a pathway not only for bacteria and bacterial by-products but also for the inflammatory mediators that are associated with the spread of cellulitis.

The basic principles of incision for drainage are as follows:

- Anesthetize the area.
- Make a vertically oriented incision at the site of greatest fluctuant swelling.
- Dissect gently, through the deeper tissues, and thoroughly explore all parts of the abscess cavity, eventually extending to the offending roots that are responsible for the pathosis. This will allow compartmentalized areas of inflammatory exudates and infection to be disrupted and evacuated.
- To promote drainage, the wound should be kept clean with warm saltwater mouth rinses. Intraoral heat application to infected tissues results in a dilation of small vessels, which subsequently intensifies host defenses through increased vascular flow.[42,100]
- A drain should be placed to prevent the incision from closing too early. One type of drain is ½-inch iodoform gauze, which is comfortable and less traumatic to the patient (Fig. 19.7). The patient should be seen the following day to remove the drain.

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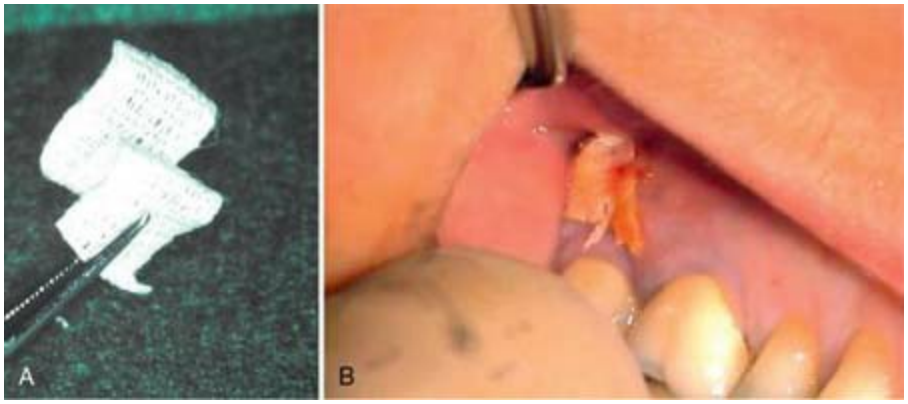

FIG. 19.7 **A,** Iodoform gauze cut to proper length. **B,** Iodoform gauze drain after 24 hours.

A) Rectangular piece of gauze having a fold is held by forceps.

B) Close-up view of the gauze absorbing the drain above the gum line of teeth.

A diffuse swelling may develop into a life-threatening medical emergency. Because the spread of infection can traverse between the fascial planes and muscle attachments, vital structures can be compromised and breathing may be impeded. Two examples are Ludwig angina and cervical fasciitis.[36] It is imperative that the clinician is in constant communication with the patient to ensure that the infection does not worsen and that medical attention is provided as necessary. Antibiotics and analgesics should be prescribed, and the patient should be monitored closely for the next several days or until there is an improvement. Individuals who show signs of toxicity, elevated body temperature, lethargy, central nervous system (CNS) changes, or airway compromise should be referred to an oral surgeon or medical facility for immediate care and intervention.

Symptomatic teeth with previous endodontic treatment

The emergency management of teeth with previous endodontic treatment may be technically challenging and time-consuming. This is especially true in the presence of extensive restorations, including posts and cores, crowns, and bridgework. However, the goal remains the same as for the management of necrotic teeth: remove contaminants from the root canal system and establish

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patency to achieve drainage. Gaining access to the periapical tissues through the root canals may require removal of posts and obturation materials, as well as negotiating blocked or ledged canals. Failure to complete root canal debridement and achieve periapical drainage may result in continued painful symptoms, in which case a trephination procedure or apicoectomy may be indicated. The ability, practicality, and feasibility to adequately retreat the root canal system must be carefully assessed before the initiation of treatment, as conventional retreatment might not be the optimal treatment plan. This is further discussed in Chapter 10.

Leaving teeth open

On rare occasions, canal drainage may continue from the periapical spaces (Fig. 19.8). In these cases, the clinician may opt to step away from the patient for some time to allow the drainage to continue and hopefully resolve on the same treatment visit.[111]

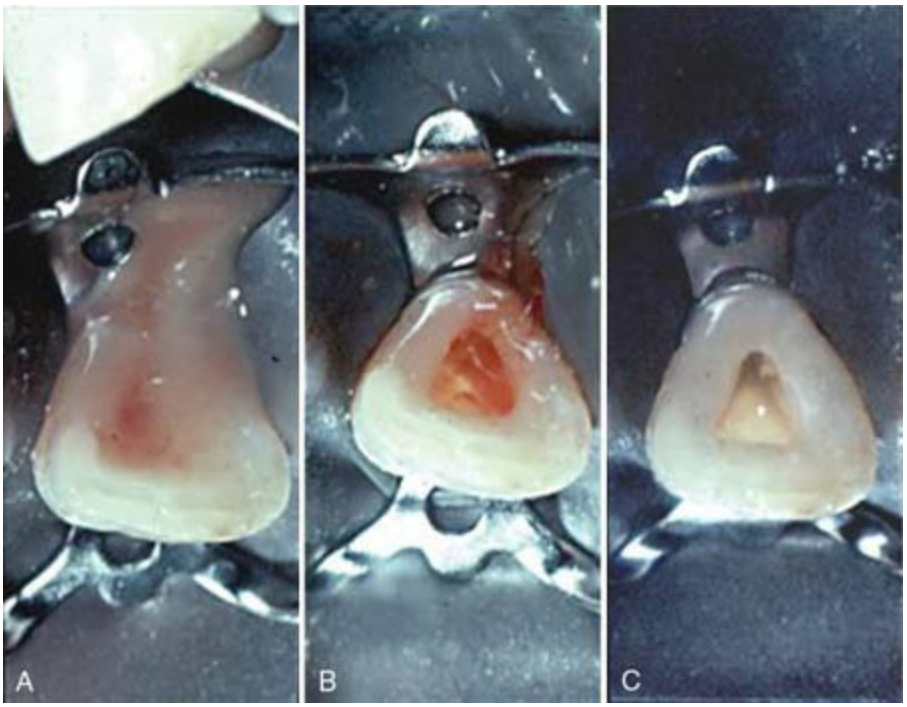

FIG. 19.8 Nonvital infected tooth with active drainage from the periapical area through the canal. **A,** Access opened and draining for 1 minute. **B,** Drainage after 2 minutes. **C,** Canal space dried after 3 minutes.

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A) Tooth isolated by rubber dam clamp shows accumulated reddish fluid below the enamel.

B) Isolated tooth shows cavity at the center filled with yellowish red fluid.

C) Isolated tooth shows cavity at the center.

Historically, in the presence of acutely painful necrotic teeth with no swelling or diffuse swelling, 19.4% to 71.2% of surveyed endodontists would leave the tooth open between visits.[25,26] However, the more current literature makes it clear that this form of treatment would impair uneventful resolution and create a more complicated procedure.[6,8,120] For this reason, *leaving teeth open between appointments is not recommended*. Foreign objects have been found in teeth left open for drainage (Fig. 19.9). There has even been a documented case report of a foreign object being found to enter the periapical tissues through a tooth that had been left open for drainage.[103] In addition, leaving a tooth open provides an opportunity for oral microorganisms to invade and colonize the root canal system if the tooth is left open for an extended period.

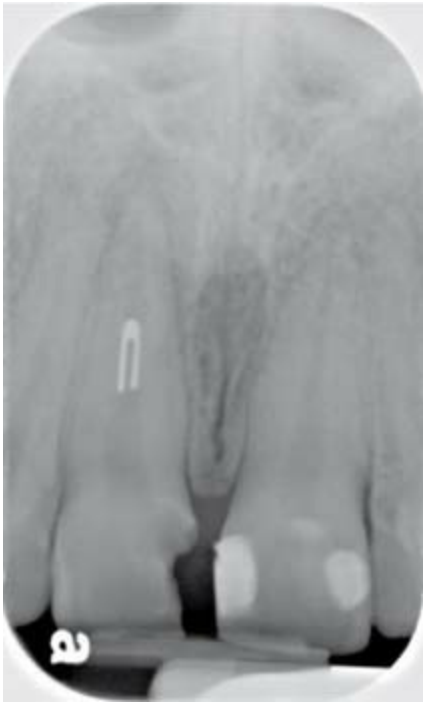

FIG. 19.9 Foreign object in tooth left open to drain. Patient used a sewing needle to clear out food particles that were blocking the canal

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and broke the needle in the tooth.

Radiograph shows inverted U shaped radiopaque line in the root canal and split end above the neck region of tooth, marked a. The adjacent tooth shows radiopaque spots on the sides of crown.

Systemic antibiotics for endodontic infections

A hundred years ago, infectious diseases were the major recognized causes of death in the world. The advent of antibiotics resulted in a significant decline in the incidence of life-threatening infections and heralded a new era in the therapy of infectious diseases, but the enthusiasm generated turned out to be premature. Over the years, microbial evolutionary responses to the selective pressure exerted by antibiotics have resulted in microbial species resistant to virtually every known antibiotic agent.[47] The rapid emergence of resistant microbial strains comes as a consequence of the astonishing power of natural selection among microorganisms. If a given member of a microbial community possesses genes of resistance against a certain antibiotic and the community is persistently exposed to the drug, the resistant microorganism is selected to emerge and multiply to the detriment of the susceptible portion of the community. Passing on the genes responsible for resistance via plasmids and quorum sensing[47] has also been shown to encourage the survival of the microbial community. The emergence of multidrug-resistant strains of several bacterial species capable of causing life-threatening infections has been reported.[47,72,90,108,121] Antibiotic resistance among obligately anaerobic bacteria is increasing, with resistance to penicillins, clindamycin, and cephalosporins noted at community hospitals and major medical centers.[48,49]

Among oral bacteria, there have been reports on emerging resistance to commonly used antibiotics. Resistance has been found in *Fusobacterium nucleatum* strains for penicillin, amoxicillin, and metronidazole, in *Prevotella intermedia* for tetracycline and amoxicillin, and in *Aggregatibacter actinomycetemcomitans* for amoxicillin and azithromycin.[64,115] Macrolides (erythromycin and azithromycin) have presented decreased activity against *Fusobacterium* and nonpigmented *Prevotella* species.[50,64,65] Production of beta-lactamase by oral bacteria has also been reported, with the most

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prominent beta-lactamase-producing bacteria belonging to the anaerobic genus *Prevotella*.[10,13,30,39,115] Kuriyama and colleagues[64] revealed that beta-lactamase production was detected in 36% of the black-pigmented *Prevotella* and 32% of the nonpigmented *Prevotella* species isolated from pus samples of oral abscesses. Susceptibility of *Prevotella* strains to several cephalosporins, erythromycin, and azithromycin has been found to correlate with amoxicillin susceptibility; amoxicillin-resistant strains can be similarly resistant to these other antibiotics.[64] This finding suggests that there is little value in the use of oral cephalosporins and macrolides in managing endodontic abscesses, mainly when penicillin-resistant strains are evident. Other enzyme-producing oral anaerobic species include strains of *F. nucleatum, Propionibacterium acnes, Actinomyces* species, and *Peptostreptococcus* species.[13,30,39,115] Facultative bacteria such as *Capnocytophaga* and *Neisseria* species have also been detected among the beta-lactamase producers.[39] Bacteria that produce beta-lactamases protect not only themselves but also other penicillin-susceptible bacteria present in a mixed community by releasing free beta-lactamase into the environment.[13]

Overuse and misuse of antibiotics have been considered the primary cause for the emergence of multidrug-resistant strains. Improper use of antibiotics includes use in cases with no infection, wrong choice of the agent, dosage or duration of therapy, and excessive consumption in prophylaxis.[88,89] Antibiotics are used in clinical practice far more often than necessary. Antibiotic therapy is warranted in about 20% of the individuals who are seen for clinical infectious disease, but they are prescribed up to 80% of the time. To complicate matters further, in up to 50% of cases, recommended agents, doses, or duration of therapy are incorrect.[69]

The appalling rise in the frequency of multidrug resistance among leading pathogens should cause great concern and incite a commitment to act carefully and responsibly. A single erroneous use of antibiotics can be a significant contribution to the current scenario of increasing microbial resistance. Diseases that were effectively treated in the past with a given antibiotic may now require the use of another drug, usually more expensive and potentially more toxic, to achieve effective antimicrobial treatment. Unfortunately, even the new prescription may not be valid.

Antibiotics are defined as naturally occurring substances of microbial

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origin or similar synthetic (or semisynthetic) elements that have antimicrobial activity in low concentrations and inhibit the growth of or kill selective microorganisms. The purpose of antibiotic therapy is to aid the host defenses in controlling and eliminating microorganisms that temporarily have overwhelmed the host defense mechanisms.[88] Based on the earlier discussion, it becomes clear that the most critical decision in antibiotic therapy is not so much *which* antibiotic should be employed but *whether* antibiotics should be used at all.[87] One should bear in mind that antibiotics are beneficial drugs classically employed to treat or help treat infectious disease and provide prophylaxis in carefully selected cases.

The majority of infections of endodontic origin are treated without the need for antibiotics. As mentioned, the absence of blood circulation in a necrotic pulp prevents antibiotics from reaching and eliminating microorganisms present in the root canal system; therefore, the source of infection is often unaffected by systemic antibiotic therapy. Antibiotics can, however, help impede the spread of the disease and development of focal infections in medically compromised patients and provide a valuable adjunct for managing selected cases of endodontic infection. In addition to the indications for systemic antibiotics discussed earlier for acute abscesses and cellulitis, antibiotics are also prescribed for prophylaxis in medically compromised patients during routine endodontic therapy, in some cases of persistent exudation not resolved after revision of intracanal procedures, and after the replantation of avulsed teeth.

Selection of antibiotics in clinical practice is either empirical or based on the results of microbial sensitivity tests. For diseases with known microbial causes, empirical therapy may be used. This is especially applicable to infections of endodontic origin because culture-dependent antimicrobial analyses of anaerobic bacteria can take too long to provide results about their susceptibility to antibiotics (7 to 14 days). Therefore, it is preferable to opt for an antimicrobial agent whose spectrum of action includes the most commonly detected bacteria. Most of the bacterial species involved with endodontic infections, including abscesses, are susceptible to penicillins,[7,57,61,65] which make them first-line drugs of choice. Because the use of antibiotics is restricted to severe infections or prophylaxis, it seems prudent to use amoxicillin, a semisynthetic penicillin with a broad spectrum

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of antimicrobial activity and one that is well absorbed in the alimentary canal. In more severe cases, including life-threatening conditions, combining amoxicillin with clavulanic acid or metronidazole may be required to achieve optimum antimicrobial effects as a result of the extended spectrum of action to include penicillin-resistant strains.[65] In patients allergic to penicillins or cases refractory to amoxicillin therapy, clindamycin is indicated. Clindamycin has potent antimicrobial activity against oral anaerobes.[61,63,65,67]

The risk/benefit ratio should always be evaluated before prescribing antibiotics. Appropriately selected patients will benefit from systemically administered antibiotics. Restrictive and conservative use of antibiotics is highly recommended in endodontic practice. Indiscriminate use (including cases of reversible or irreversible pulpitis) is contrary to sound clinical practice, as it may cause a selective overgrowth of intrinsically resistant bacteria, predisposing patients to secondary and super-infections, rendering drugs ineffective against potentially fatal medical infectious diseases.

Analgesics

As a more thorough description of pain medications can be found in Chapter 6, the following information is merely a summary of pain control using analgesics. Because pulpal and periapical pain involve inflammatory processes, the first choice of painkillers is nonsteroidal antiinflammatory drugs (NSAIDs).[69] However, no pain medication can replace the efficacy of thoroughly cleaning the root canal system to rid the tooth of the source of infection.[40]

Aspirin has been used as an analgesic for more than 100 years. In some cases, it may be more effective than 60 mg of codeine[21]; its analgesic and antipyretic effects are equal to those of acetaminophen, and its antiinflammatory effect is more potent.[24] However, aspirin's side effects include gastric distress, nausea, and gastrointestinal ulceration. In addition, its analgesic effect is inferior to that of ibuprofen, 400 mg. When NSAIDs and aspirin are contraindicated, such as in patients for whom gastrointestinal problems are a concern, acetaminophen is the preferred nonprescription analgesic. A recommended maximum daily dose of 4 g of acetaminophen is

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currently in force, and a further reduction of this dosage has been proposed to reduce the chance of acetaminophen-related liver toxicity.[68,101]

For moderate to severe pain relief, ibuprofen, an NSAID, is superior to aspirin (650 mg) and acetaminophen (600 mg) with or without codeine (60 mg). Also, ibuprofen has fewer side effects than the combinations with an opioid.[21,58] The maximal dose of 3.2 g in 24 hours should not be exceeded. Patients who take daily doses of aspirin for its cardioprotective benefit can take occasional doses of ibuprofen; however, it would be prudent to advise such patients to avoid regular doses of ibuprofen.[1] These patients would gain more relief by taking a selective cyclooxygenase (COX)-2 inhibitor, such as diclofenac or celecoxib.

Because of their antiinflammatory effect, NSAIDs can suppress swelling to a certain degree after surgical procedures. The good analgesic effect combined with the additional antiinflammatory benefit make NSAIDs, especially ibuprofen, the drug of choice for acute dental pain in the absence of any contraindication to their use. Ibuprofen has been used for more than 30 years and has been thoroughly evaluated.[24] If the NSAID alone does not have a satisfactory effect in controlling pain, then the addition of an opioid may provide additional analgesia. However, in addition to other possible side effects, opioids may cause nausea, constipation, lethargy, dizziness, and disorientation.

Laboratory diagnostic adjuncts

Chapter 15 discusses culturing techniques and indications. Because the results of culturing for anaerobic bacteria usually require at least 1 to 2 weeks, it is not considered routine in the management of an acute endodontic emergency. Thus, in an endodontic emergency, antibiotic treatment, *when indicated* (see Chapter 19), should begin immediately, because oral infections can progress rapidly.

Flare-ups

An endodontic flare-up is defined as an acute exacerbation of a periradicular pathosis after the initiation or continuation of nonsurgical root canal

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treatment.[3] The incidence may be from 2% to 20% of cases.[56,78,84,117] A meta-analysis of the literature, using strict criteria, showed the flare-up frequency to be about 8.4%.[113] Endodontic flare-ups are more prevalent among females under the age of 20 years and may occur more in maxillary lateral incisors; in mandibular first molars, when there are large periapical lesions; and in the retreatment of previous root canals.[109] The presence of pretreatment pain may also be a predictor of potential posttreatment flare-ups.[56,110,117] Fortunately, there is no decrease in the endodontic success for cases that had a treatment flare-up.[60]

Endodontic flare-ups may occur for a variety of reasons, including preparation beyond the apical terminus, over-instrumentation, pushing dentinal and pulpal debris into the periapical area,[41] incomplete removal of pulp tissue, overextension of root canal filling material, chemical irritants (such as irrigants, intracanal medicaments, and sealers), hyper occlusion, root fractures, and microbiologic factors.[102] Although many of these cases can be pharmacologically managed (see Chapter 19), refractory cases may require periapical surgery, reentry into the tooth, the establishment of drainage either through the tooth or via trephination, or, at a minimum, adjustment of the occlusion.[21,97,102] The prophylactic use of antibiotics to decrease the incidence of flare-ups has been met with some controversy. Whereas earlier investigators[79] found that antibiotic administration before treatment of necrotic teeth reduced the incidence of flare-ups, more recent studies found antibiotic use either less effective than analgesics or not to affect in reducing interappointment emergencies or posttreatment symptoms.[94,110,117]

Cracked and fractured teeth

Described in detail in Chapters 1 and 22, cracks and incomplete fractures can be challenging to locate and diagnose, but their detection can be an essential component in the management of an acute dental emergency. In the early stages, cracks are small and difficult to discern. Removal of filling materials, applications of dye solutions, selective loading of cusps, transillumination, and magnification are helpful in their detection (Fig. 19.10). As the crack or fracture becomes more extensive, it can become easier to visualize. Because cracks are difficult to find and their symptoms can be so variable, the name

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cracked tooth syndrome has been suggested,[16] even though it is not indeed a syndrome. Cracks in vital teeth often exhibit a sudden and sharp pain, especially during mastication. Cracks in nonvital or obturated teeth tend to have more of a “dull ache” but can still be sensitive to mastication.

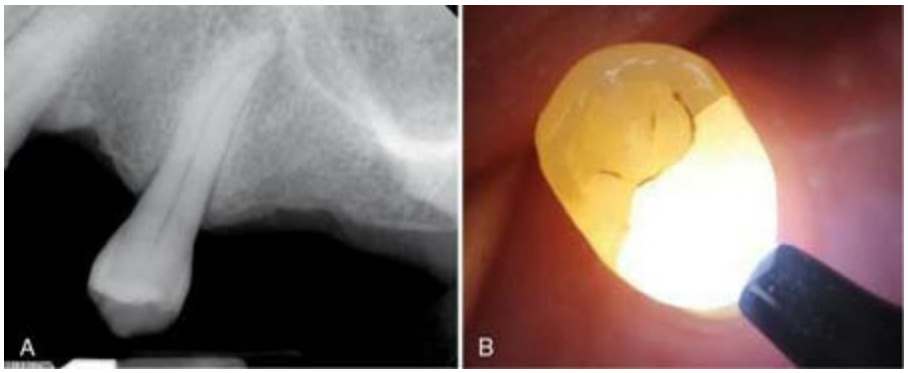

FIG. 19.10 **A,** Preoperative radiograph. **B,** Transillumination.

A) Radiograph shows posterior view of tooth with radiolucent root canal and tip.

B) Close-up view shows illuminated occlusion view of tooth.

The determination of the presence of a crack or fracture is paramount because the prognosis for the tooth may be directly dependent on the extent of the crack or the longitudinal fracture. Management of cracks in vital teeth may be as simple as a bonded restoration or a full coverage crown. However, even the best efforts to manage a crack may be unsuccessful, often requiring endodontic treatment or extraction. Fractures in nonvital or obturated teeth may be more challenging. In addition, it must be determined whether the crack or fracture was the cause of pulpal necrosis and whether there has been an extensive periodontal breakdown. If so, the prognosis for the tooth is generally poor; thus, extraction is recommended (Fig. 19.11).

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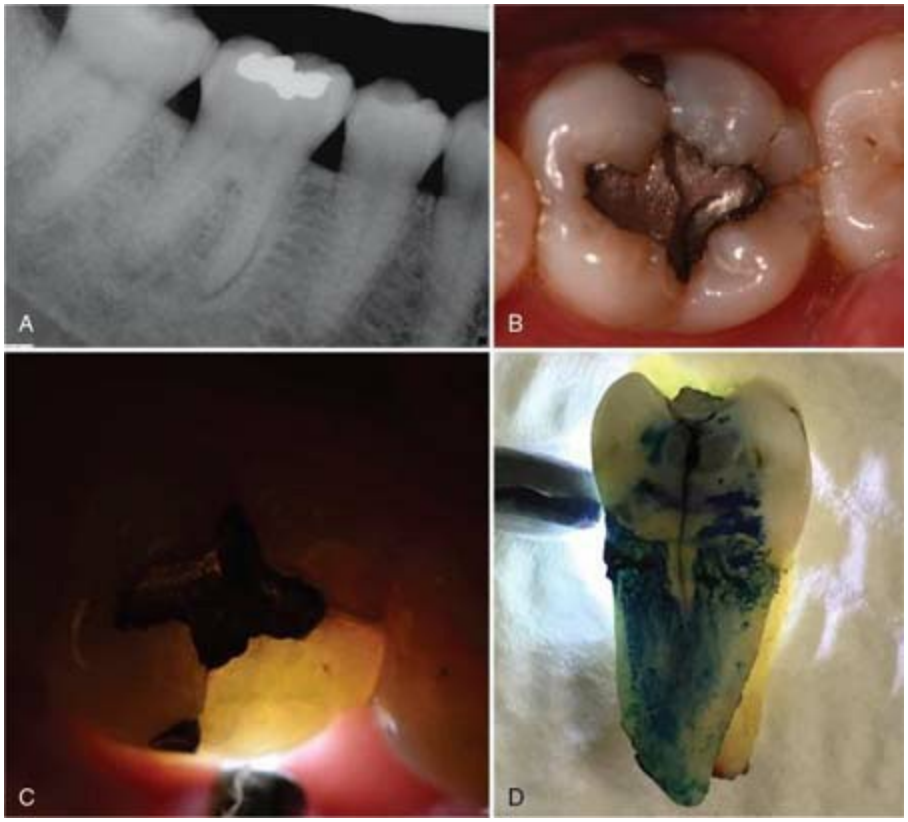

FIG. 19.11 **A,** Preoperative radiograph. **B,** Clinical view. **C,** Transillumination. **D,** Extracted tooth showing the extension of the stained longitudinal fracture.

A) Radiograph shows four teeth with less-density tissue on gum line. The first molar has white patch on the crown and the pulp is partially radiolucent.

B) Close-up view shows filled cavity of tooth that has cracks inside.

C) The cavity of tooth being examined under light shows soft tissue.

D) Extracted tooth stained in blue has fracture extending from the crown below the neck region.

Summary

The management of endodontic emergencies is an essential part of dental practice. It can often be a disruptive part of the day for the clinician and staff, but it is an invaluable solution for the distressed patient. Methodical diagnosis and prognostic assessment are imperative, with the patient being informed of the various treatment alternatives.

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References

1. Abramowicz M. *Do NSAIDs interfere with the cardioprotective effects of aspirin?*, Med Lett Drugs Ther 2004;46: 61.
2. Albahaireh ZS, Alnegrish AS. *Postobturation pain after single and multiple-visit endodontic therapy: a prospective study*, J Dent 1998;26: 227.
3. American Association of Endodontics. Glossary of endodontic terms ed 7 2003; American Association of Endodontists Chicago.
4. Ashkenaz PJ. *One-visit endodontics*, Dent Clin North Am 1984;28: 853.
5. Asgary S, Hassanizadeh R, Torabzadeb H, et al: *Treatment outcomes of 4 vital pulp therapies in mature molars*, J Endod 2018;44: 529-535.
6. Auslander WP. *The acute apical abscess*, N Y State Dent J 1970;36: 623.
7. Baumgartner JC, Xia T. *Antibiotic susceptibility of bacteria associated with endodontic abscesses*, J Endod 2003;29: 44.
8. Bence R, Meyers RD, Knoff RV. *Evaluation of 5,000 endodontic treatment incidents of the open tooth*, Oral Surg Oral Med Oral Pathol 1980;49: 82.
9. Bender IB. *Pulpal pain diagnosis: a review*, J Endod 2000;26: 175.
10. Bernal LA, Guillot E, Paquet C, et al: *Beta-lactamase producing strains in the species Prevotella intermedia and Prevotella nigrescens*, Oral Microbiol Immunol 1998;13: 36.
11. Beus H, Fowler S, Drum M, et al: *What is the outcome of*

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www.konkur.in

an incision and drainage procedure in endodontic patients? a prospective, randomized, single-blind study, J Endod 2018;44: 193-201.

12. Boutsioukis C, Psimma Z, Kastrinakis E. *The effect of flow rate and agitation technique on irrigant extrusion ex vivo*, Int Endod J 2014;47: 487.
13. Brook I. *beta-Lactamase-producing bacteria in mixed infections*, Clin Microbiol Infect 2004;10: 777.
14. Deleted in page review.
15. Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals*, Endod Dent Traumatol 1985;1: 170.
16. Cameron CE. *The cracked tooth syndrome*, J Am Dent Assoc 1976;93: 971.
17. Carrotte P. Endodontics. *part 3. Treatment of endodontic emergencies*, Br Dent J 2004;197: 299.
18. Charara K, Friedman S, Sherman A, et al: *Assessement of apical extrusion during root canal irrigation with the novel GentleWave system in a simulated apical environment*, J Endod 2016;42: 135.
19. Chestner SB, Selman AJ, Friedman J, et al: *Apical fenestration: solution to recalcitrant pain in root canal therapy*, J Am Dent Assoc 1986;77: 846.
20. Chong BS, Pitt Ford TR. *The role of intracanal medication in root canal treatment*, Int Endod J 1992;25: 97.
21. Cooper SA, Beaver WT. *A model to evaluate mild analgesics in oral surgery outpatients*, Clin Pharmacol Ther 1976;20: 241.
22. Creech JH, Walton RE, Kaltenbach R. *Effect of occlusal*

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www.konkur.in

relief on endodontic pain, J Am Dent Assoc 1984;109: 64.
23. Desi P, Himel V. *Comparative safety of various intracanal irrigation systems*, J Endod 2009;35: 545.
24. Dionne RA, Phero JC, Becker DE. Management of pain and anxiety in the dental office 2002; Saunders Philadelphia.
25. Dorn SO, Moodnik RM, Feldman MJ, et al: *Treatment of the endodontic emergency: a report based on a questionnaire—part I*, J Endod 1977;3: 94.
26. Dorn SO, Moodnik RM, Feldman MJ, et al: *Treatment of the endodontic emergency: a report based on a questionnaire—part II*, J Endod 1977;3: 153.
27. Eleazer PD, Eleazer KR. *Flare-up rate in pulpally necrotic molars in one-visit versus two-visit endodontic treatment*, J Endod 1998;24: 614.
28. Field JW, Gutmann JL, Solomon ES, et al: *A clinical radiographic retrospective assessment of the success rate of single-visit root canal treatment*, Int Endod J 2004;37: 70.
29. Flynn TR. *Anatomy of oral and maxillofacial infections*, Topazian RG Goldberg MH Hupp JR Oral and maxillofacial infections ed 4 2002; WB Saunders Philadelphia 188-213.
30. Fosse T, Madinier I, Hitzig C, et al: *Prevalence of betalactamase-producing strains among 149 anaerobic gram negative rods isolated from periodontal pockets*, Oral Microbiol Immunol 1999;14: 352.
31. Fouad AF, Rivera EM, Walton RE. *Penicillin as a supplement in resolving the localized acute apical abscess*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81: 590.
32. Galani M, Tewari S, Sangwan P, et al: *Comparative*

Telegram: @dental_k

www.konkur.in

evaluation of postoperative pain and success rate after pulpotomy and root canal treatment in cariously exposed mature permanent molars - A randomized controlled trial, J Endod 2017;43: 1953.

33. Gatchel RJ. *Managing anxiety and pain during dental treatment*, J Am Dent Assoc 1992;123: 37.
34. Gatewood RS, Himel VT, Dorn S. *Treatment of the endodontic emergency: a decade later*, J Endod 1990;16: 284.
35. Germack M, Sedgley CM, Sabbah W, et al: *Antibiotic use in 2016 by members of the American Association of Endodontists: report of a national survey*, J Endod 2017;43: 1615-1622.
36. Goldberg MH, Topazian RG. *Odontogenic infections and deep fascial space infections of dental origin*, Topazian RG Goldberg MH Hupp JR Oral and maxillofacial infections ed 4 2002; WB Saunders Philadelphia 158-187.
37. Deleted in page review.
38. Grodinsky M, Holyoke EA. *The fasciae and fascial spaces of the head, neck, and adjacent regions*, Am J Anat 1938;63: 367.
39. Handal T, Olsen I, Walker CB, et al: *Beta-lactamase production and antimicrobial susceptibility of subgingival bacteria from refractory periodontitis*, Oral Microbiol Immunol 2004;19: 303.
40. Hargreaves KM, Keiser K. *New advances in the management of endodontic pain emergencies*, J Calif Dent Assoc 2004;32: 469.
41. Harrington GW, Natkin E. *Midtreatment flare-ups*, Dent Clin North Am 1992;36: 409.
42. Harrison JW. *Irrigation of the root canal system*, Dent Clin North Am 1984;28: 797.

Telegram: @dental_k

www.konkur.in

43. Hasler JF, Mitchel DF. *Analysis of 1628 cases of odontalgia: a corroborative study*, J Indianap Dist Dent Soc 1963;17: 23.
44. Hasselgren G. *Pains of dental origin*, Dent Clin North Am 2000;12: 263.
45. Hasselgren G, Reit C. *Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings*, J Endod 1989;15: 254.
46. Haug SR, Solfjeld AF, Ranheim LE, et al: *Impact of case difficulty on endodontic mishaps in an undergraduate student clinic*, J Endod 2018;44: 1088.
47. Hayward CMM, Griffin GE. *Antibiotic resistance: the current position and the molecular mechanisms involved*, Br J Hosp Med 1994;52: 473.
48. Hecht DW. *Prevalence of antibiotic resistance in anaerobic bacteria: worrisome developments*, Clin Infect Dis 2004;39: 92.
49. Hecht DW, Vedantam G, Osmolski JR. *Antibiotic resistance among anaerobes: what does it mean?*, Anaerobe 1999;5: 421.
50. Heimdahl A, von Konow L, Satoh T, et al: *Clinical appearance of orofacial infections of odontogenic origin in relation to microbiological findings*, J Clin Microbiol 1985;22: 299.
51. Henry BM, Fraser JG. *Trephination for acute pain management*, J Endod 2003;29: 144.
52. Henry M, Reader A, Beck M. *Effect of penicillin on postoperative endodontic pain and swelling in symptomatic necrotic teeth*, J Endod 2001;27: 117.
53. Hohl TH, Whitacre RJ, Hooley JR, et al: A self instructional guide: diagnosis and treatment of odontogenic infections 1983; Stoma Press Seattle.

Telegram: @dental_k

www.konkur.in

54. Holmes-Johnson E, Geboy M, Getka EJ. *Behavior considerations*, Dent Clin North Am 1986;30: 391.
55. Horrobin DF, Durnad LG, Manku MS. *Prostaglandin E 1 modifies nerve conduction and interferes with local anesthetic action*, Prostaglandins 1997;14: 103.
56. Imura N, Zuolo ML. *Factors associated with endodontic flareups: a prospective study*, Int Endod J 1995;28: 261.
57. Jacinto RC, Gomes BP, Ferraz CC, et al: *Microbiological analysis of infected root canals from symptomatic and asymptomatic teeth with periapical periodontitis and the antimicrobial susceptibility of some isolated anaerobic bacteria*, Oral Microbiol Immunol 2003;18: 285.
58. Jain AK, Ryan JR, McMahon G. *Analgesic efficacy of low-dose ibuprofen in dental extraction*, Pharmacotherapy 1986;6: 318.
59. Keenan JV, Farman AG, Fedorowica Z, et al: *A cochrane systematic review finds no evidence to support the use of antibiotics for pain relief in irreversible pulpitis*, J Endod 2006;32: 87.
60. Kerekes K, Tronstad L. *Long-term results of endodontic treatment performed with a standardized technique*, J Endod 1979;5: 83.
61. Khemaleelakul S, Baumgartner JC, Pruksakorn S. *Identification of bacteria in acute endodontic infections and their antimicrobial susceptibility*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94: 746.
62. Kim E, Lee SJ. *Electronic apex locator [Review]*, Dent Clin North Am 2004;48: 35.
63. Kuriyama T, Karasawa T, Nakagawa K, et al: *Bacteriologic features and antimicrobial susceptibility in isolates from orofacial odontogenic infections*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 600.

Telegram: @dental_k

www.konkur.in

64. Kuriyama T, Karasawa T, Nakagawa K, et al: *Incidence of beta-lactamase production and antimicrobial susceptibility of anaerobic gram-negative rods isolated from pus specimens of orofacial odontogenic infections*, Oral Microbiol Immunol 2001;16: 10.
65. Kuriyama T, Williams DW, Yanagisawa M, et al: *Antimicrobial susceptibility of 800 anaerobic isolates from patients with dentoalveolar infection to 13 oral antibiotics*, Oral Microbiol Immunol 2007;22: 285.
66. Kvist T, Molander A, Dahlen G, et al: *Microbiological evaluation of one- and two-visit endodontic treatment of teeth with apical periodontitis: a randomized, clinical trial*, J Endod 2004;30: 572.
67. Lakhassassi N, Elhajoui N, Lodter JP, et al: *Antimicrobial susceptibility variation of 50 anaerobic periopathogens in aggressive periodontitis: an interindividual variability study*, Oral Microbiol Immunol 2005;20: 244.
68. Larson AM, Polson J, Fontana RJ, et al: *Acetaminophen-induced acute liver failure: results of a United States multicenter, prospective study*, Hepatology 2005;42: 1364.
69. Lee M, Winkler J, Hartwell G, et al: *Current trends in endodontic practice: emergency treatments and technological armamentarium*, J Endod 2009;35: 35.
70. Levitt GW. *The surgical treatment of deep neck infections*, Laryngoscope 1970;81: 403.
71. Lipton JA, Ship JA, Larach-Robinson D. *Estimated prevalence and distribution of reported orofacial pain in the United States*, J Am Dent Assoc 1993;124: 115.
72. Madigan MT, Martinko JM, Parker J. Brock biology of microorganisms ed 9 2000; Prentice-Hall Upper Saddle River, NJ.
73. Matusow RJ, Goodall LB. *Anaerobic isolates in primary*

Telegram: @dental_k

www.konkur.in

pulpal–alveolar cellulitis cases: endodontic resolutions and drug therapy considerations, J Endod 1983;9: 535.

74. Metka ME, Liem VML, Parsa A, et al: *Cone-beam computed tomographic scans in comparison with periapical radiographs for root canal length measurement: an in situ study*, J Endod 2014;40: 1206.
75. Mitchell RP, Yang S, Baumgartner JC. *Comparison of apical extrusion of NaOCl using the EndoVac or needle irrigation of root canals*, J Endod 2010;36: 338.
76. Deleted in page review.
77. Moos HL, Bramwell JD, Roahen JO. *A comparison of pulpectomy alone versus pulpectomy with trephination for the relief of pain*, J Endod 1996;22: 422.
78. Morse DR, Koren LZ, Esposito JV, et al: *Asymptomatic teeth with necrotic pulps and associated periapical radiolucencies: relationship of flare-ups to endodontic instrumentation, antibiotic usage and stress in three separate practices at three different time periods*, Int J Psychosom 1986;33: 5.
79. Morse DR, Furst ML, Belott RM, et al: *Infectious flare-ups and serious sequelae following endodontic treatment: a prospective randomized trial on efficacy of antibiotic prophylaxis in cases of asymptomatic pulpal–periapical lesion*, Oral Surg 1987;64: 96.
80. Nagle D, Reader A, Beck M, et al: *Effect of systemic penicillin on pain in untreated irreversible pulpitis*, Oral Surg Oral Med Oral Pathol 2000;90: 636.
81. Natkin E. *Treatment of endodontic emergencies*, Dent Clin North Am 1974;18: 243.
82. Nusstein J, Reader A, Nist R, et al: *Anesthetic efficacy of the supplemental intraosseous injection*, J Endod 1998;24: 487.

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www.konkur.in

83. Nyerere JW, Matee MI, Simon EN. *Emergency pulpotomy in relieving acute dental pain among Tanzanian patients,* BMC Oral Health 2006;6: 1.
84. Oginni AO, Udoye CI. *Endodontic flare-ups: comparison of incidence between single and multiple visit procedures in patients attending a Nigerian teaching hospital*, BMC Oral Health 2004;4: 4.
85. Ogundiya DA, Keith DA, Mirowski J. *Cavernous sinus thrombosis and blindness as complications of an odontogenic infection*, Oral Maxillofac Surg 1989;47: 1317.
86. Oliet S. *Single-visit endodontics: a clinical study*, J Endod 1998;24: 614.
87. Pallasch TJ. *Antibiotics in endodontics*, Dent Clin North Am 1979;23: 737.
88. Pallasch TJ. *Pharmacokinetic principles of antimicrobial therapy*, Periodontol 2000 1996;10: 5.
89. Pallasch TJ, Slots J. *Antibiotic prophylaxis and the medically compromised patient*, Periodontol 2000 1996;10: 107.
90. Patel R. *Clinical impact of vancomycin-resistant enterococci*, J Antimicrob Chemother Suppl 3, 2003;51: iii13.
91. Pekruhn RB. *The incidence of failure following single-visit endodontic therapy*, J Endod 1986;12: 68.
92. Penesis VA, Fitzgerald PI, Fayad MI, et al: *Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: a randomized controlled trial with one-year evaluation*, J Endod 2008;34: 251.
93. Peters LB, Wesselink PR. *Periapical healing of endodontically treated teeth in one and two visits obturated in the presence or absence of detectable*

Telegram: @dental_k

www.konkur.in

microorganisms, Int Endod J 2002;35: 660.

94. Pickenpaugh L, Reader A, Beck M, et al: *Effect of prophylactic amoxicillin on endodontic flare-up in asymptomatic, necrotic teeth*, J Endod 2001;27: 53.
95. Reddy SA, Hicks ML. *Apical extrusion of debris using two hand and two rotary instrumentation techniques*, J Endod 1998;24: 180.
96. Roane JB, Dryden JA, Grimes EW. *Incidence of postoperative pain after single- and multiple-visit endodontic procedures*, Oral Surg Oral Med Oral Pathol 1983;55: 68.
97. Rosenberg PA, Babick PJ, Schertzer L, et al: *The effect of occlusal reduction on pain after endodontic instrumentation*, J Endod 1998;24: 492.
98. Rudner WL, Oliet S. *Single-visit endodontics: a concept and a clinical study*, Compend Contin Educ Dent 1981;2: 63.
99. Rugh JD. *Psychological components of pain*, Dent Clin North Am 1987;31: 579.
100. Sandor GK, Low DE, Judd PL, et al: *Antimicrobial treatment options in the management of odontogenic infections*, J Can Dent Assoc 1998;64: 508- Comment in *J Can Dent Assoc* 65:602, 1999.
101. Schilling A, Corey R, Leonard M, et al: *Acetaminophen: old drug, new warnings*, Cleve Clin J Med 2010;77: 19- doi: 10.3949/ccjm.77a.09084.
102. Seltzer S, Naidorf IJ. *Flare-ups in endodontics. 1. Etiological factors*, J Endod 1985;11: 472.
103. Simon JH, Chimenti RA, Mintz GA. *Clinical significance of the pulse granuloma*, J Endod 1982;8: 116.
104. Siqueira JF, Rocas IN. *Microbial causes of endodontic flareups*, Int Endod J 2003;36: 433.

Telegram: @dental_k

www.konkur.in

105. Sjogren U, Figdor D, Persson S, et al: *Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis*, Int Endod J 1997;30: 297.
106. Southard DW, Rooney TP. *Effective one-visit therapy for the acute periapical abscess*, J Endod 1984;10: 580.
107. Sutherland S, Matthews DC. *Emergency management of acute apical periodontitis in the permanent dentition: a systematic review of the literature*, J Can Dent Assoc 2003;69: 160.
108. Tendolkar PM, Baghdayan AS, Shankar N. *Pathogenic enterococci: new developments in the 21st century*, Cell Mol Life Sci 2003;60: 2622.
109. Torabinejad M, Dorn SO, Eleazer PD, et al: *The effectiveness of various medications on postoperative pain following root canal obturation*, J Endod 1994;20: 427.
110. Torabinejad M, Kettering JD, McGraw JC, et al: *Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps*, J Endod 1988;14: 261.
111. Torabinejad M, Walton R. Endodontics: principles and practice ed 4 2009; Saunders St. Louis.
112. Trope ME, Delano EO, Orstavik D. *Endodontic treatment of teeth with apical periodontitis: single vs. multivisit treatment*, J Endod 1999;25: 345.
113. Tsesis I, Faivishevsky V, Fuss Z, et al: *Flare-ups after endodontic treatment: a meta-analysis of literature*, J Endod 2008;34: 1177.
114. Turkun M, Cengiz T. *The effects of sodium hypochlorite and calcium hydroxide in tissue dissolution and root canal cleanliness*, Int Endod J 1997;30: 335.
115. van Winkelhoff AJ, Winkel EG, Barendregt D, et al: *beta-Lactamase producing bacteria in adult periodontitis*,

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J Clin Periodontol 1997;24: 538.

116. Vera J, Ochoa J, Romero M, et al: *Intracanal cryotherapy reduces postoperative pain in teeth with symptomatic apical periodontitis: A randomized multicenter clinical trial*, J Endod 2018;44: 4.
117. Walton R, Fouad A. *Endodontic interappointment flare-ups: a prospective study of incidence and related factors*, J Endod 1992;18: 172.
118. Weiger R, Axmann-Krcmar D, Löst C. *Prognosis of conventional root canal treatment reconsidered*, Endod Dent Traumatol 1998;14: 1.
119. Weiger R, Rosendahl R, Lost C. *Influence of calcium hydroxide intracanal dressings on the prognosis of teeth with endodontically induced periapical lesions*, Int Endod J 2000;33: 219.
120. Weine FS, Healey HJ, Theiss EP. *Endodontic emergency dilemma: leave tooth open or keep it closed?*, Oral Surg Oral Med Oral Pathol 1975;40: 531.
121. Whitney CG, Farley MM, Hadler J, et al: *Increasing prevalence of multidrug-resistant Streptococcus pneumoniae in the United States*, N Engl J Med 2000;343: 1917.
122. Yun MW, Hwang CF, Lui CC. *Cavernous sinus thrombus following odontogenic and cervicofacial infection*, Eur Arch Otorhinolaryngol 1991;248: 422.

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*The authors acknowledge the outstanding work of Drs. Samuel O. Dorn, J. Craig Baumgartner, Jeffrey W. Hutter, and Louis Berman in previous editions of this text.

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20: Managing iatrogenic events

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CHAPTER OUTLINE

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An iatrogenic event is defined as a procedure “induced inadvertently by a

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physician or surgeon" (Merriam-Webster dictionary). This is true for dentistry as well, with cases of endodontic malpractice being among the most prevalent in dentistry.[70,132,293] These occurrences are not limited to the United States, with other countries reporting similar percentages of endodontic malpractice claims.[257]

Although iatrogenic accidents cannot be prevented with absolute certainty, their occurrences can be greatly reduced with the aid of the "Three Ts": training, technique, and technology. Incumbent with the first (training) is expertise with the other two factors, especially regarding technology and, more specifically, the dental operating microscope (DOM) together with cone-beam computed tomography (CBCT).[12,182,272] They have proven themselves indispensable in treatment planning based on the diagnosis,[272] locating canals including complex anatomical spaces and periapical pathosis,[12,160,180,183,197,291,322,347] removing separated instruments,[324,346] repairing perforations,[268] preventing iatrogenic accidents,[182,364] and enhancing the delivery of quality care.[174] The superior visualization with the aid of CBCT imaging permits more accurate clinical evaluations including the identification of the canal root morphology that cannot be visualized with the DOM,[48] and allowing for a precise assessment of the challenge and the appropriate treatment plan for its resolution. Illumination and magnification along with the 3D imaging are indispensable for all of the treatment scenarios that follow, which are therefore assumed to be performed with the DOM and CBCT imaging, which has proven as accurate as direct visualization.[13]

Sodium hypochlorite

Often happening without warning, the progression of events following sodium hypochlorite (NaOCl) extrusion into periradicular tissues is both rapid and startling to witness. In addition to the physical outcomes, the experience is likely to leave the patient wary of both the procedure and practitioner.

Although the properties and methods of delivering NaOCl are covered in Chapter 8, the gravity of the consequences of its misuse merits a brief highlight. NaOCl is an effective irrigating solution in concentrations ranging from 0.5% to 6%[149,373] and is able to disrupt the biofilm,[58] dissolve necrotic

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tissues,[211] and remove organic components of the smear layer.[20] However, it is extremely cytotoxic, irrespective of the tissue it contacts,[231] with concentrations as low as 0.01% lethal to fibroblasts in vitro.[138] NaOCl injection into vital tissue initiates hemolysis and ulceration, damages endothelial and fibroblast cells, and inhibits neutrophil migration.[107,109] A study investigating the physical effects of NaOCl on bone ex vivo found degradation of the organic matrix collagen and no sign of living cellular contents (Fig. 20.1).[163]

FIG. 20.1 Femur shaft cross sections (**A**) untreated bone section; (**B**) saline-treated bone; (**C**) sodium hypochlorite (NaOCl)-treated bone section. Grossly, NaOCl caused significant changes in cancellous structure, leaving large structural craters of apparent demineralization.

Three cross-sections of femur shaft are marked A through C.

A) It has wet cavity with few holes and stretched connective tissue along the periphery.

B) The cavity has dried perforated layer.

C) The cavity shows the complete degradation of collagen cells with large number of irregular chambers.

Source: (From Kerbl FM, DeVilliers P, Litaker M, Eleazer PD: Physical effects of NaOCl on bone: an ex vivo study. *J Endod* 38:357, 2012.)

The majority of reported NaOCl complications appear to be due to an inaccurate working length, iatrogenic widening of the radicular foramen, lateral perforation of the root, or wedging of the irrigation needle.[171,316] The pattern of facial distention is comparable to that encountered with a cervicofacial emphysema (discussed later), dissecting fascial spaces in a similar fashion, with the concomitant tissue destruction magnifying the effect and impeding recovery. This swelling is further exacerbated by the presence

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of effervescing agents such as hydrogen peroxide, potentiating the effect of the hypochlorite and amplifying soft-tissue involvement.[23,186] The initial patient reaction to the fluid insult is severe and immediate pain, with marked edema or bruising that may continue to extend over the injured side of the face, cheek, or lips (Fig. 20.2). There may be spontaneous and profuse hemorrhaging from the canal space, and, in the case of maxillary posterior teeth, the patient may report periorbital pain,[24,107] chlorine taste, or irritation of the throat.[80] The amount that has been expressed into the periradicular area, the spatial location of the fluid introduction, and the proximity to sensitive anatomic structures will dictate the severity and duration of the reaction. Paresthesia, either transient[24] or permanent,[255] can occur, especially if interventional treatment is not rendered immediately. In addition to sensory deprivation, motor nerve dysfunction secondary to irreversible chemical damage has been reported (Fig. 20.3).[236] In this case, there was distinct loss of upper lip and cheek function, with problems in the musculature. In mandibular teeth, extension into the submandibular, submental, or sublingual regions can compromise the airway, compelling immediate hospitalization and surgical intervention to avert a life-threatening episode.[35]

FIG. 20.2 **A,** Clinical presentation 3 days following the extrusion of sodium hypochlorite (NaOCl) during treatment of the maxillary right canine. The patient had difficulty opening her right eye, and the swelling had extended to the submandibular/sublingual regions on the

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affected side; she also experienced limited mouth opening. There was altered sensation infraorbitally and in the region of the upper right lip, but no reported paresthesia of the alveolar or facial nerves. **B,** One-month postextrusion, the ecchymosis had resolved, with a return of sensation and full function. Therapy was completed without incident.

A) Close-up shows the lateral view patient's face and neck with suffering from extensive bruising extending below the neck to mouth and buccal region along with right eye depicted as purple-blue patches on skin.

B) Close-up view shows the front view of face and neck with disappearance of bruises.

Source: (From de Sermeño RF, da Silva LA, Herrera H: Tissue damage after NaOCl extrusion during root canal treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 108:e46, 2009.)

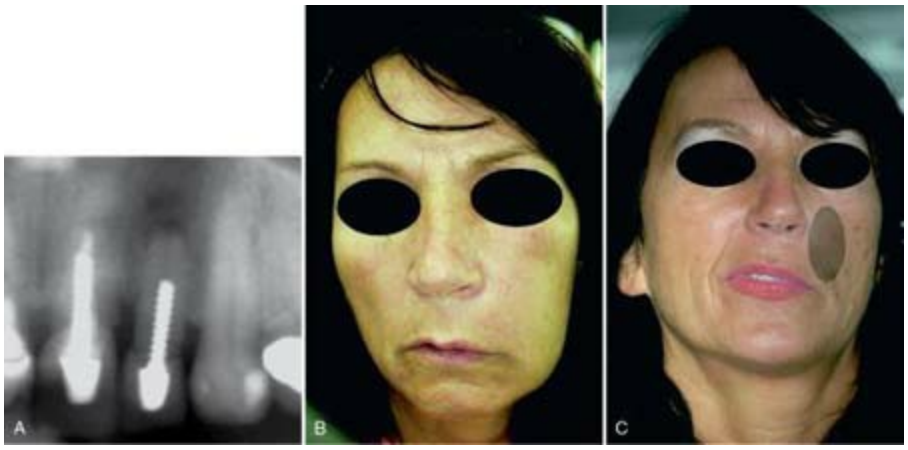

FIG. 20.3 **A,** Radiograph of the maxillary left lateral incisor (#10, #22) before post removal. Radicular root resection had been completed, and the canal space is empty. Prior to recementation, the canal was irrigated with 3% NaOCl, precipitating the extrusion event. **B,** Twenty-four hours postextrusion, there was altered sensation infraorbitally and from the upper left lip to the left lip corner. In addition, the buccal branch of the facial nerve was affected, as evidenced by a distinct loss of upper lip and cheek function (the corner of the mouth could not be pulled up by the mimic musculature). The mouth opening was limited to 20 mm (**C**), 3 years postincident. The weakness of the mimic musculature of the left face side is clearly visible. Attempts to laugh resulted in a hanging left lip corner secondary to the weakness of the motor innervation by the facial nerve. In the gray-marked area there is a permanent hypoesthesia.

A) Radiograph shows three teeth with the second tooth having a squarish

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radiolucent patch at the root apex. Crown and three-fourth part of the canal are radiopaque.

B) Close-up of face of a patient shows the front view of patient's face with extremely thin upper lip and inflammation in the lower left of chin.

C) Close-up of face of a patient shows the front view of face with weak muscles around the mouth.

Source: (From Pelka M, Petschelt A: Permanent mimic musculature and nerve damage caused by sodium hypochlorite: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 106:e80, 2008.)

Upon realizing that an extrusion event has occurred, it is imperative that the clinician cease further treatment and immediately engage in measures targeted at diminishing the effect of the chemical; ignoring the event reduces outcomes.[187,255] Treatment protocols are empirical and are predicated on cause and severity. With the emphasis on pain control, local anesthetics and analgesics can be administered, along with cold compresses, in an effort to moderate swelling and edema. Directed at diluting the effect of the extrusion, it has been suggested that the clinician immediately irrigate the canal with normal saline to encourage bleeding, both diluting the irritant and removing it from the site of the injury.[80,206] In addition, clinicians may immediately irrigate the canal and periradicular areas of the extrusion with saline, using a negative pressure irrigation device with the cannula placed at the radicular foramen. In one report, the subsequent reaction was short lived with this approach.[267]

After one day, warm compresses are substituted for the cold, and warm oral rinses are prescribed to stimulate the local microcirculation. Antibiotics are usually prescribed, and their administration commensurate with the severity of hard- and soft-tissue destruction and necrosis; milder cases can be treated with oral medications, whereas severe presentations are better controlled and modulated in a hospital setting with intravenous routes of delivery. The use of corticosteroids is equivocal; the control of the spreading edema must be balanced with the increased risk of infection. The patient should be monitored closely; a marked increase in the size or extent of the swelling or signs of impending airway obstruction mandate immediate referral to the hospital or a maxillofacial surgeon for more aggressive treatment and management. Lastly, reassuring patients that recovery, despite

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their current appearance, is usually complete and uneventful, will ease their anxiety. They should be informed as to why it occurred and what to expect during their recovery. Recovery should be monitored and documented daily until the resolution is imminent.

Prevention involves attention to detail and an appreciation for fluid dynamics. In summary, the clinician should do the following:

- Establish an accurate working length and avoid over-instrumentation/enlargement of the radicular foramen.
- If irrigating using positive pressure, employ a small side-vented needle placed no closer than 2 mm from the working length. Express the fluid slowly and observe that it is venting through the access cavity.[34]
- Carefully assess the canal integrity for signs of perforation or other large portals of fluid egress.
- Avoid wedging the needle tip in the canal space or inserting it beyond the working length.
- Confirm the identity of the solution prior to injection or irrigation.[124]

Instrument separation

The introduction of nickel titanium (NiTi) rotary instruments into endodontics revolutionized the way the root canal system is instrumented. However, with the advent of rotary NiTi files, there has also been a perceived increase in the occurrence of separated instruments,[101,281] with some reports indicating that separation of NiTi rotary instruments occurs more frequently than hand instruments.[343] On radiographic examination, separated instruments can usually be discerned by the distinct radiopacity within the canal. However, if the canal with a separated instrument is subsequently obturated with radiopaque materials, it may not be visualized on the radiograph. Upon removal of filling materials from the canal, a separated instrument may be visualized under the DOM or become apparent on radiographs for the first time (Fig. 20.4).

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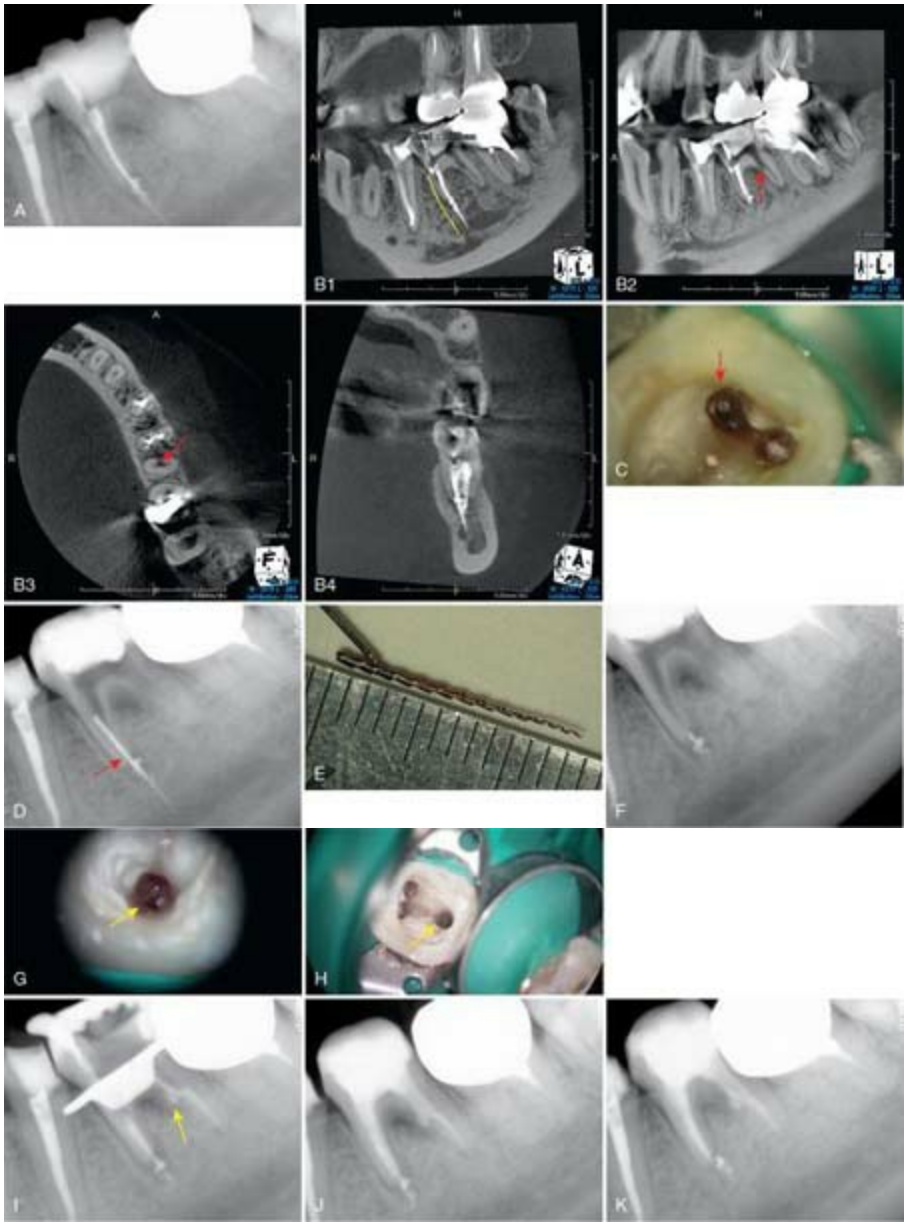

FIG. 20.4 **A,** Radiograph of the mandibular left first molar with a long indistinct separated instrument obviously longer than 4.5 mm in the mesial root that is extruded beyond the radicular foramen. **B1,** Sagittal view of cone-beam computed tomography (CBCT) showing the radicular end of the separated instrument is extended into the IAN and it measures 14 mm on it. **B2,** Sagittal view of CBCT showing root perforation in the distal canal *(red arrow)*. **B3,** Axial view of CBCT showing the perforation in the distal canal *(red arrow)*. **B4,** Coronal view of CBCT showing the radicular end of the separated instrument is in the IAN. **C,** Separated instrument in the mesiobuccal canal upon removal of root filling materials *(red arrow)*. **D,** Radiograph showing removal of the root fillings in the mesiobuccal canal revealed the overall length of the separated instrument *(red arrow)*. **E,** Retrieved separated

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instruments measuring 14 mm on the ruler. **F,** Radiograph showing the completion of the separated instrument removal. **G,** Perforation site in the distal canal *(yellow arrow)*. **H,** Perforation repaired with mineral trioxide aggregate (MTA) *(yellow arrow)*. **I,** Postoperative radiograph showing root canal fillings in all the canals and perforation repair *(yellow arrow)*. **J,** One-month postoperative radiograph revealing the periradicular lesions still present. **K,** Three-month postoperative radiograph showing periradicular healing.

Set of seven radiographs and two photographs marked A through F shows the removal of a long and sharp instrument in first molar of the lower jaw.

Set of photographs and radiographs marked G through K shows the healing of cavity along one side of the root canal near bifurcation in tooth.

Causes of instrument separation

A common cause of instrument separation is improper use.[101,285] Typical examples of improper use include inadequate access, overuse of the instrument, too much radicular pressure during instrumentation,[287,339] and the continuous use of a large instrument in a curved canal.[127,251] Additional contributing factors are operator experience,[229,331] rotational speed,[66,194] canal curvature (radius),[195,251] instrument design and technique,[39,177] torque setting,[102] manufacturing process,[4] the type of NiTi alloys used,[106] the type of rotational motion (continuous rotations vs. reciprocating motions),[235] the type of tooth,[64,218,299] and absence of a glide path.[233] Based on these factors, it is not surprising that the reported incidence of separated NiTi files ranges from 0.4% to 23%.[4,6,240,253,288,317]

Separated instruments seem to occur more frequently in molars than in anterior teeth, especially in mandibular molars. This high incidence of instrument fracture in molars may be related to accessibility to the canal, diameter of the root canal, and root canal curvature.[64,218,299] Endodontists have reported the incidence of instrument separation to be about 5%.[4,229] The clinician must inform the patient of the instrument separation and treatment options as well as potential complications of both the separated instrument retention and its attempted removal.

Endodontic instruments fracture as a result of cyclic fatigue or torsional fatigue[60] or a combination of both types of fatigue.[285,297,345] Cyclic fatigue is

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caused by repetitive compressive and tensile stresses acting on the outer portion of a file rotating in a curved canal, leading to cyclic failure without previous signs of plastic deformation.[229] Torsional failure occurs when the tip of the instrument binds but the shank of the file (driven by the handpiece) continues to rotate.[285] Shear fracture of the material then occurs when the maximum strength of the material is exceeded.[179] Clinically, cyclic fatigue seems to be more prevalent in curved root canals, whereas torsional failure might happen even in a straight canal.[55,372] Cyclic-fatigued instruments are reported to become less resistant to torsional fatigue failure,[168] whereas the use of rotary instruments at a high torque setting is reported to increase the risk of cyclic fatigue failure.[101] In general, smaller sized instruments with smaller tapers are more resistant to cyclic fatigue and more susceptible to torsional fatigue, while larger sized instruments with greater tapers tend to be opposite to those types of fatigue. Therefore smaller instruments should be used to shape the canal where cyclic fatigue is expected to occur. Interestingly, the use of reciprocal motions (as opposed to rotational motions) is reported to extend the life span of an instrument[69,371] and increase cyclic fatigue resistance.[235] It is recommended that instruments be used in a reciprocating motion when either torsional or cyclic fatigue is expected to occur while shaping a severely curved canal and that the instruments used to prepare such curved root canals be discarded after use to prevent instrument fracture.

Management of separated instruments

The management of separated instruments includes nonsurgical orthograde (mechanical or chemical) or surgical approaches. The mechanical removal involves the use of tools dedicated to separated instrument removal such as extractors, wire loops, post removal systems, ultrasonics, and laser irradiation (see also Chapter 10). The chemical removal involves the use of chemical solvents for instrument corrosion and electrochemical process for instrument dissolution. Surgical approaches include radicular surgery, intentional replantation, root amputation, or hemisection, which are in general considered relatively invasive due to the amount of dentin loss.

For a nonsurgical approach in removing a separated file, various methods and devices have been developed (see also Chapter 10). When a separated

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instrument extends above the root canal orifice, it can often be easily removed with a hemostat, Steiglitz forceps, a modified Castroviejo needle holder,[96] or a Perry plier.[357] A spoon excavator or a Caulfield retriever, or EndoCowboy (Koehrer Medical Engineering, Neuss, Germany) can also be used to engage and remove the separated instrument with coronally directed pressure. In the event that a hand file can bypass the separated instrument, a "braiding technique" can be used whereby several Hedström files are inserted along the bypassed instrument, the files are twisted to grasp the separated instrument, and it is then extracted as one unit. When a fractured instrument is below the canal orifice and cannot be bypassed, one basic method for removing the separated instrument requires the exposure of approximately 2 mm of the coronal portion of the separated file. This allows the use of an extracting device for engaging the file, securing it, and removing it. The Masserann kit (Micro-Mega, Besancon, France) is a popular system for the removal of separated instruments located within the straight part of the canal. This kit involves the use of trephine burs to expose the coronal portion of the separated instrument and for creating a space for an extractor. An extractor is then used to grasp and remove the separated instrument. The limitation of this system is that it cannot be used for cases involving separated instruments located beyond the midroot or in a curved canal, as this technique involves a considerable amount of dentin sacrifice that can weaken the root structure and increase the risk of perforation.[369] An alternative to the Masserann kit is the Endo Safety System.[359] This system differs from the Masserann kit in that it uses smaller diameter trephine burs and the extractor has its own unique mechanism for grasping instruments. The Endo Extractor (Brasseler Inc., Savannah, GA) can also be used in these types of cases. This system consists of a trephine bur to expose the coronal portion of the separated instrument and a hollow tube extractor that is threaded over the exposed portion of the separated instrument and bonded to it with cyanoacrylate adhesive.[110] The Canal Finder System (FaSociété Endo Technique, Marseilles, France) or the EndoPuls System (EndoTechnic, San Diego, CA) provides a different approach to a separated instrument. The system is composed of a handpiece and files that are designed exclusively for the system.[189] The system produces a vertical movement with a maximum amplitude of 1 to 2 mm, which decreases when the speed increases.[147] This vertical movement of the

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file will also help bypass an instrument fragment. The flutes of the file can mechanically engage with the separated instrument, and with the vertical vibration, the instrument fragment can be loosened or even retrieved.[148] The mechanical movement of the file can get aggressive, so the clinician must use caution to avoid root perforation, especially in a curved canal. These systems are technique sensitive and therefore results can vary between cases.

Ultrasonic instruments are very effective for the removal of separated instruments.[54,212,280] However, it is important to realize that separated NiTi files tend to fracture repeatedly when ultrasonics ia continuously applied to them, whereas stainless steel instruments are more resistant and are more easily removed with ultrasonics than NiTi instruments.[280,324,354] Small ultrasonic instruments allow continuous and improved vision of the field of operation. The use of ultrasonics, especially when performed under the DOM, enhances both preparation and removal procedures and can provide safety and accuracy in the process.[218,324,353] The ultrasonic tip is placed on the staging platform between the exposed portion of the separated file and the canal wall, and it is vibrated around it in a counterclockwise direction, applying an unscrewing force to the separated file. This technique will help with removing most rotary separated instruments that have a clockwise cutting action. If it is known that the separated file has a counterclockwise cutting action, then a clockwise rotation will be necessary. The energy applied will aid in loosening the file, and occasionally the file will suddenly exit out of the canal.

However, the use of ultrasonics does have some drawbacks. Upon ultrasonic activation, a separated file can be accidentally extruded into the bone, either radicularly or through a perforation when ultrasonics i applied to the top of the separated file or from the outer wall to the side of it. In addition, the ultrasonic activation of a separated instrument has been reported to separate into small fragments from the original fragment while troughing around it.[53] Smaller file fragments can easily get pushed into a more radicular level if they are pushed on the top by ultrasonics, making it more difficult to remove them due to the additional procedure, which would eventually increase the risk of perforation and extra dentin sacrifice, especially in the apical third or beyond the curve. At the same time, smaller file fragments can be considered easier and faster to remove than longer file fragments if they

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are visible simply because shorter file fragments requires less dentin sacrifice to loosen it than longer ones. On the other hand, such long instruments as separated at the shaft can also be easily removed as they can simply be grasped with a plier or forceps around the orifice and pulled out.[254] If the coronal end of the long file fragment is out of reach of the plier or the forceps, it will not be as easy. It all depends on how the separated instruments are managed to be successful in removing them as there are many variables involved in the procedures that affect the success in instrument retrieval.

The success rate for removing separated files varies from 33% to 95%[6,64,253,279,354] with the time required for using ultrasonic techniques varying between 3 and over 60 minutes.[8,212,331] The variations in success rates are due to the location of fractured instruments, the diameter of the root canal,[299] the degree of canal curvature,[8,252,313] the radius of curvature,[8,251] operator experience,[8,331] operator fatigue, and the length of the separated instrument.[330] Visualization and accessibility to the separated instrument play a major role in file retrieval.[64] In general, the success rate for removal is high for separated files located before the canal curvature, moderate for those located at the curvature, and low for those located beyond the curvature.[195,358] The success rate is also more favorable when the canal curvature is less and the radius of curvature is longer.[8] The combination of ultrasonic techniques and microscopes typically improves success rates in file removal.[64,100,218,354]

It has been recommended that removal attempts of separated instruments from root canals should not exceed 45 to 60 minutes because the success rates may drop with increased treatment time.[324] The reduced success rate could be related to operator fatigue or overenlargement of the canal, which compromises the integrity of the tooth and increases the risk of perforation. Any case where the separated instrument can be visualized under the DOM may be considered less challenging to manage.

It would be the worst-case scenario if the separated file was decided to be retained only after a lot of dentin was sacrificed and the canal perforation occurred during the instrument retrieval attempts. Hence it is in the best interests of both the patient and the clinician to make an accurate diagnosis and a predictable treatment plan using CBCT to prevent any procedural mishaps. In other words, separated instrument removal attempts should be

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made only after the preparation is completed based on the treatment plan that always includes surgical interventions and the retention of the separated instrument, as the separated file can be incorporated as part of the obturation and kept under observation.[92,97,253,314]

Conditions for separated instrument removal attempts

Root canal preparations for separated instrument removal followed by removal attempts can be made in either a dry or a wet environment. Dry conditions provide better visibility when using the DOM, preventing fewer additional procedural accidents.[281,313,324,353] For this reason, the root canal preparations for instrument removal should be conducted in a dry condition. However, heat generation by ultrasonics is inevitable,[115,134,181,199,299,313,324,353] and a temperature rise above 10°C on the external root surface can damage the periodontal tissues.[85,325] In addition, the separated instrument will be susceptible to secondary fracture when ultrasonic tips are in contact with the file.[332,353] Therefore it is recommended that pulsing and in/out motions be used to prevent the secondary fracture as well as the temperature rise during ultrasonic activation in preparation. At the same time, the ultrasonic tip should be activated at the lowest possible power setting.[42,88,200,353] Only after the loosening of the separated instrument is confirmed in preparation, the removal attempts can be made in a wet condition with a fluid to take advantage of cavitation and acoustic stream, which facilitates instrument removal. Ethylenediaminetetraacetic acid (EDTA) solution or oil with the lowest surface tension is recommended as the fluid for this purpose as EDTA solution removes hard-tissue debris produced during ultrasonic removal attempts and the oil enhances lubrication together with both cavitation and acoustic stream to remove the separated instrument.

Root canal preparation techniques

Root canal preparation for visible instrument retrieval

In the root canal preparation stage for instrument retrieval, the goal is to make the separated instrument “dance” by enlarging the canal and loosening it with ultrasonics based on the diagnosis and the treatment plan whether ultrasonic

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oscillation or the loop is used to get the separated instrument out in the instrument retrieval stage. The majority of separated instruments are NiTi,[317] with the mean length of separated instruments being 2.5 to 3.5 mm.[157] Many NiTi rotary instruments used have a.04 to.08 taper range with a tip size of #20 to #30, resulting in a coronal diameter of about 0.30 to 0.58 mm. If the coronal diameter of the separated instrument is smaller than 0.45 mm, then a #3 modified Gates Gliden bur (#3 MGG bur from the Terauchi File Retrieval Kit [TFRK] [DELabs Santa Barbara, CA]) should be used to enlarge the canal to the separated instrument, followed by the micro-trephine bur (MT bur) (DELabs for exposing the coronal portion of the separated instrument, with less risk than ultrasonics for secondary fracture of the separated instrument (see Fig. 20.20, *A* and *D*).[331,332] The inner diameter of the micro-trephine bur is 0.45 mm and it is then used, in a counter clockwise rotation, with the intent of unscrewing the separated instrument. This bur should be used at 600 rpm in wet conditions; if the rotational speed exceeds 600 rpm, unintentional ledge formation may occur, especially in a curved canal greater than 15 degrees.

If the coronal diameter of the separated instrument is larger than 0.45 mm or the canal curvature is greater than 15 degrees, then a larger NiTi rotary instrument, with tip size 0.15 mm (three sizes) larger than the coronal diameter of the separated instrument, should be used to enlarge the canal coronal to the separated instrument, followed by ultrasonic instruments to expose the coronal semicircular portion.

First, a few drops of mineral based oil with lubricant additives is placed into the canal on the separated file to facilitate the separation of it from the canal wall, and then excess oil is removed with paper points or mild airblows for the visible root canal preparation with ultrasonic tips. The mineral oil almost instantly permeates the spaces between the canal walls and the tightly wedged file fragment and separates them just like loosening a rusty tightened screw from the metal plate (Fig. 20.5). Then the spoon tip with the concave portion facing the separated file (TFRK-6/12, DELabs) (see Fig. 20.20, *B1*, *C2*, and *D*) will be introduced into the thin gap between the separated file and the inside curve of the canal to create one quarter space of the circle (90-degree semicircular space) that is approximately one third of the separated file length. Second, the straight tip (TFRK-S, DELabs) (see Fig. 20.20, *B2*,

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C2, and *D*) is introduced to expand the space to complete the 180-degree semicircular space on the inside curve until the separated file gets loosened. When the depth of the space created is longer than one third of the file length and the semicircular space is greater than 180 degrees, the separated file will most likely get loosened (Fig. 20.6). The ultrasonic tip used for this purpose should be as thin as possible, allowing the clinician to both visualize the operative field and prevent both overenlargement of the canal wall and pushing the separated instrument radicularly (Fig. 20.7). In addition, the ultrasonic tip should always be applied to the area on the inner canal curve being extended in the radicular direction (Fig. 20.8) where the outer canal wall is in contact with the separated instrument to prevent secondary fracture, not where the outer canal wall is not in contact with the separated file (see Fig. 20.8, *H*).[332] If the ultrasonic tip is placed in the space outside the curve and ultrasonic energy is given to the separated instrument from the outer curve, the force applied will redirect the separated instrument in an apical direction (see Fig. 20.8, *D* and *E*), eventually pushing it more apically and making the instrument retrieval more difficult, whereas the force applied from the inner curve to the separated instrument will consequently reorient the separated instrument in a coronal direction (see Fig. 20.8, *F* and *G*). In most cases, the coronal one third of the separated file is the main portion engaged in the canal wall with more stress leaning toward the outer wall. This portion can get less resistant to mechanical force for disengagement when the mineral based oil injected permeates the tiny interfacial space between them to loosen the separated file. Therefore cutting the inner dentin wall contacting the coronal third of the separated file should typically be enough to disengage it (Fig. 20.9). For example, if the separated file fragment is 3 mm long, the 180-degree semicircular space needs to be extended radicularly about 1 mm on the inside curve to help the ultrasonic tip loosen it. Due to the force directed from the inner wall to the fragment during this ultrasonic preparation phase, the separated file may come out accidentally by ultrasonic activation at any moment (Fig. 20.10, *A*). However, a fatigued separated instrument is already extremely susceptible to secondary fracture by ultrasonic activation.[332,353] For this reason it is safer to create a semicircular space on the inner wall instead of troughing around it, and both pulsing and pecking motions should be applied to it while ultrasonics is activated to prevent

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instrument fracture and temperature rise, especially in dry conditions.[85,325,332,353] The depth of the semicircular space created on the inner curve must be not only longer than one third of the fragment length but also evenly the same length along the canal walls to allow the disengagement of the separated instrument. If the separated instrument is not loosened after the space creation, the depth of this space should be extended in a radicular direction until it is loosened. In general the longer the file fragment is, the more preparation time is needed to loosen it. The separated instrument must be seen "dancing," "not flexing," before making instrument retrieval attempts. The difference between the separated instrument dancing and flexing is that the separated instrument is considered dancing when it is seen moving from one place to another place, whereas it is still considered flexing when seen moving from one place to another and getting back to the original place (Fig. 20.11).

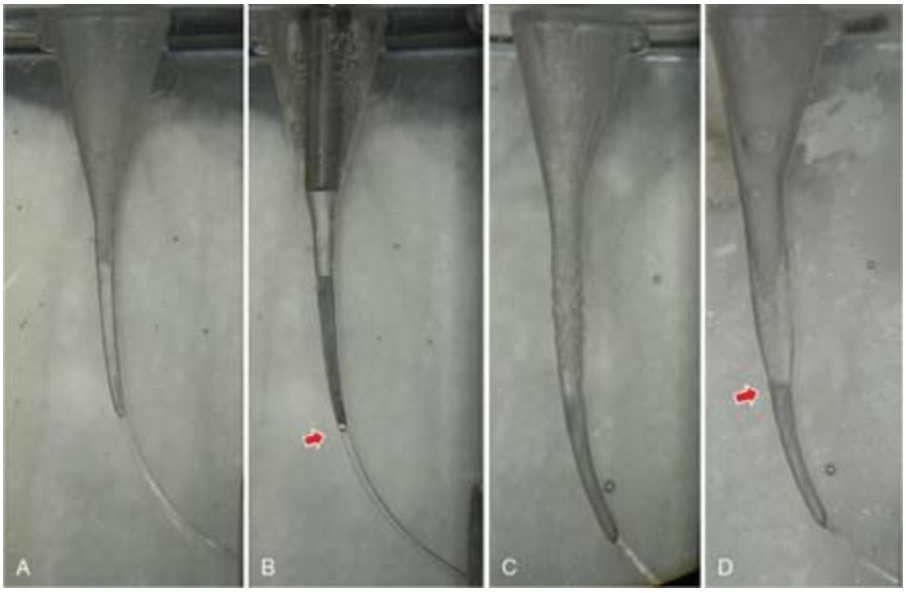

FIG. 20.5 **A,** Separated file covered with debris. **B,** Mineral oil immediately permeated the spaces between the canal walls and the separated file revealing the surface of the file and broke through the entire file. **C,** Separated file covered with debris. **D,** Water did not permeate those spaces at all.

Set of four radiographs marked A through D shows the role of mineral oil and water in removing the separated files from canal walls.

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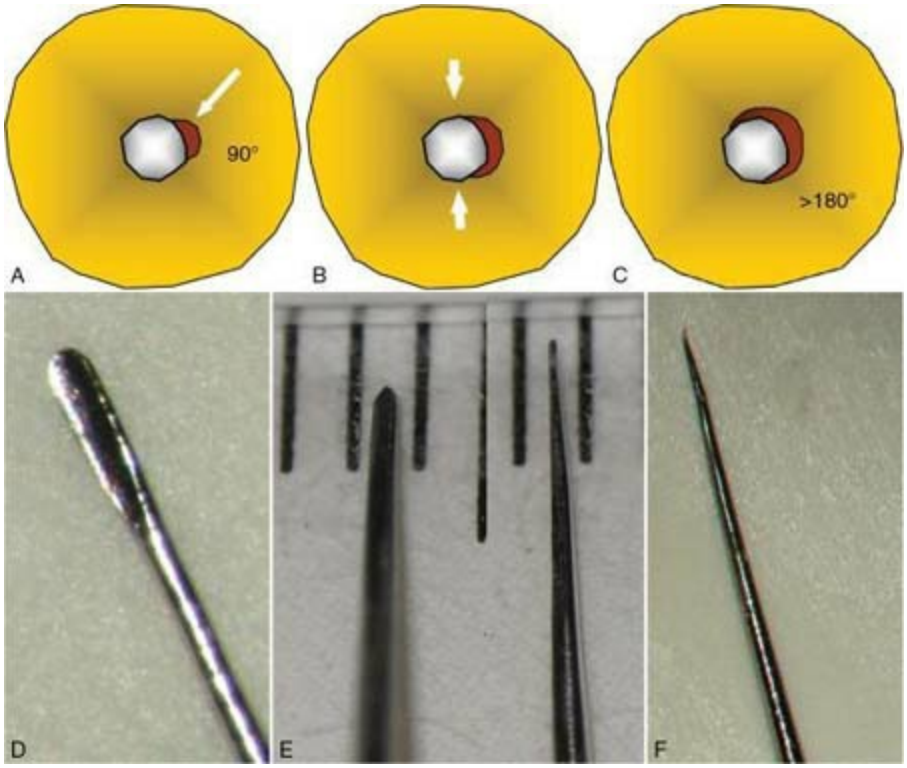

FIG. 20.6 **A**, Ninety-degree semicircular space created on the inner canal wall with the spoon tip on the microscopic view *(white arrow)*. **B**, A 150-degree semicircular space created on the inner canal wall with the straight tip, which is still not wide enough to make it "dance" because of the canal walls on both sides still holding the separated file *(white arrows)*. **C**, More than 180-degree semicircular space created on the inner canal wall with the straight tip, which results in loosening the separated file. **D**, Magnification of the spoon tip. **E**, Magnification of the Katana tip; a frontal view on the left (0.5 mm in width) and a side view on the right (0.1 mm in thickness). **F**, Magnification of the straight tip (0.1 mm in diameter).

Set of six illustrations marked A through F shows 90-degree, 150-degree, and more than 180-degree space created along one side of the root canal in tooth by using spoon tip, Katana tip, and straight tip, respectively.

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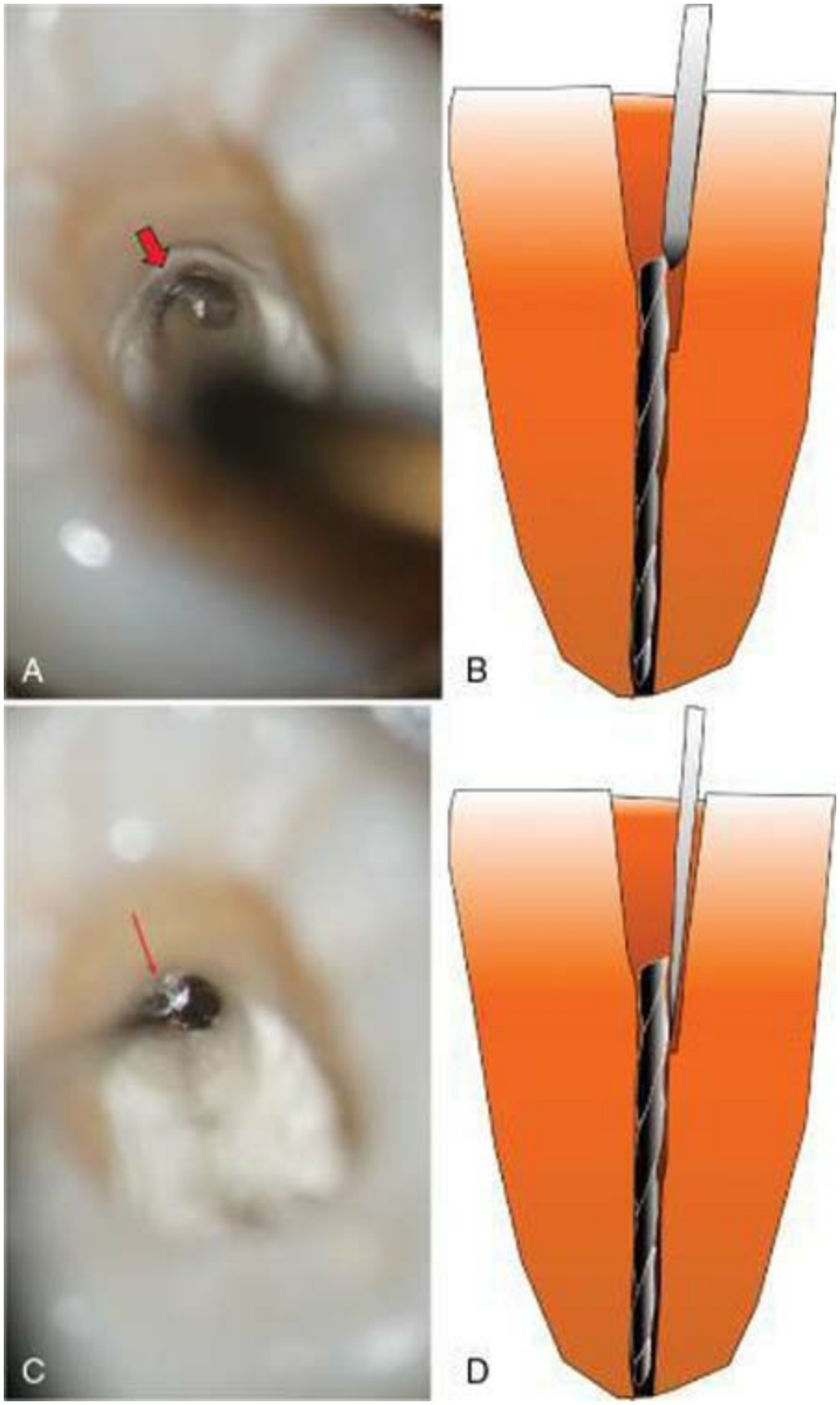

FIG. 20.7 **A**, Thicker ultrasonic tip touches the top of the separated instrument before being placed into the space on the canal wall *(red arrow)*. **B**, Side view shows the thicker ultrasonic tip in relation to the separated instrument, which may result in pushing it into a radicular direction. **C**, Thinner ultrasonic tip can be placed into the space on the

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canal wall without touching the top of the separated instrument *(red arrow)*. **D**, Side view shows the thinner ultrasonic tip in relation to the separated instrument.

Set of four illustrations marked A through D shows thick ultrasonic tip touching the top of spiral shaped separated file, whereas thin ultrasonic tip covering the space between the separated file and canal wall, respectively.

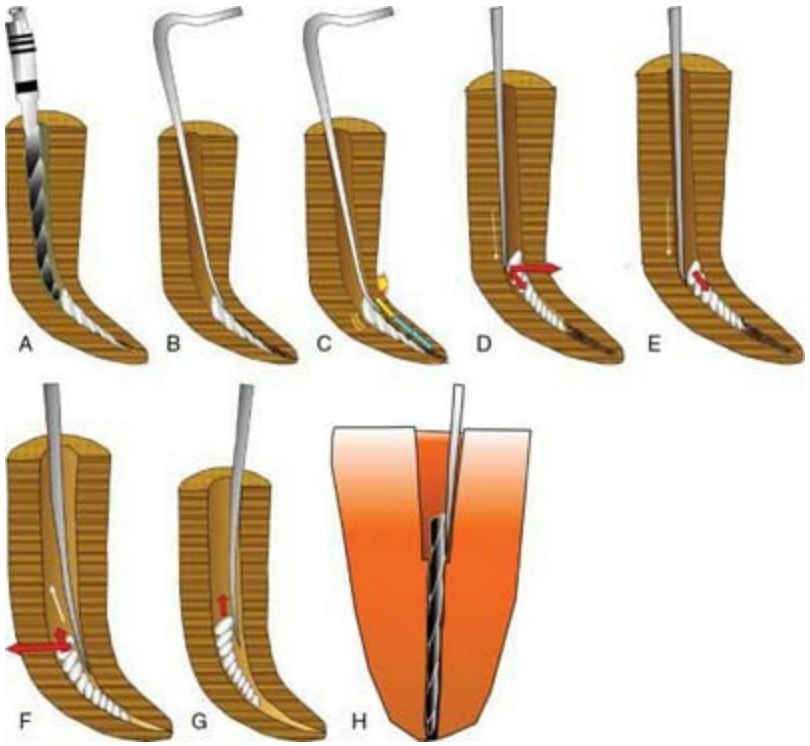

FIG. 20.8 **A,** Canal enlarged to the separated file with a large diameter rotary file. **B,** Space created on the inside curve with the spoon ultrasonic tip, followed by the straight tip. **C,** Space extended to about one third of the file length until the separated file dances. **D,** Ultrasonic activation from the outer curvature. **E,** Separated file redirected in a radicular direction by ultrasonic activation from the outer wall. **F,** Ultrasonic activation from the inner curvature. **G,** Separated file redirected in a coronal direction by ultrasonic activation from the inner wall. **H,** Ultrasonic tip placed on the separated file where there is no canal wall behind it, which may result in secondary fracture.

Set of seven illustrations marked A through G shows the widening of root canal resulting in the movement of separated file in upward direction. Next to it, illustration H shows the thin ultrasonic tip placed little below the tip at one side of separated file.

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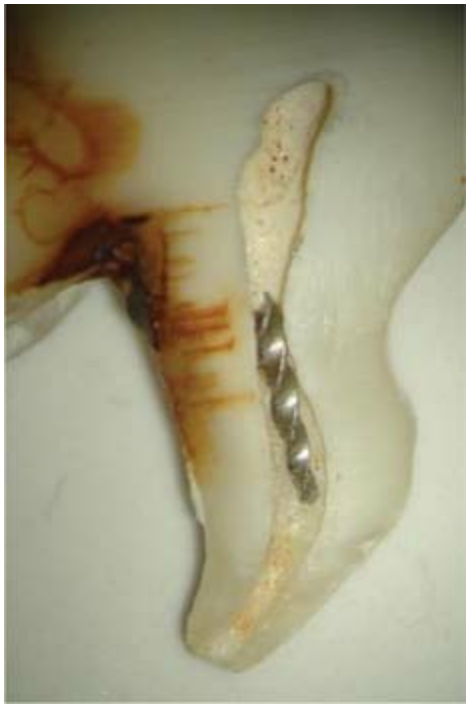

FIG. 20.9 The coronal one third of the file length is typically the part to be stuck in the canal walls, whereas the radicular portion of the file is typically not in contact with the canal walls. Stress is concentrated mostly on the coronal one third of the file length.

The extracted tooth shows root canal having a spiral rod stuck almost at the center.

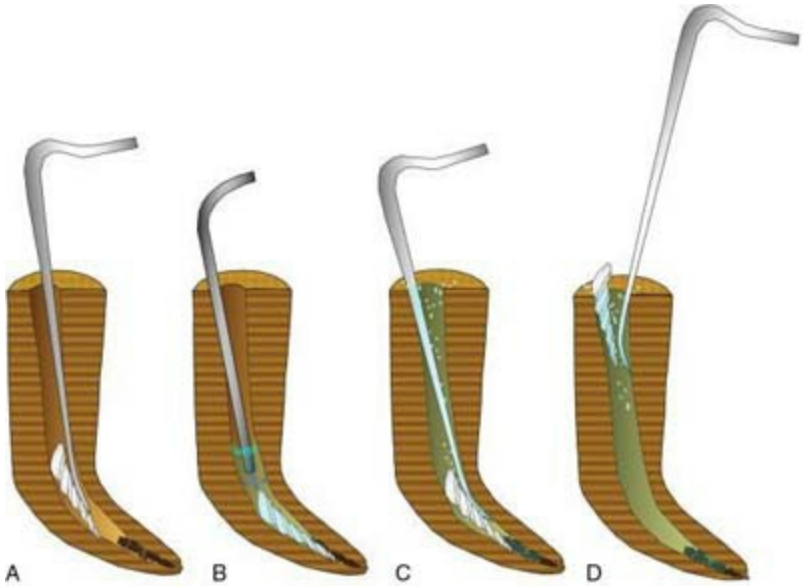

FIG. 20.10 **A,** Separated file may accidentally come out with ultrasonic

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activation during a preparation phase. **B,** EDTA solution is put into the canal to take advantage of cavitation and acoustic streaming effects for instrument retrieval. **C,** Ultrasonics must be activated within the space created. **D,** Separated file is removed from the canal with ultrasonics in the presence of EDTA solution or oil.

Set of figures marked A through D shows the steps involved in removing the conical spiral piece from the root canal by using endosonic file and blue-colored EDTA solution.

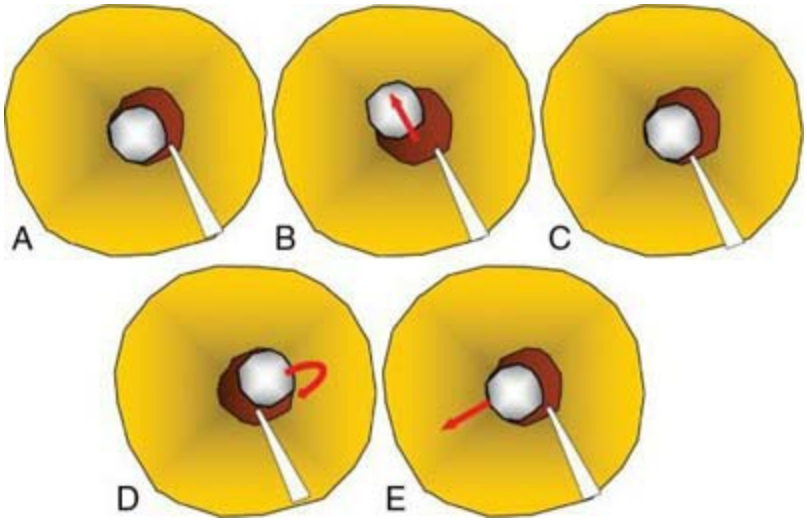

FIG. 20.11 **A**, Separated file in the canal with the space created on the inner wall *(right side)*. **B**, Separated file shifted to a different place in the space created *(red arrow)*. This movement shows the separated file is "dancing." **C**, Separated file in the canal with the space created on the inner wall *(right side)*. **D**, Separated file flexing to a different place *(red arrow)*. **E**. Separated file returning to the original place *(red arrow)*. This movement shows the separated file is "flexing."

Set of five figures marked A through E shows a circular separated file in the canal located at lower left, upper left, lower left, central right, and its original position, respectively.

Root canal preparation for nonvisible instrument retrieval

When the separated file is not visible beyond the curve, the mineral oil should be present all the time during the procedure to help it loosen the file fragment while preparing the root canal for instrument retrieval with ultrasonics. The key to success in loosening the separated file in non-visible

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status is to get a sticky feeling with ultrasonic tips in the space on the inner canal wall. The ultrasonic tip used in this situation should be thin and sharp enough to feel the tiny gap between the inner canal wall and the separated instrument. First, a thin spoon-shaped tip (TFRK-6/12, DELabs) or a customized sword-shaped tip (any ultrasonic tip converted into a sword-shaped tip or a “Katana” tip) is precurved and placed in the space to grope for a sticky feeling while obtaining a tactile sensation on the fingers holding the handpiece (Fig. 20.12). If a larger sized tip is used, the tip will most likely hit on the top of the separated instrument before being placed into the gap between the fragment and the inner wall. It is also very typical to hear a clicking sound and get a sticky feeling when a sharp ultrasonic tip shifts from the separated file to the canal wall into the gap. When it is placed in the gap with a sticky feeling, a radiograph should be taken with the ultrasonic tip in it to confirm its position. Then the tip is activated in it to create a 90-degree semicircular space. The ultrasonic tip is simply activated directly on the file fragment from the inner curve in both pecking and pulsing motions to prevent secondary fracture and breakage of the tip without creating a 180-degree semicircular space or expanding the space laterally as it is impossible to do so in nonvisible status. The intensity of ultrasonic activation should be as low as practical for the same reasons. As this conservative root canal preparation continues with ultrasonic oscillation directed to the separated instrument from the inner wall, any dentin walls contacting the separated file will be eliminated by indirect ultrasonic oscillation transferred to it from the vibrating tip, which will eventually result in loosening it (Fig. 20.13). When the sticky feeling is lost from the ultrasonic tip or the separated file emerges from the nonvisible curve with ultrasonics, the root canal preparation for instrument retrieval can be considered complete, and then the instrument retrieval attempts should be made from this point (Fig. 20.14). The advantage of this conservative preparation technique is the clinician can be minimally invasive in preparation, while the disadvantage of this technique is that it is unpredictable when to make the separated instrument dance and the secondary fracture or breakage can occur if it is performed aggressively. It also requires the patience to be successful in loosening the fragment in this technique as it is not predictable.

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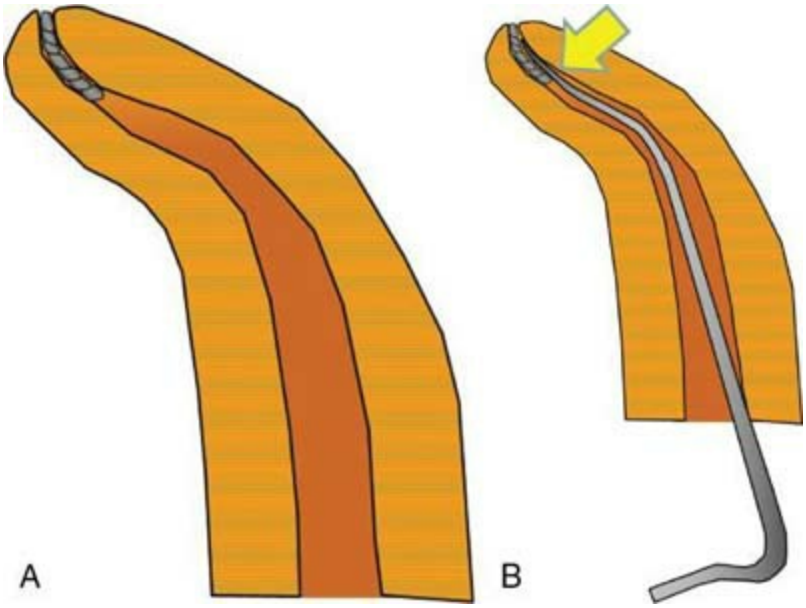

FIG. 20.12 **A**, Nonvisible separated file around the second curve, beyond the first curve. **B**, Thin ultrasonic tip placed in the space on the inner canal wall of the second curve.

A) A spiral shaped piece is inserted into the root apex of tooth.

B) Sharp ultrasonic tip touches the piece near the root apex.

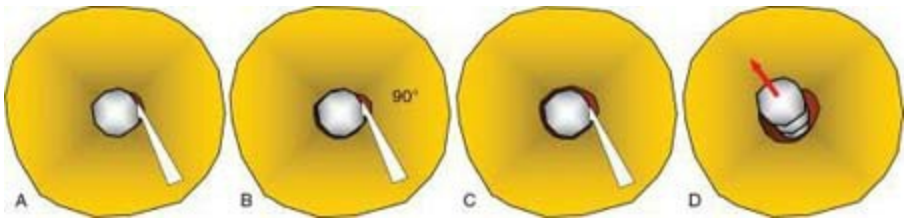

FIG. 20.13 **A**, Thin ultrasonic tip placed into the gap between the inner wall and the separated file and activated. **B,** Ninety-degree semicircular space created in this gap with ultrasonics. **C,** Ultrasonics activated directly on the separated file from this space. **D,** Canal walls contacting the separated file eliminated with ultrasonic oscillation transferred to the separated file from the vibrating ultrasonic tip. **D,** Separated file loosened and dislodged from the original position *(red arrow)*.

Set of four illustrations marked A through D shows the removal of circular separated file in the root canal from its original position by using ultrasonic tip

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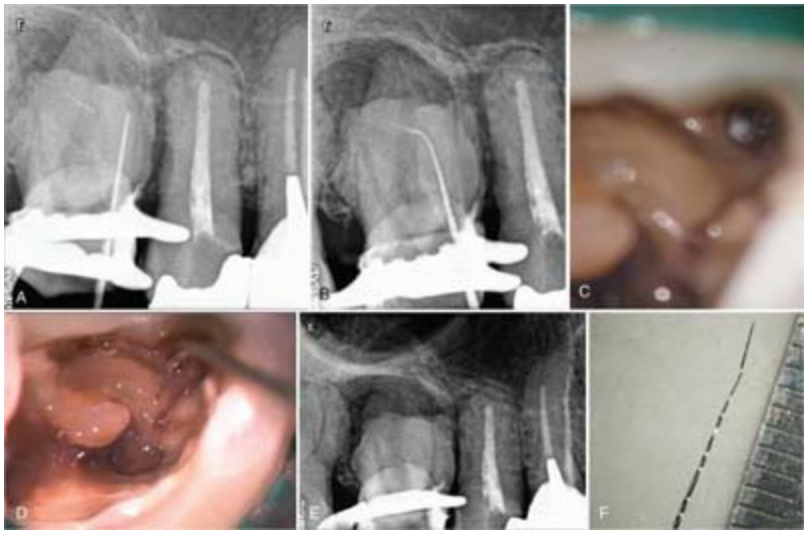

FIG. 20.14 **A,** Separated file beyond the severe curve in relation to the file placed in the mesiobuccal (MB) canal. **B,** Thin ultrasonic tip placed in the gap between the inner wall and the separated file. **C,** Separated file is not visible in the MB. **D,** Separated file coming out of the canal. **E,** Removal of the separated file confirmed on a radiograph. **F,** Retrieved separated file along with the original file

Set of radiographs and photographs marked A through F shows the removal of a piece of the separated file left out in the canal in the mid root of tooth and the piece further placed close to original file.

When the separated file is found to be loose beyond the radicular foramen, it is not necessary to prepare the canal for its retrieval because it is not engaged in the canal wall, but it just lies in the radicular extent of the canal space (Fig. 20.15). The separated file in this status can be simply irrigated out of the canal using saline with ultrasonic irrigation devices such as ProUltra PiezoFlow (Dentsply, Tulsa, OK) and GentleWave (Sonendo, Inc, Laguna Hills, CA) (Fig. 20.16).

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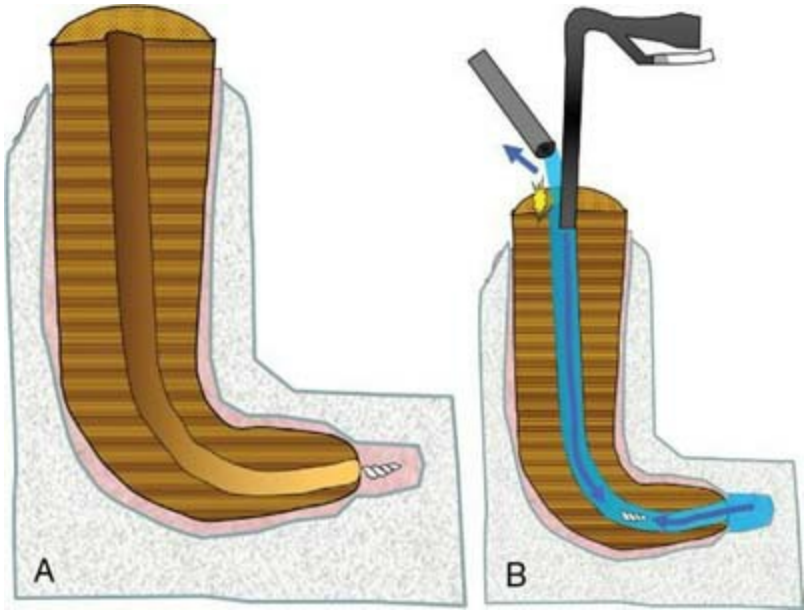

FIG. 20.15 **A**, Separated file loose beyond the radicular foramen. **B,** Ultrasonic irrigation such as ProUltra PiezoFlow using saline may help retrieve the separated file which is not engaged in the canal wall.

A) Separated file lies close to the root tip of tooth.

B) Separated file moves to the root canal near its apex, which comes out by using ProUltra PiezoFlow instrument and blue-colored solution.

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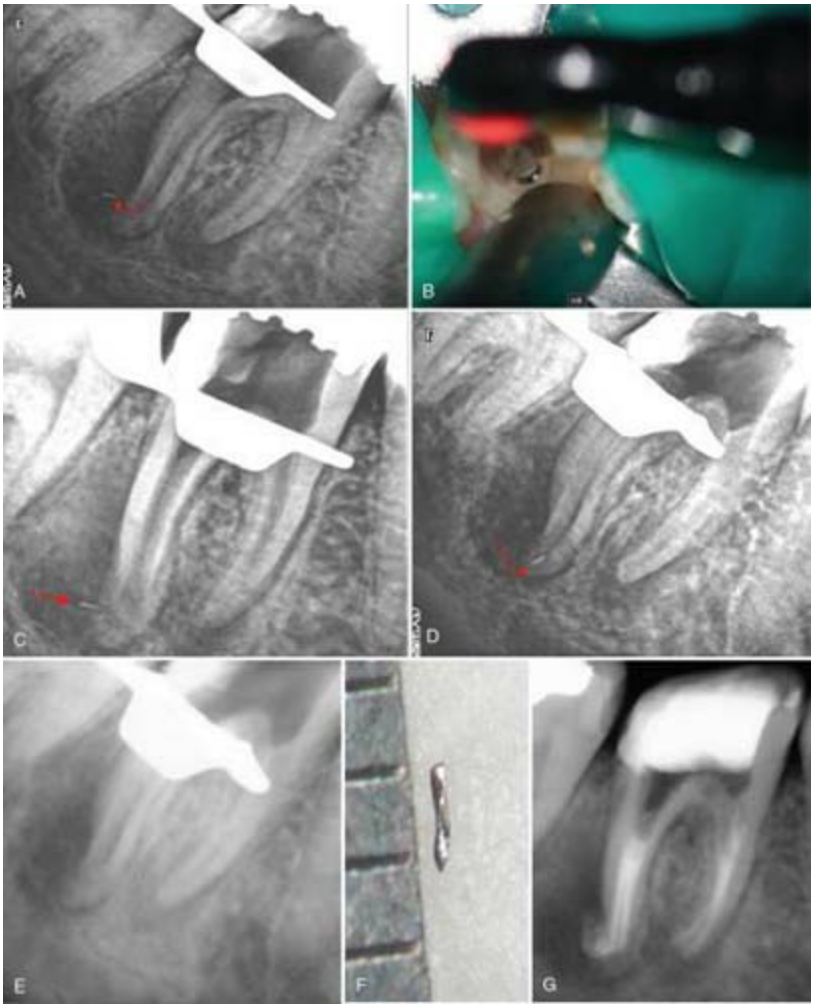

FIG. 20.16 **A,** Separated file pushed beyond the radicular foramen. **B,** PiezoFlow activated using saline to create cavitation and acoustic stream. **C,** Separated file approaching the radicular foramen with the PiezoFlow. **D,** Separated file brought back into the canal. **E,** Separated file retrieval confirmed on the radiograph. **F,** Retrieved separated file on a ruler. **G,** Root canals obturated with mineral trioxide aggregate (MTA).

Set of one photograph and six radiographs marked A through G shows the removal of separated file, depicted as spiral shaped piece, from outside the root apex to top of the root by using PiezoFlow instrument.

Instrument retrieval techniques

Use of ultrasonic oscillation

Once the root canal preparation for instrument retrieval is completed, it is

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important to confirm that the canal wall is smooth from the separated file to the coronal extent, with no overhangs on the outside canal wall. A retrospective study and the author's empirical findings show that when the separated instrument is shorter than 3 mm or between 3.1 and 4.4 mm with the curvature smaller than 30 degrees, the clinician will most likely be able to retrieve it with ultrasonics alone.[330] Then the canal is rinsed with the silicone based oil or EDTA solution and is filled with it up to the cavosurface, "not just in the root canal alone," in order to take advantage of cavitation and acoustic streaming for instrument retrieval in wet conditions (see Fig. 20.10, *B*). The ultrasonic tip placed is continuously activated at a power setting that is about 5% higher than it was in the preparation stage with short up-and-down strokes within the space created on the inner curve until the separated instrument is removed, which is typically completed in 10 seconds (see Fig. 20.10, *C* and *D*) (Fig. 20.17). If the separated instrument didn't come out of the canal in 10 seconds, there are three possibilities of the failure; the first one is that the space between the ultrasonic tip and the outer canal wall might be smaller than the diameter of the separated instrument (Fig. 20.18) or this space might be closed during the ultrasonic retrieval attempts, the second one is that the separated instrument might be pushed on the top back into the original position or from the outer wall by the ultrasonic tip (see Fig. 20.18, *F*), and the third one is that the amount of the fluid filled in the canal at the time of the ultrasonic removal attempts might not be sufficient enough to let the separated file flow out of the canal with the aid of acoustic streaming and cavitation (Fig. 20.19). If any of those possibilities is not applicable to the case and the instrument retrieval attempts were not successful with ultrasonics in wet conditions, or when the separated instrument is longer than 4.5 mm or between 3.1 and 4.4 mm with the curvature greater than 30 degrees, the loop (Yoshi Loop, DELabs) (Fig. 20.20, *C1*, *C2*, and *D*) should be used to retrieve the separated instrument.[330]

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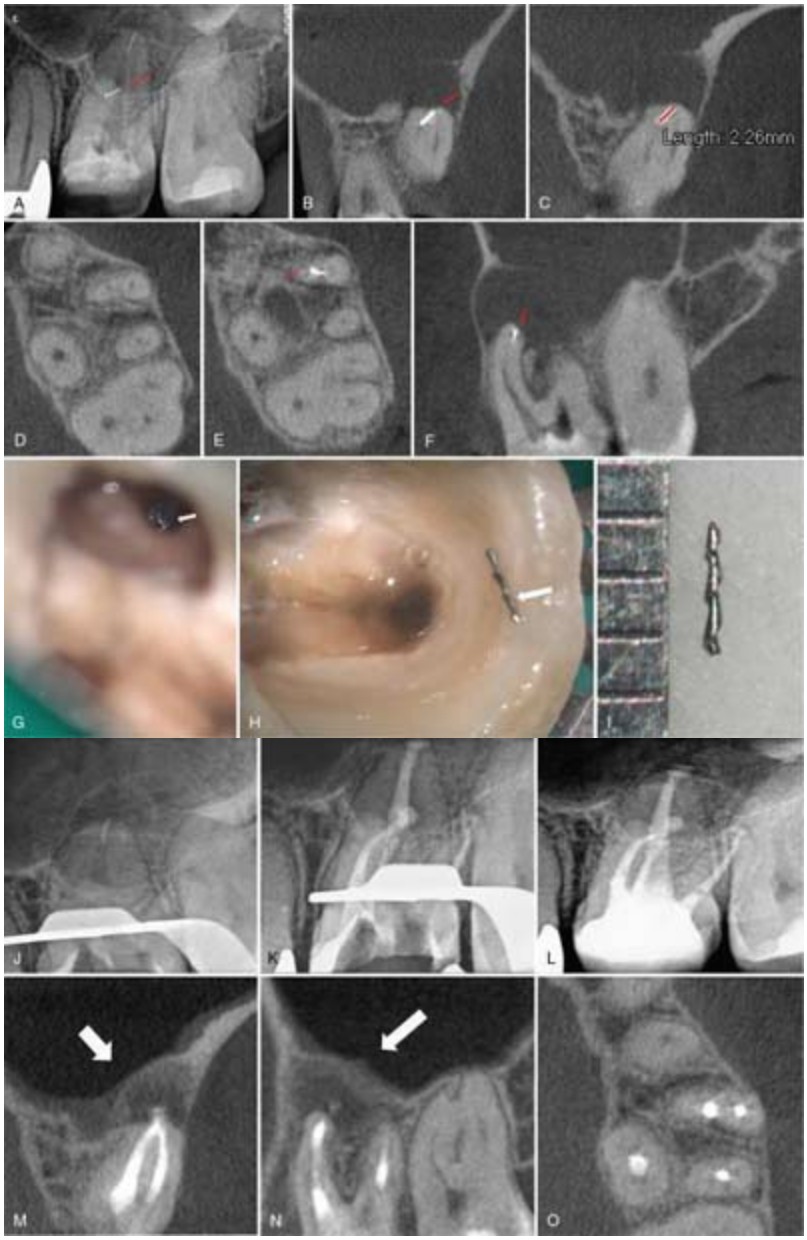

FIG. 20.17 **A**, Preoperative radiograph of a maxillary left first molar showing a separated instrument beyond the curve in the mesiobuccal root canal with large periapical lesions *(red arrow)*. **B**, Coronal view of cone-beam computed tomography (CBCT) shows the separated instrument in the buccally curved MB2 canal with a large periapical lesion developing into the sinus floor *(red arrow)*. **C**, Separated instrument measures 2.26 mm on the coronal view. **D** and **E**, Axial view shows the separated instrument in the MB2 canal directed toward the MB1 canal. **F**, Sagittal view shows the separated instrument in the MB2 canal lying in a distal direction with a large periapical lesion *(red arrow)*. **G**, Magnification of a partially visible portion of the separated instrument in the MB2 canal *(white arrow)*. **H**, Magnification of the separated instrument that came out of the canal with ultraspnics *(white*

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arrow). **I**, Retrieved separated instrument measured 2.3 mm on a ruler. **J**, Interoperative radiograph confirming removal of the separated instrument from the MB2. **K,** Postoperative radiograph showing the final root fillings with mineral trioxide aggregate (MTA), which reveals dentin sacrifice for removal of the separated instrument was minimum. **L**, Three-month postoperative radiograph showing nice periapical healing. **M**, Three-month postoperative coronal view of CBCT showing periapical bone reformation associated with the mesiobuccal root *(white arrow)*. **N**, Three-month postoperative sagittal view of CBCT showing periapical bone reformation associated with the mesiobuccal root *(white arrow)*. **O**, Three-month postoperative axial view of CBCT showing root fillings in MB, MB2, DB, and P root canals.

Set of radiographs and photographs marked A through I shows the separated file left out near the center of root canal in first molar of upper jaw being removed, followed by its magnified view on roller.

Set of six radiographs marked J through O shows the filling in root canal and high-density tissue in periapical region after removing the separated file in left first molar of upper jaw.

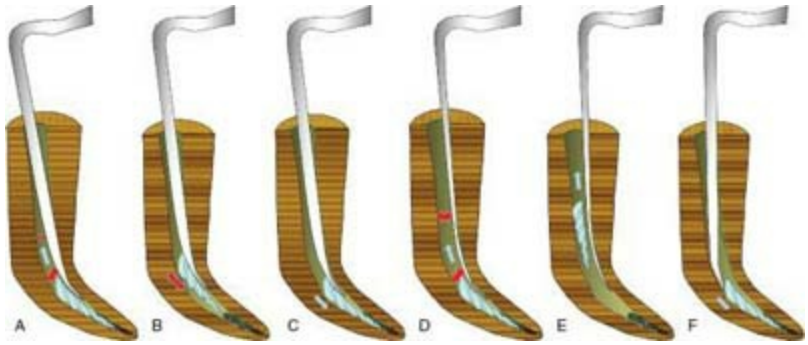

FIG. 20.18 **A**, Space between the ultrasonic tip and the canal wall is smaller than the diameter of the separated file. **B,** Separated file comes out and bumps against the ultrasonic tip. **C**, Separated file bounces back into the original position. **D**, Space between the ultrasonic tip and the canal wall is wider than the diameter of the separated file. **E**, Separated file comes out through the space. **F,** Ultrasonic tip placed on the top of the separated file in the space on the outer wall.

Set of six illustrations of root marked A through F depicts the separated file reaching the center and coming out from the canal. After which, the separated file again moves to its original position deep into the root apex.

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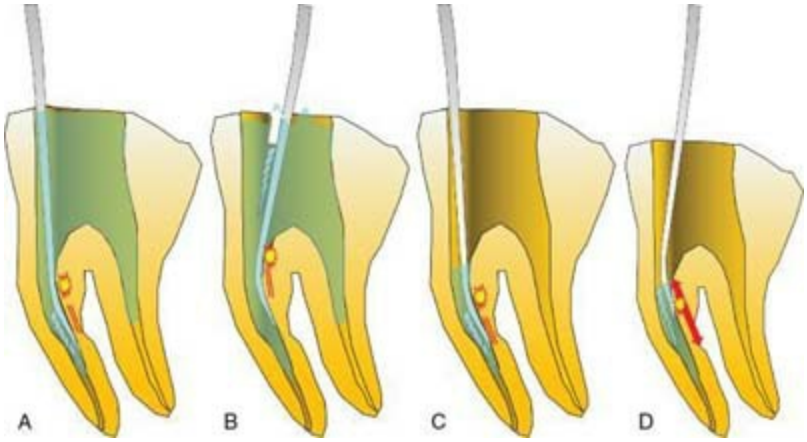

FIG. 20.19 **A,** Fluid filled in the canal and the pulp chamber. **B,** Separated file is coming out of the canal to the pulp chamber in the fluid with ultrasonics *(white arrow)*. **C,** Fluid filled only in the canal. **D,** Separated file fluctuating within the canal space filled with fluid *(red arrows)*.

Four illustrations of root system of tooth marked A through D show a spiral separated file moving back and forth below the pulp chamber in the left root canal due to less blue solution.

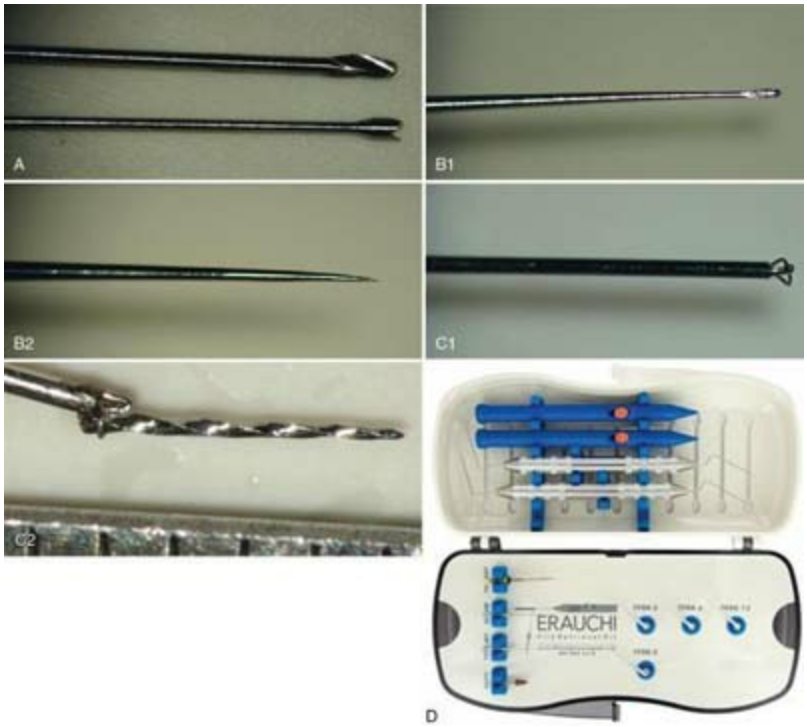

FIG. 20.20 Terauchi File Retrieval Kit (TFRK). **A,** Modified #3 Gates Glidden bur that is also called GG-3M *(upper)* and microtrephine bur

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that is also called TFRK-MT *(lower)*. **B1,** TFRK-6 ultrasonic tip (the tip portion looks like a spoon and is also called a spoon tip. It has a micro-concave on the spoon, which turns toward the handpiece at the 6 o'clock position. **B2,** TFRK-S ultrasonic tip (the tip portion looks like a sharp spear and is also called a straight tip). **C1,** Yoshi loop (TFRK-L), which captures a separated instrument. This micro-lasso is comprised of a tiny wire loop at the end of a stainless steel cannula, with a sliding handle that tightens the loop when pulled. **C2,** Yoshi loop holding a separated file from an actual case. **D,** Terauchi File Retrieval Kit (TFRK) in an autoclavable cassette case. The kit includes a GG-3M, TFRK-MT, TFRK-6 ultrasonic tip, TFRK-12 ultrasonic tip, TFRK-S ultrasonic tip, Yoshi loop, TFRK- GPR, and TFRK-ME, High Speed Polishing Point (HSPP), NiTi GT #70/.12 Rotary File (GT70.12).

A) Two instruments have rod with flattened end.

B1) A conical rod with oval end.

B2) A wide conical rod with sharp end.

C1) A tubular instrument has wing-shaped end with wide base and a thin protruding tube at the center.

C2) A spiral shaped instrument.

D) Pack of Terauchi File Retrieval Kit.

Longer separated instruments, especially around a severe curve, are apt to be touching all over the canal walls, which creates more friction as it is reoriented in a coronal direction and makes it more resistant to instrument retrieval with ultrasonics alone, requiring more mechanical force to pull it out of the canal. This is why the loop is necessary to retrieve those types of separated instruments.

Use of the loop

It requires at least 0.4-mm-diameter space to place the loop in the canal, and the coronal portion of the separated instrument needs to be peripherally exposed by at least 0.7 mm to be grasped by the Yoshi loop (Fig. 20.21, *A*), The loop size must be adjusted to fit the coronal diameter of the separated file using an endodontic explorer, such as a DG16 Endo Explorer (Hu-Friedy, Chicago, IL) (see Fig. 20.21, *G*). The tip of a DG16 is placed in the loop and the loop is contracted around it. The apical portion of the DG16 is used to make the loop size smaller and the coronal portion is used to make it larger

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when the loop is tightened around it. The loop is then pre-bent to 45 degrees instead of 90 degrees to both facilitate the placement of the loop over the separated instrument and be minimally invasive as bending it to 90 degrees requires more room compared to 45 degrees (see Fig. 20.21, *E* and *F*). The loop is then brought into the canal and placed over the exposed portion of the separated file as the loop is pushed back to 90 degrees upon it (see Fig. 20.21, *B* and *C*). Subsequently, the loop is tightened over the separated instrument by sliding down the handle. The separated instrument will be retrieved by pulling the loop out of the canal in various directions with a swaying motion to dislodge it from the canal walls (see Figs. 20.21, *D* and 20.20, *C2*). A couple of pull motions in several directions will usually dislodge the file fragment and complete the case for separated file removal.

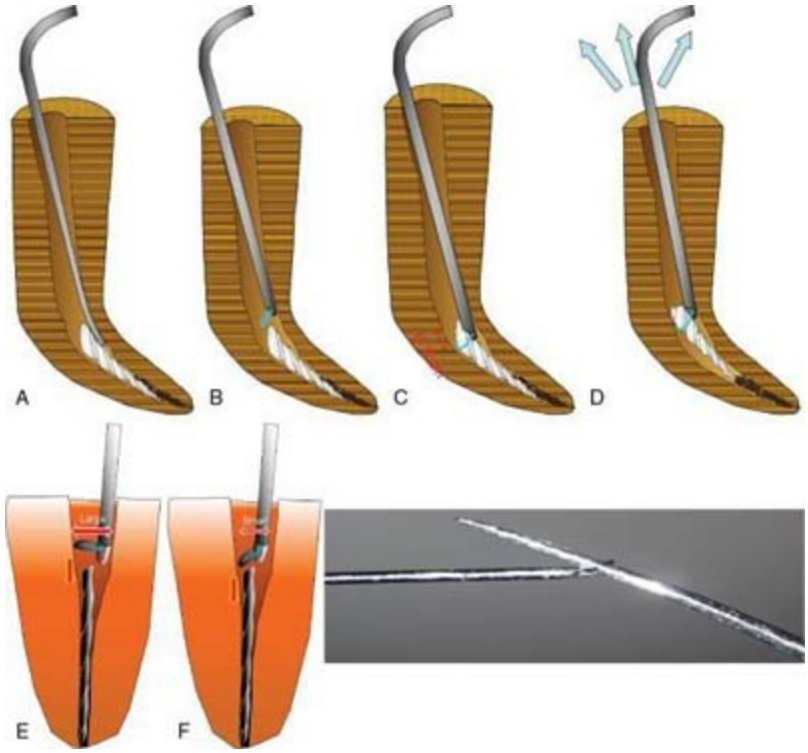

FIG. 20.21 **A,** Coronal portion of the separated file should be exposed at least 0.7 mm above the bottom of the space created around it. A #40 plugger should be placed in the space to assess the available space prior to the placement of the loop. **B,** Approaching the separated file with the loop bent to 45 degrees against it. **C,** Loop is pushed back to 90 degrees as it is placed over the separated file. Removal with the loop requires the diameters of the loop device (0.4 mm) and the separated instrument and a depth of at least 0.7 mm. **D,** Separated file

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is pulled out with the loop device in various directions. **E,** Loop prebent to 90 degrees occupying more room in the canal. **F,** Loop pre-bent to 45 degrees requiring minimum room in the canal. **G,** Loop size is adjusted using an endodontic explorer.

Set of four illustrations marked A through D shows the process of removal of separated file from the root apex by using loops on endodontic explorer, followed by two illustrations marked E and F that depict the separated file moving back to the root apices due to large and small loops. The last illustration marked G shows a sharp conical rod placed on the tip of endodontic explorer.

Surgical approaches for the management of separated instruments represent a second category of procedures for dealing with this iatrogenic event (see also Chapter 11). Surgical approaches should be considered in the following situations[201]:

- As a last resort if other nonsurgical approaches fail, posttreatment disease develops, and the tooth is strategically important to retain
- As a first approach when periradicular pathosis is present at the time of instrument separation, especially if the separated instrument is found in the apical third of the canal coexistent, presumably with persistent extraradicular infection
- As a first approach when the radicular portion of the separated instrument is already outside the root, when an orthograde approach is unsuccessful, or when a significant amount of dentin must be sacrificed for instrument removal

Prognosis

When an endodontic instrument separates in a canal during cleaning and shaping procedures, and if it hinders adequate cleaning of the canal, then the prognosis may be affected. Moreover, attempts to remove a separated instrument from a curved canal may cause additional complications such as excessive removal of dentin structure, secondary instrument fracture, root perforation, or even root fracture.[134,286,313,317] However, there are cases in which the prognosis is not compromised, depending on what stage of instrumentation the file fracture occurred, the preoperative diagnosis of the pulp and periradicular tissues, and whether or not the separated file can be

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removed or bypassed.[317] The presence of a separated instrument per se in a canal or outside the root does not necessarily condemn the case to postoperative disease. Rather, it is the residual presence of any infected tissue that determines the prognosis. The closer the root canal preparation is to the terminus at the time of instrument separation, the better the outcome will be.[339] It has been suggested that instrument separation occurring in a later stage of canal instrumentation, especially if it is at the apex, has the best prognosis because the canal is probably well debrided and may be relatively free from microorganisms.[338] If the preoperative canal is not infected, and no radicular periodontitis is associated with the root, then the presence of the separated instrument should not affect the prognosis[63] and the separated instrument can be incorporated into root canal filling materials. If the separated file can be removed predictably without sacrificing a significant amount of dentin, the prognosis will not be reduced. In other words, the treatment prognosis depends more on the removal of microbial infection than on instrument removal.

Ledge formation

Ledges may be created as a result of instrumentation causing radicular transportation, which also causes zipping of the canal and possible canal perforation (Fig. 20.22). The *Glossary of Endodontic Terms* of the American Association of Endodontists[10] defines a ledge as “an artificial irregularity created on the surface of the root canal wall that impedes the placement of an instrument to the apex of an otherwise patent canal.” Canal transportation[10] is defined as “the removal of canal wall structure on the outside curve in the radicular half of the canal due to the tendency of files to restore themselves to their original linear shape during canal preparation.” The presence of a ledge hinders adequate shaping and cleaning of the canal in the areas radicular to the ledge. Consequently, there is a possible causal relationship between ledge formation and an unfavorable endodontic treatment outcome.[61,62,118,125,133,159,215,377]

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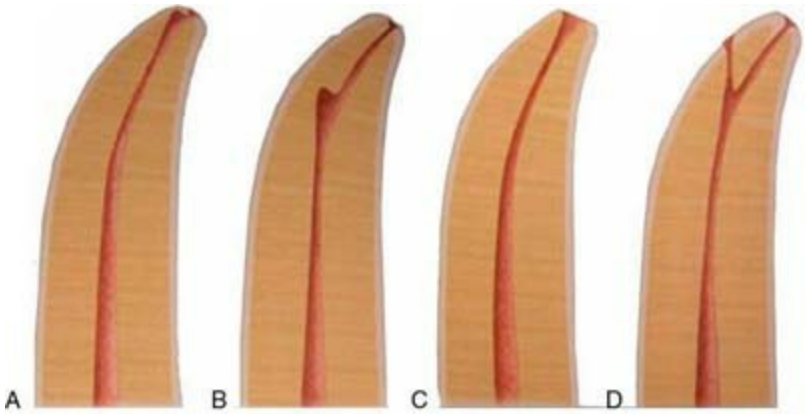

FIG. 20.22 Schematic diagrams showing the most common preparation errors. **A,** Radicular zip. **B,** Ledge. **C,** Radicular zip with perforation. **D,** Ledge with perforation.

A) Root has a curved root canal with flattened tip.

B) Root canal has a protuberance near the apex.

C) Root canal has wide apex.

D) Root canal has an accessory branch near the apex.

Causes of ledge formation

Ledge formation can occur during biomechanical preparation of the canal system, especially when the canals are more curved. There are a number of factors associated with ledge formation, such as the instrumentation technique, instrument type, root canal curvature, tooth type, working length, master radicular file size, clinician's level of expertise, and canal location. Failure to precurve the instruments, the inability to achieve a proper glide path to the apex, and forcing large files into curved canals are perhaps the most common reasons for ledge formation.[153] The incidence of ledge formation was reported to increase significantly when the curvature of the canal was greater than 20 degrees; more than 50% of the canals in the study were ledged when the canal curvature was greater than 30 degrees.[118] Jafarzadeh[156] described 14 possible causes of ledge formation (Box 20.1).

Box 20.1

14 Possible Causes of Ledge Formation

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1. Not extending the access cavity sufficiently to allow adequate access to the radicular part of the root canal[61,125,153,159,185,352]
2. Loss of instrument control if endodontic treatment is attempted via a proximal surface cavity or through a proximal restoration[153]
3. Incorrect assessment of the root canal direction[61,159,185]
4. Incorrect root canal length determination[61,159,185]
5. Forcing the instrument into the canal wall[356]
6. Using a noncurved stainless steel instrument that is too large for a curved canal[61,153,159,250,352]
7. Failing to use the instruments in sequential order[61,125,159,185]
8. Rotating the file excessively at the working length[125,356]
9. Inadequate irrigation or lubrication during instrumentation[352]
10. Over-relying on chelating agents[356]
11. Attempting to retrieve separated instruments[61,159,185]
12. Removing root filling materials during endodontic retreatment[61,159,185]
13. Attempting to negotiate calcified root canals[61,159,185]
14. Inadvertently packing debris in the radicular portion of the canal during instrumentation[61,352]

Management of ledge formation

Once a canal is ledged, the endodontic treatment becomes difficult and the prognosis may be compromised. When a ledge is formed, early recognition of its location, using radiographs including CBCT and magnification, will facilitate its management.[125] The canal is usually “straightened” at the point where the file stops negotiating the curve, and there is suddenly a looser feeling of the file within the canal, with no more tactile sensation of the tip of the instrument binding in the canal. If the radiograph reveals that the tip of the instrument deviates away from the canal curvature, then it is highly likely that a ledge formation has occurred on the canal wall. The tip of the instrument placed in the canal should appear at the midpoint of the root on the radiographs because the canal is always in the center of the single-canaled root. If there are more than two canals in one root, then it is natural that the instrument placed in the canal appears off-center, which makes it essential to refer to CBCT for the number of the canals in the root before the

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instrumentation is initiated (Fig. 20.23).

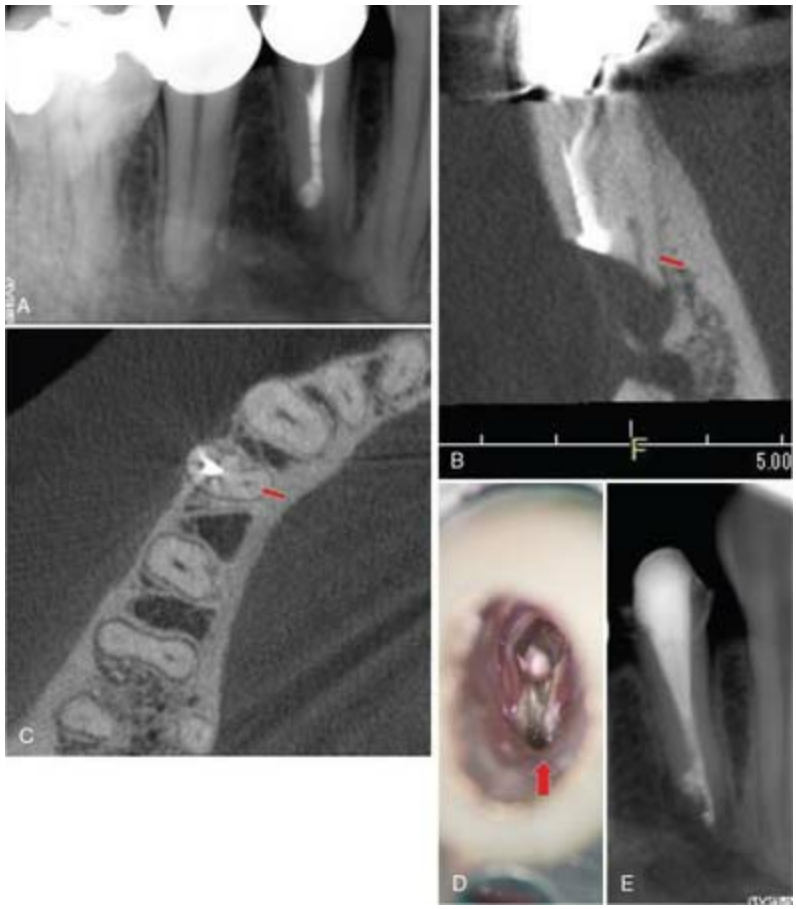

FIG. 20.23 **A,** Preoperative radiograph showing the failing outcome of endodontic surgery for the mandibular right first premolar with the root canal filling in one root canal. **B,** Cone-beam computed tomography (CBCT) image in coronal view showing a missed lingual canal *(red arrow)*. **C,** CBCT image in axial view showing the missed lingual canal lingual *(red arrow)* to the mesiodistal midline in relation to the buccal canal buccal to the midline with the same distance between the midline and the lingual canal. **D,** Magnification of the missed lingual canal *(red arrow)*. **E,** Postoperative radiograph showing root canal obturation in both the buccal and the lingual root canals.

Three radiographs and a photograph marked A through D show the radiolucent patches and cavity in lingual canal of first premolar, respectively. Radiograph marked E shows the filling in both buccal and lingual canals.

When a ledge formation is identified, the shortest file possible that can reach the working length should be selected to bypass the ledge. Shorter

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instruments have more stiffness and allow the clinician's fingers to be placed closer to the tip of the instrument, which consequently provides greater tactile sensation and more control over the instrument.[156] It usually requires determination, perseverance, and patience to bypass a ledge once it is formed.[61] The successful management of a ledge depends on whether both the ledge and the orifice of the original pathway can be visualized, reached, or felt with instruments.[191] Any case where the ledge can be visualized under the DOM may be considered less complex to treat. Cases in which the ledge is not directly visible are more difficult to treat, as tactile sensation and negotiation require patience and experience.[61] However, 3D radiographic images obtained from CBCT reveal the locations and the directions of both the ledge and the original pathway to the canal terminus as the ledge always forms on the outer wall of the curved root canal in the straight line direction from the access. CBCT helps the clinician locate the entrance of the original pathway to the radicular foramen. First the ledge needs to be recognized under the DOM and both the ledge and the original canal need to be located on the 3D radiographic images. The maximum curvature of the root apical to the ledge can be identified on the axial view of CBCT as it is exactly the same as what is seen under the DOM. The maximum curvature can be located simply by viewing the axial slices from the coronal aspect before the ledge to the radicular aspect past the ledge in parallel with the coronal straight line to the ledge, and then a sagittal slice or a coronal slice is cut over the center of the curved root on the axial slice with the maximum curvature. On the sagittal view or the coronal view, the relationship between the ledge and the original pathway can be recognized, and with this view, the entrance to the original canal can be measured to locate its position under the DOM in reference to the ledge and the orifice from several reference points to it as the original canal is always in the center of the single-canaled root and the ledge is always in the straight line. CBCT without doubt, compared to 2D radiography, provides additional information for making an accurate diagnosis and therefore helps the clinician manage the ledge formation with a more predictable outcome and negotiate the original canal.[232,331]

Bypassing a ledge

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Using hand instruments

Based on the information from CBCT, the original pathway can be predictably identified and opened up under the DOM using micro-hand instruments. In addition, the ledge can always be visualized under the DOM as it is created in the straight-line direction. In addition, caries detector dye (Kuraray America, Inc., NY) or methylene blue dye can stain the orifice of the original pathway to locate it in comparison with dentin wall. Small hand files dedicated to microscopic use (such as the micro-Opener with #10/.06 taper) may help flare the orifice of the original pathway on the inner wall of the root canal coronal to the ledge (Fig. 20.24, *A*). The micro-Opener is available only in 16 mm and is short enough to provide tactile sensation and better control. However, in some cases a longer instrument is required to reach the ledge present in the radicular third of a long canal. In these instances, a hand instrument holder such as an EndoHandle (Venta, Logan, UT) may help the clinician easily negotiate the canal radicular to the ledge. Any hand files can be attached to the handle for microscopic use. Other hand instruments are available specifically for this purpose (TFRK microexplorer instrument [TFRK-ME] or Gutta-Percha Removal Instrument [TFRK-GPR, DELabs] and ELES instrument [DenMat Lompoc, CA]) that are long enough to reach the ledge formed in the radicular third. Both instruments are made of stainless steel. The TFRK-ME has a 0.1 mm biconical tip with a.08 taper for the first 1-mm portion from the radicular end and a.06 taper for the next 10-mm portion, which gives the instrument more stability in push-pull motions than a.02 tapered K-file. The TFRK-ME is precurved to the original pathway in the radicular 2 to 3 mm and pecked with to explore the narrow original canal on the opposite side of the canal wall with the ledge (Fig. 20.25), while the ELES and the TFRK-GPR are used to reduce the ledge in push-pull motions as the 0.2 mm diameter tip portion of the ELES with a.06 taper is coated with fine diamond particles on the tip portion (Fig. 20.26) and the TFRK-GPR has a sharp conical tip. They can be used in sequence to widen the orifice of the original pathway, which results in reducing or removing the ledge. The maximum diameter of the TFRK-ME and the TFRK-GPR is 0.78 mm, which provides stability and maintains a high visibility under the DOM when it is precurved, whereas the maximum diameter of the ELES instrument is 0.68 mm, which provides more flexibility and more visibility, even around

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a curve, to facilitate the widening the orifice of the original pathway without worsening the ledge. Once the first TFRK-ME or any other micro-hand file has successfully bypassed the ledge, subsequently the ELES or the TFRK-GPR instrument or the same micro-instrument can be used in a push-pull motion to enlarge and reestablish the original pathway (Fig. 20.27), followed by hand instruments (such as ProTaper S1 and S2 hand instruments, DENTSPLY Sirona Tulsa Dental Specialties, Tulsa, OK) to both shape and reduce the ledge. S1 and S2 ProTaper Gold files (DENTSPLY Sirona Tulsa Dental Specialties) can also be precurved with ease and used as hand files in short pumping motions for the same purposes after the preliminary enlargement of the original pathway with those micro-hand instruments (Fig. 20.28).

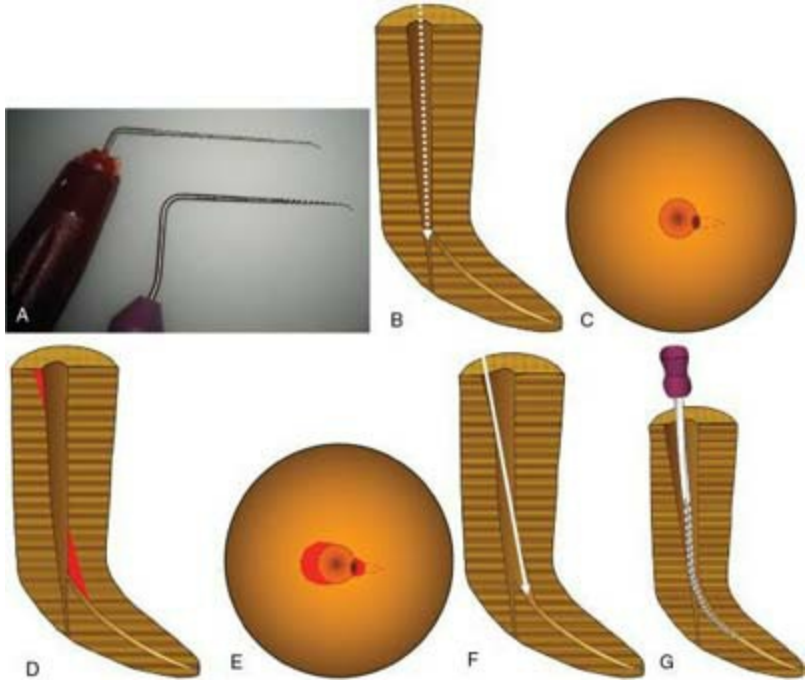

FIG. 20.24 **A,** Precurved microinstruments such as EndoHandle with a precurved #25 Hedstrom File (Venta Endo, Logan, UT) and micro-Opener (Dentsply Sirona) to explore a ledged canal for the original pathway and to reduce irregularities over the ledge. **B,** Ledge is formed with perforation in a radicular straight direction. **C,** Microscopic view showing the ledged canal in the center and a narrow access to the original pathway on the right side. **D,** The canal orifice as well as the access to the original pathway needs to be flared in an anticurvature direction *(red portions)*. **E,** Microscopic view showing red portions to be removed for easy access to the original pathway. **F,** Access to the

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original pathway was flared to facilitate file insertion. **G,** Bypassing file used in short push-pull strokes to create a distinct pathway to the original canal as the final glide path.

A) Photograph of instrument with a curved and sharp file.

B) A dashed arrow points toward the accessory canal near the root apex.

C) Circular canal has a brown spot on the left with a dashed cone.

D) Root canal has red portion along the side of root canal opposite to the accessory canal.

E) Circular canal has a brown spot, followed by a red portion and a dashed cone on the left.

F) A straight arrow points toward the root canal near the bifurcation area.

G) Endohandle reaches near the root apex.

FIG. 20.25 **A,** Micro-hand instrument (TFRK-ME) to explore canals for the original pathway and bypass a ledged canal. **B,** Magnified image of a TFRK-ME.

Two photographs marked A and B show the full view of TFRK-ME with curved needles in both ends and partial magnified view of TFRK-ME with a curved needle on one end, respectively.

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FIG. 20.26 Micro-hand instrument with diamond coating *(ELES)* to bypass/eliminate/reduce a ledge and enlarge the orifice of the original pathway.

A TFRK-ME with ELES coating at the center and curved needles on both end on a white background is shown.

FIG. 20.27 Schematic diagrams showing bypassing the ledge with ledge removal hand instruments. **A,** Ledge is created in a radicular

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straight direction around a curve. **B,** Original pathway is explored and opened up with the TFRK-ME in short push-pull strokes. **C,** Original pathway is widened with the ELES instrument. **D,** ELES instrument used in push-pull motions to reduce the ledge. **E,** Precurved TFRK-GPR is placed into the original canal and moved with short up and down strokes over the ledge. **F,** Ledge was reduced with the precurved TFRK-GPR. **G,** Precurved straight ultrasonic tip can also be used here to reduce the ledge instead of the ELES instrument. **H,** File with a biconical tip is brought into the canal to eliminate/reduce the ledge and negotiate the original canal. The primary cone of the biconical tip has no cutting tip. As it hits the curve or irregularities on the canal wall, it will slip alongside the curve and scoot over the irregularities. **I,** Secondary cone of the biconical tip removes the points at the transition angles and provides a smooth surface that guides the tip portion into the original pathway. **J,** Orifice of the original canal over the ledge needs to be enlarged for easy file insertion after the ledge has been bypassed successfully. **K,** Orifice of the original canal can be efficiently widened with a precurved instrument such as a ProTaper Gold S1 rotary file and a #15/.04 Vortex Blue rotary file used as a hand file, and the apical patency established. **L,** Original canal can be sequentially prepared with larger NiTi files. **M,** Postoperative canal showing the ledge is significantly reduced.

Set of thirteen illustrations marked A through M shows the changes in ledge canal by using TFRK-GPR.

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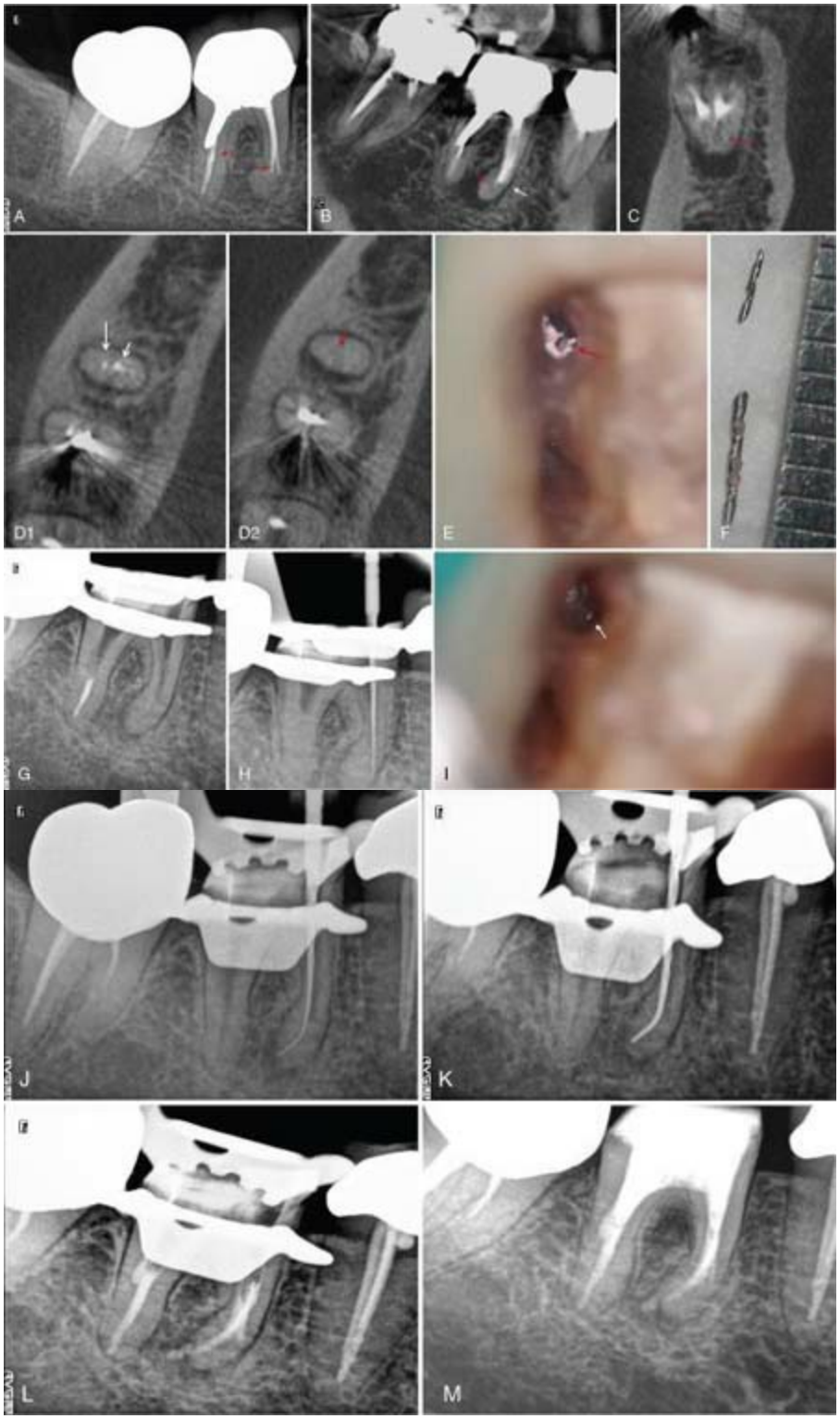

FIG. 20.28 **A,** Preoperative radiograph of a mandibular right first molar showing a periradicular lesion associated with the mesial root and two separated files in the mesial and the distal root *(red arrows)*. Note there are two separated files in the mesial and the distal roots in addition to the fact that the root filling in the mesial root deviates away from the

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center of the root. **B,** Cone-beam computed tomography (CBCT) image in sagittal view showing the original canal *(red arrow)* in the center of the root while the ledge *(white arrow)* turns away in the straight line in the mesial root. **C,** CBCT image in coronal view showing the MB and the ML canals merge into one canal *(red arrow)* in the apical third. **D1** and **D2,** Consecutive CBCT images in axial view showing the MB canal and the ML canal *(white arrows)* join into one oval canal *(red arrow)*. **E,** Magnification of the MB canal showing a separated file *(red arrow)*. **F,** Retrieved separated files from the mesial root and the distal root. **G,** Interoperative radiograph showing removal of the two separated files. **H,** Placement of a small file into the MB canal reveals a perforation on the outer wall in the straight line form from the orifice on the interoperative radiograph. **I,** Ledge *(red arrow)* and the original pathway *(white arrow)* were identified in the MB canal under the DOM based on the axial view of CBCT and the orifice of the original canal was enlarged with a precurved #10/.06 micro-opener with short push-pull strokes. **J,** Placement of a precurved small hand file (ProTaper S1) reveals it in the original canal reaching the working length on the interoperative radiograph. **K,** Placement of the final shaping file into the MB canal confirms it in the original canal reaching the working length without transportation on the interoperative radiograph. **L,** Postoperative radiograph shows root canal fillings with mineral trioxide aggregate (MTA) including the ledged canal with the perforation. **M,** Three-month postoperative radiograph shows a dissolving periapical lesion.

Set of eight radiographs and two blurred photographs marked A through I shows the removal of separated files from mesial and distal roots of first molar in the lower jaw, followed by the formation of ledge canal along with original pathway in the root near the apex.

Set of four radiographs marked J through M shows the filling of root canals along with ledge in first molar of lower jaw and development of hard tissue in place of the radiolucent patch at the root apex.

As an alternative technique to bypass the ledge when the clinician wishes to use K files without using the DOM, precurving of a #8 or #10 hand file is a critical step. The small file should have a distinct curve at the tip. This file should be used initially to negotiate and explore the original pathway of the canal to the radicular foramen.[61,153,352] A rubber stopper with a directional indicator pointed in the direction of the original pathway should be set in the instrument.[153] A slight rotation of the file combined with a “pecking” motion can often help advance the instrument to the full working length of the canal.[153,352] If this technique is unsuccessful, the clinician should flare the

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canal coronal to the ledge in an anticurvature direction to obtain wider straight-line access to the original pathway of the canal, followed by a smaller K-file with the tip precurved to bypass the ledge (see Fig. 20.24).

Using ultrasonic tips

Small diameter ultrasonic tips improve both cutting efficiency and visibility with the added benefit of reducing the need for sacrificing sound dentin in bypassing or removing the ledge.[192] The only drawback to the thinner tips could be that they are more prone to fracture during ultrasonic activation.[351] Therefore thinner ultrasonic tips should be activated in a pulsing and pecking fashion at the lowest possible power setting to prevent fracture, overextending the ledge, and heat generation as ultrasonics may get very aggressive in cutting.[282]

The use of longer ultrasonic tips may also facilitate bypassing or reducing the ledge in the radicular third as long as it is performed under the DOM. The entrance to the original canal just coronal to the ledge can be quickly enlarged with small diameter ultrasonic tips if it can be visualized under the DOM or felt with a sticky feeling from the micro-hand instruments (Fig. 20.29). First, an appropriate size of a small diameter ultrasonic tip (such as TFRK-S, DELabs and ET25, Satelec Corp, Merignac Cedex, France) is selected and precurved in the direction of the original canal. Then it is simply placed into the orifice of the original pathway in a radicular direction and activated in pecking and short push-pull motions at the same time, which will automatically enlarge the opening of the true canal in a few seconds so that files can consecutively be placed into the original canal for root canal preparation. Once the orifice of the original pathway gets wide enough for file insertion, negotiation files need to be placed into the canal to establish patency and to smooth out the irregularities on the canal wall that may have been created with ultrasonics, followed by root canal preparation with NiTi rotary or hand files (Fig. 20.30).

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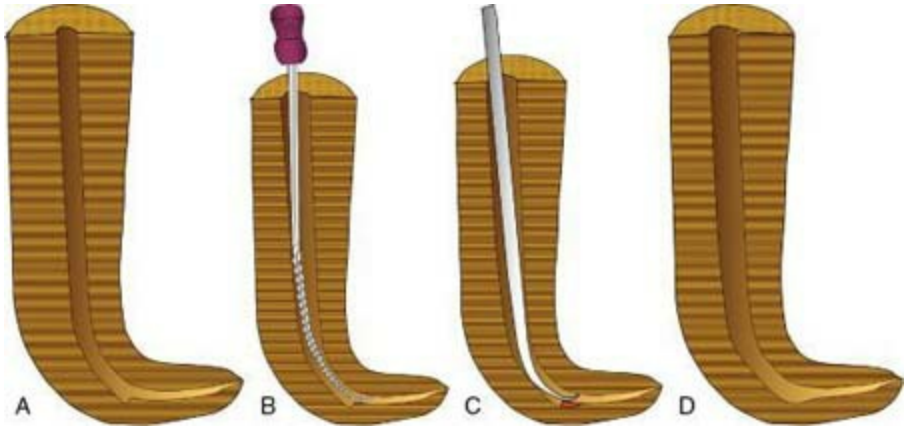

FIG. 20.29 Nonvisible ledge removal with ultrasonics. **A,** Ledge formed beyond the curve. Cone-beam computed tomography (CBCT) is taken to locate both the ledge and the original pathway. **B,** Canal coronal to the ledge is flared in an anticurvature direction with NiTi rotary files in brushing motions to possibly visualize the ledge and the original pathway. Original pathway to the canal terminus is explored with a #10 precurved K-file or the precurved TFRK-ME for a sticky feeling and then radiographs are taken with it inserted in the canal to see if it is in the original canal beyond the ledge. **C,** Straight ultrasonic tip is precurved the same way as the hand instrument used and placed into the original canal to eliminate/reduce the ledge and to open up the entrance to the original pathway. The red portion needs to be smoothed out to facilitate the insertion of the next larger file into the original pathway. **D,** Postoperative canal showing the ledge is significantly reduced.

Set of four illustrations A through D shows the reduction of ledge in size TFRK-ME and ultrasonic tip.

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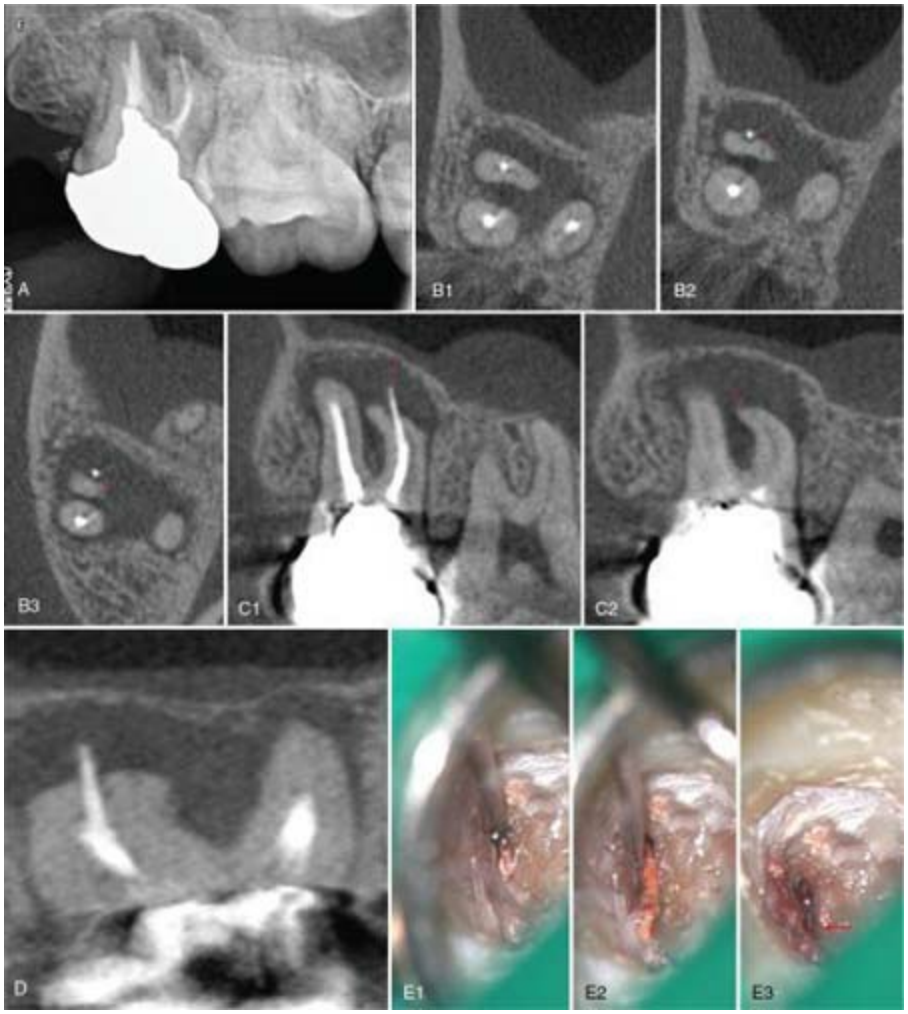

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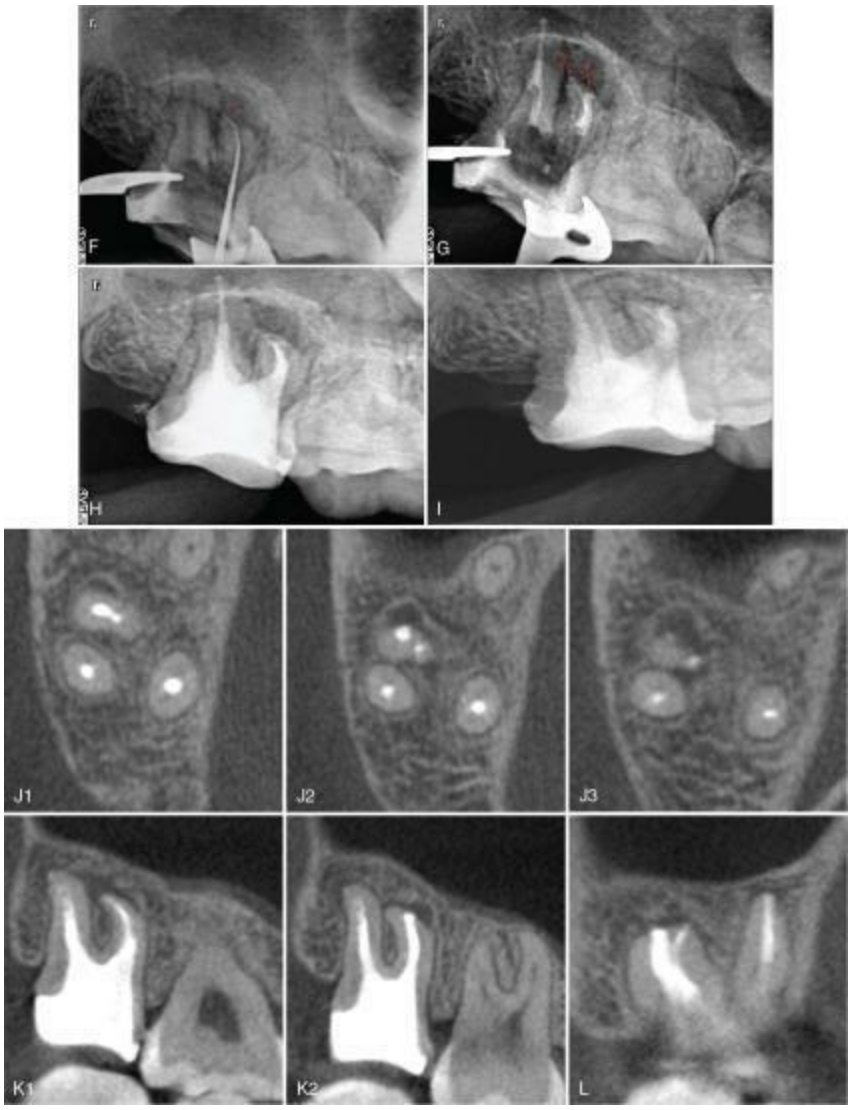

FIG. 20.30 **A,** Preoperative radiograph of a maxillary right second molar showing periradicular lesions. Note the root filling in MB is overextended beyond the radicular foramen. **B,** A series of cone-beam computed tomography (CBCT) images in axial view from the apical third to the radicular foramen (**B1** to **B3**) reveals an oval MB canal turning round toward the radicular foramen and the original pathway *(red arrows)* curves in a distopalatal direction with the root filling turning away from the original canal (off-center). **C1** and **C2,** A pair of CBCT images in sagittal view clearly show both the overextended root filling (*red arrow* in **C1**) and the presence of the distally curved original canal (*red arrow* in **C2**). **D,** CBCT image in coronal view also shows a large lesion associated with the MB canal, with the root filling extruded beyond the radicular foramen (perforation). **E1 to E3,** A series of microscopic images shows the overextended root fillings being removed with the TFRK-GPR (**E1** and **E2**) and the original canal (**E3,** *red arrow*) was located in the MB canal and enlarged with ultrasonics. **F,** Original canal was negotiated and then the apical patency was

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established with a precurved micro-opener followed by a precurved ProTaper S1. **G,** Postoperative radiograph showing the completed root fillings including the original canal and the ledged canal with perforation *(red arrows)*. **H,** Three-month follow-up showing healing in progress. **I,** Six-month follow-up showing nice periapical healing. **J,** A series of 6-month postoperative CBCT images in axial view from the apical third to the radicular foramen (**J1** to **J3**) reveals root fillings in both the original canal and the ledged canal with nice periapical healing. **K,** A pair of 6-month postoperative CBCT images in sagittal view (**K1** and **K2**) shows nice periapical healing compared to the preoperative CBCT images. **L,** Six-month CBCT image in coronal view also shows nice periapical healing.

Set of seven radiographs and three photographs marked A through E 3 shows a radiolucent patch at the root apex and the filled root canals extended beyond the apex in second molar of the upper jaw, which undergo removal and resulting in the restoration of original canal without filling.

Set of ten radiographs marked F through L shows the fillings in root canals including ledge resulting in thee healing of radiolucent patches at the root apex of second molar in the upper jaw.

Using rotary instruments

Before using rotary instruments to bypass the ledge, the canal should be enlarged to the area coronal to the ledge for better visualization of the original pathway and then the orifice of the original pathway must be located and opened. Precurved smaller diameter rotary files with smaller tapers such as PathFiles (Dentsply Sirona, Ballaigues, Switzerland) and Race 10 (FKG Dentaire, La Chaux-de-Fond, Switzerland) are also beneficial for negotiating beyond a ledge. First, the radicular 2 to 3 mm of the file is precurved to the original pathway based on the coronal or the sagittal view of CBCT and is rotated at 100 rpm or with 90° CW/30° to 60° CCW reciprocating motions at 150 rpm in short up-and-down motions as it is brought slowly into the canal and moved radicularly (Fig. 20.31). If a ledge or sharp radicular curvature is encountered, the instrument is withdrawn about 1 mm and immediately reinserted. This allows the precurved tip to move to a different orientation within the canal as it is reinserted. This may require one or two withdrawals and re-insertions to negotiating the original canal. This procedure is followed in sequence by the next larger rotary files to preliminarily enlarge the original pathway and reduce the ledge prior to the conventional root canal preparation

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with NiTi rotary instruments.

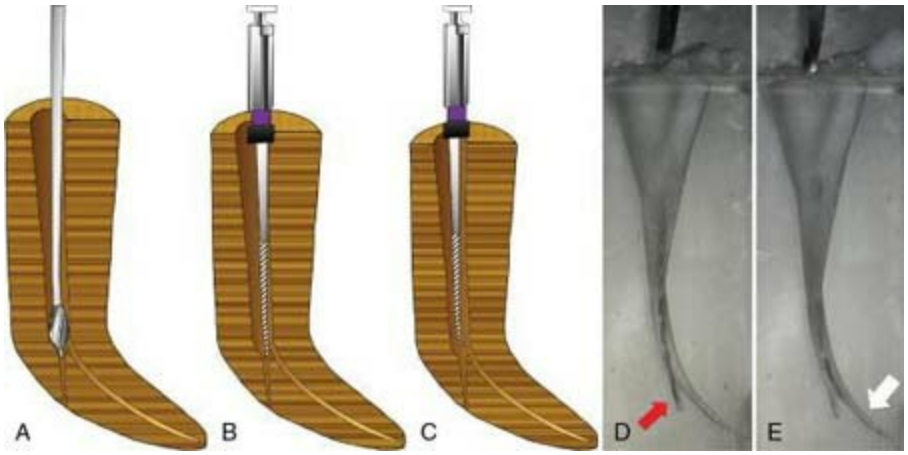

FIG. 20.31 **A,** Canal coronal to the ledge is enlarged with a #3 GG bur. **B,** Small straight rotary file (#10/.02 or #10/.04) rotating at 300 RPM or faster will not get into the original pathway. **C,** Small precurved rotary file rotating at 100 RPM or 150 RPM with 90-degree clockwise and 30-degree counterclockwise reciprocating motions will most likely get into the original pathway. **D,** Small precurved rotary file rotating at 300 RPM ended up going into the ledged canal *(red arrow)*. **E,** Small precurved rotary file rotating at 100 RPM was able to get into the original canal *(white arrow)*.

Set of three illustrations and two radiographs marked A through E shows 3 GG bur and rotatory files inserted into either ledged canal or original canal of the root.

Potential complications of removal or bypassing a ledge

During ledge removal or bypassing a ledge, complications can occur, including root fracture, root perforation, worsening the ledge, and instrument separation. Since a ledge is likely to form around a curve, attempts to bypass or eliminate it may often involve dentin sacrifice, which may eventually compromise the integrity of the tooth and increase the risk of perforation or root fracture. Care should be taken not to remove excessive root structure or overstress the instruments during the ledge management. If a ledge cannot be bypassed with any of the techniques described earlier, and if signs or symptoms of the periradicular lesion persist, then the treatment options are limited to either the use of GentleWave, which applies advanced fluid

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dynamics, acoustics, and tissue dissolution chemistry to clean and disinfect the entire root canal system, regardless of any anatomic complexities (including the original pathway beyond the ledge), leading to success in eliminating infection from the canal system without shaping them where traditional instruments cannot access[301,302,349] or surgical intervention such as periradicular surgery or intentional replantation.[153]

Prevention of ledge formation

Glide path preparation helps clinicians prevent mishaps such as ledge formation and canal transportation as it guarantees that the root canal diameter is sufficiently large to receive the first shaping instrument.[27,234] Precurving the instruments and not forcing them into the canal is one of the most important considerations in the prevention of ledge formation.[153]

NiTi reciprocating instrumentation shows a lower frequency of procedural errors such as ledge formation and instrument separation even in the hands of novice clinicians, compared to K-file/NiTi rotary instrumentation.[73,128,202,283,328] The use of NiTi files and instruments with noncutting tips helps to maintain original root canal curvatures.[153] The concept of use of these instruments is that the rounded tip does not cut into the wall but will slip alongside it.[152,153] In addition to the tip design, flexibility of an instrument also plays an important role in keeping the instrument centered in the canal, with NiTi instruments favored over stainless steel instruments.[143,150,223] The use of novel NiTi alloy instruments with new heat treatment methods such as the Controlled Memory alloy technology (Coltene/Whaledent AG) and the GOLD alloy technology (Dentsply Maillefer, Ballaigues, Switzerland) show less canal transportation and more canal centering ability than conventional NiTi alloy instruments.[135,227,244,348]

Prognosis

A ledge in a canal is analogous to any intracanal obstruction that hinders the cleaning and shaping of the root canal to the radicular terminus. The presence of a nonnegotiated ledge may have a similar prognosis as a canal with a retained separated instrument. If the ledge can be bypassed or eliminated, and if the canal beyond the ledge can be cleaned without excessive enlargement

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or perforation, then the prognosis should not be that greatly reduced.[338,339] Therefore the prognosis in general for such a case depends not only on the efficacy of the disinfection process but also on the host response to remaining debris or biofilms (bacteria) that are traditionally not removed with conventional root canal therapy (Fig. 20.32).[74]

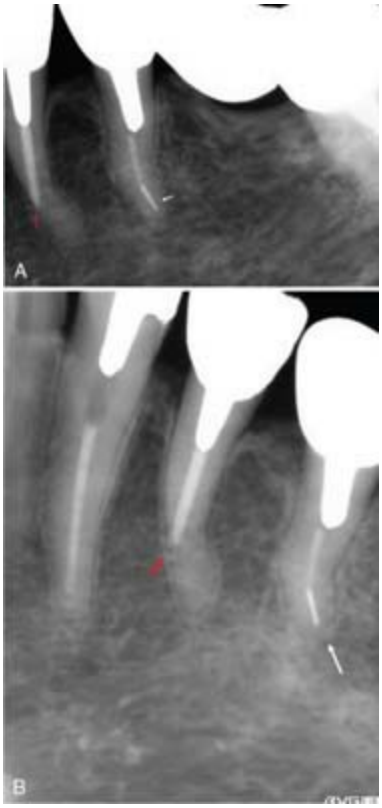

FIG. 20.32 **A,** Radiograph taken in 2010 for evaluation of the ledge *(red arrow)* in the mandibular left first premolar and the separated file *(white arrow)* in the mandibular left second premolar showing no radiographic periapical pathosis and both of the teeth were asymptomatic. **B,** Radiograph taken in 2019 for evaluation of those teeth showing the same without any sign of periapical pathosis *(red and white arrows)*.

Set of two radiographs marked A and B shows ledge canal and separated file in two different teeth of lower jaw.

Radicular extrusion of root canal filling

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materials

The extrusion of root filling materials beyond the radicular foramen or the perforation site may occur either when the canal is obturated or during the removal of obturation materials. Radicularly extruded materials such as obturating materials, necrotic pulp tissue, bacteria, medicaments, and irrigants have been associated with posttreatment inflammation and flare-up or the lack of radicular healing.[213,295,305,335] In vivo histologic studies show the most favorable histologic outcome when the root filling material remains within the radicular constriction. When gutta-percha is extruded into the periradicular tissue, there can be a severe inflammatory reaction.[265] However, other types of materials, such as ProRoot mineral trioxide aggregate (MTA) (DENTSPLY Sirona Tulsa Dental Specialties) and EndoSequence BC Sealer (Brasseler, Savannah, GA), are more biocompatible[1,57,111,117,167] and do not seem to produce much periradicular inflammation if extruded beyond the root of the tooth.[93,312]

Studies show that almost all instrumentation techniques or types of NiTi instruments promote the radicular extrusion of debris including root filling materials.[7,144,329,336] Furthermore, gutta-percha and Resilon filling materials obtained from teeth associated with refractory radicular periodontitis have indicated the presence of bacterial biofilm and *E. faecalis*.[122,214,223,296] Another study stressed the possibility of gutta-percha–centered *E. faecalis* biofilm as well as the role of tissue fluids such as saliva and serum in the biofilm formation.[108] *E. faecalis* adheres well to gutta-percha with and without canal sealers, with the biofilm formation more effective when it is in contact with serum in periradicular tissues. Because bacterial biofilm is considered the primary cause of chronic and recurrent endodontic infections,[341] it is crucial to keep gutta-percha filling materials within the canal system as much as possible to both prevent potential periradicular periodontitis and obtain the optimal results.[26,83,289]

Uncontrolled obturation can cause the extrusion of root canal filling materials into the maxillary sinus[86] or into the territory of the inferior alveolar nerve (IAN).[246] The systematic review shows most of the nerve injury cases with lateral condensation fully recovered over time, compared with none of the cases with vertical condensation that recovered.[273] Serious

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complications such as maxillary sinusitis, aspergillosis infection, paresthesia, dysesthesia, and similar neural complications may occur after extruding the root filling materials including a core carrier of an obturator into such sensitive areas.[30,173,203]

Endodontic-related paresthesia can be caused mechanically, thermally, chemically, or a combination of these via the passage of endodontic materials into the vicinity of the major nerves such as the IAN or its branches.[3,249] When the IAN is injured, the pain is not only located in the immediate periradicular area but may radiate and refer to the surrounding or distant structures.[222] Inflammation caused by mechanical irritation is likely to persist until the extruded materials are removed, and it may become permanent secondary to the mechanical, thermal, or chemical insult.[136] These extruded materials include root canal irrigants, sealers, and paraformaldehyde-containing pastes.[350] Most extrusion cases with resin-based sealers showed full recovery from nerve injuries (62%), but only 27% of those with paraformaldehyde based sealers showed full recovery.[273] The use of paraformaldehyde-containing pastes is not recommended for endodontic obturation[210,249] because paraformaldehyde is a polymeric hydrate of formaldehyde that releases formaldehyde gas and may cause permanent damage to the nerve when it is in contact with water.[114] On the other hand, extrusion of endodontic materials, even into the inferior alveolar canal, will not always result in altered sensation.[246] The possible explanation for this is that some extruded root canal sealants demonstrate their neurotoxic properties only when they come into direct contact with the individual fascicles through the pathway created by over-instrumentation, whereas they are less harmful to the sensory nerves when they are outside the epineurium.

Significantly higher or lower pH materials than those of body fluids (around 7.35) are also likely to cause cellular necrosis of tissues when they are in direct contact with these materials. The clinician must also consider the pH of some of the routinely used endodontic and related dental materials. It has been reported that an alkaline pH of root canal sealers could neutralize the lactic acid from osteoclasts and prevent dissolution of mineralized components of teeth; therefore root canal sealers, especially calcium silicate–based cements or sealers, can contribute to hard tissue formation by activating alkaline phosphatase.[321] Commonly used endodontic medicaments

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are as follows[47,105,245,258,303]:

Formocresol: pH 12.45 +/− 0.02
NaOCl: pH 11 to 12
Calcium hydroxide (Calyxl): pH 10 to 14
ProRoot MTA: pH 11.7 to 7.12 at 3 hours to 28 days
Endosequence BC Sealer: pH 10.31 to 11.16 at 3 hours to 240 hours
MTA Fillapex: pH 9.68 to 7.76 at 3 hours to 168 hours
AH plus: pH 7.81 to 7.17 at 3 hours to 240 hours
Sealapex: pH 9.72 to 9.63 at 3 hours to 24 hours
Antibiotic-corticosteroid paste (Ledermix): pH 8.13
Pulp Canal Sealer: 8.07 to 7.55 at 3 hours to 24 hours
Eugenol: pH 4.34 +/−0.05
Iodoform paste: pH 2.90 +/−0.02

Neural damage from those higher or lower pH materials may be permanent and can cause neuropathic pain (i.e., dysesthesia) or anesthesia.

Orbital pain and headache may occur secondary to the local compression of overextended material into the maxillary sinus.[365] Sinus infection caused by zinc-oxide-eugenol–based filling materials extruded into the maxillary sinus is often associated with aspergillus growth and biofilms (Fig. 20.33).[21,22,112,175,274] Although aspergillosis of the paranasal sinuses is a relatively rare finding in non-immunocompromised patients, it is known as an opportunistic infection.[166] Many studies have demonstrated that maxillary sinus aspergillosis is generally caused by extruded root canal filling material containing zinc oxide and formaldehyde.[21,22,175,188,319] However, even those extruded materials that are not considered toxic[315] may cause an additional foreign body reaction.

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FIG. 20.33 Maxillary sinus aspergillosis. **A,** Preoperative radiograph showing a lesion associated with the palatal root, from which the extrusion of the root filling materials occurred in the maxillary sinus. **B,** Cone-beam computed tomography (CBCT) image in sagittal view showing the relation between mucositis on the maxillary sinus floor and the gutta-percha filling materials. **C,** CBCT image in axial view showing the relation between mucositis on the mesial wall of the maxillary sinus and the extruded root filling materials. **D,** CBCT image in coronal view showing the relation between the palatal root and the extruded root filling materials. **E,** Postoperative radiograph of orthograde root canal retreatment on the maxillary left first molar showing the mesiobuccal, distobuccal, and palatal root canals were adequately filled without overextension of the filling materials. **F,** Aspergillus was found on the filling materials retrieved from the maxillary sinus.

Set of five radiographs and a specimen marked A through F shows the tooth with maxillary sinus undergoing filling in root canals along with mesial, distal and palatal root, followed by the extraction of filling materials that has aspergillus on it.

The systematic review shows nerve injuries caused by overextruded endodontic materials were reported to be 72% for the first or the second mandibular molars followed by the second mandibular premolars (19%), and none in maxillary teeth.[273]

Causes of extrusion of obturation materials beyond the

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radicular foramen

The extrusion of obturation material that was thought to be the cause of endodontic failure is reported to be as low as 3%,[311] with the majority of causes thought to be more from coronal leakage. However, several researchers have seen a higher frequency of failure and unhealed periradicular lesions in cases where the root filling material had extruded beyond the radicular foramen without filling small anatomical spaces within the root canal system and harboring biofilms on the surface of the extruded material.[26,30,40,239,296] Overextension of the root canal filling materials occurs mainly for lack of material control.[43,126] The degree of extrusion of obturation materials into periradicular tissues depends on the techniques used for root canal preparation and obturation,[266] which include accidental over-instrumentation and incorrect canal working length measurement. Caution should also be taken when obturating a canal with open apices and larger minor foramen diameters, which are often found in cases with radicular inflammatory resorption. Warm vertical compaction techniques produce a homogeneous obturation that adapts well to the canal walls[290,362] but may result in the extrusion of gutta-percha filling into the periradicular tissues and may damage them mechanically and thermally.[43] Carrier-based obturation was also found to have more radicular extrusion of gutta-percha when compared with warm vertical compaction.[184] The type of canal shaping may also be a contributing factor. The canal shape created with the Profile GT system is more prone to extrusion of gutta-percha when obturated with the carrier-based technique than the canal shape created using Profile 0.06 instruments. The Profile GT system created a more parallel preparation coronally and a tapered preparation radicularly, whereas the Profile 0.06 system created a preparation with uniform taper from the apex to its most coronal extent.[266] Therefore possibly the more continuously tapered the canal shape is, the less obturation material is extruded beyond the radicular foramen.

When endodontic retreatment is performed, irritants in the form of root filling materials can be extruded into the periradicular tissues.[7,144,329] Techniques involving push-pull filing strokes usually create a greater mass of debris than those involving some sort of rotational motions.[5,38,94,165,205,254]

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Management of obturation materials extruded beyond the radicular foramen

Nonsurgical management of extrusion of obturation materials

Methods to remove the gutta-percha filling material from the root canal include the use of heat-carrying instruments, ultrasonics, solvents, and rotary or reciprocating or hand files. Mechanized methods to remove filling materials are faster but may possibly be no different from manual methods in terms of the amount of remaining material in the root canal.[129,164,269,270,327] Heat-carrying instrument may damage the periradicular tissues if the removal attempts extend beyond the radicular foramen. The complete removal of gutta-percha from the root canal system has proven difficult.[129,327,368] Poorly compacted gutta-percha obturation can easily be removed in large masses of gutta-percha root fillings with micro-hand instruments under the DOM or Hedström files in combination with solvents such as chloroform,[59,154] although the extruded portion of root canal fillings will often remain in the periradicular tissue (Fig. 20.34).[59] The relative ease of gutta-percha removal suggests a failure of previous endodontic treatment, possibly related to poor obturation compaction or canal shaping, which resulted in both radicular extrusion and failure of the radicular seal. Comparatively, well-filled canals with radicular extrusion do not appear to have a significant failure rate.[15,154] On the other hand, the extrusion of the MTA root filling material into the periradicular tissues does not appear to negatively affect the periradicular healing due to the high biocompatibility and antibacterial properties that the calcium silicate–based cement possesses,[196,326] compared to gutta-percha filling materials (Fig. 20.35). Hence the MTA root filling extruded into periapical tissues does not have to be removed.

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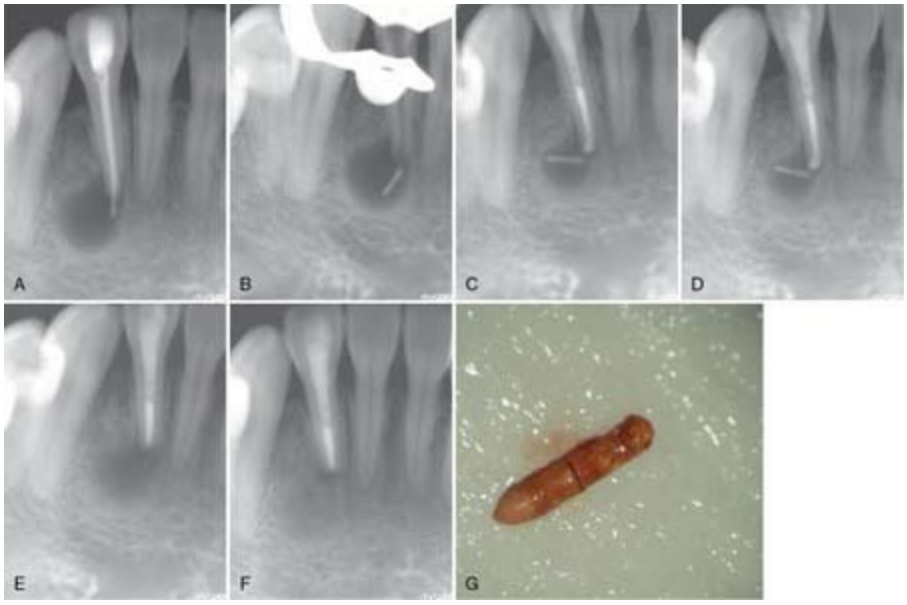

FIG. 20.34 Root filling materials extruded beyond the radicular foramen. **A,** Preoperative radiograph showing overextension of the root filling material with a large lesion associated with the root. **B,** Root filling in the canal was mechanically removed, but the extruded portion of it remained in the periradicular tissues. **C,** Mineral trioxide aggregate (MTA) was filled in the canal with the extruded portion remaining in the lesion. **D,** Six-month postoperative radiograph showing the reduced size of the lesion, but the lesion surrounding the extruded portion stayed the same in size for the next 6 months, indicating infection of the extruded gutta-percha point. The extruded portion was surgically removed from the lesion at this point. **E,** Three-month follow-up radiograph after the surgery showing the resolving lesion. **F,** Twelve-month follow-up radiograph after the surgery showing a more periradicular healing. **G,** Gutta-percha point retrieved from the lesion.

Set of six radiographs marked A through F shows the healing of extended filling along with radiolucent patch in the apical foramen of tooth. Next to it, Specimen G shows extended gutta-percha.

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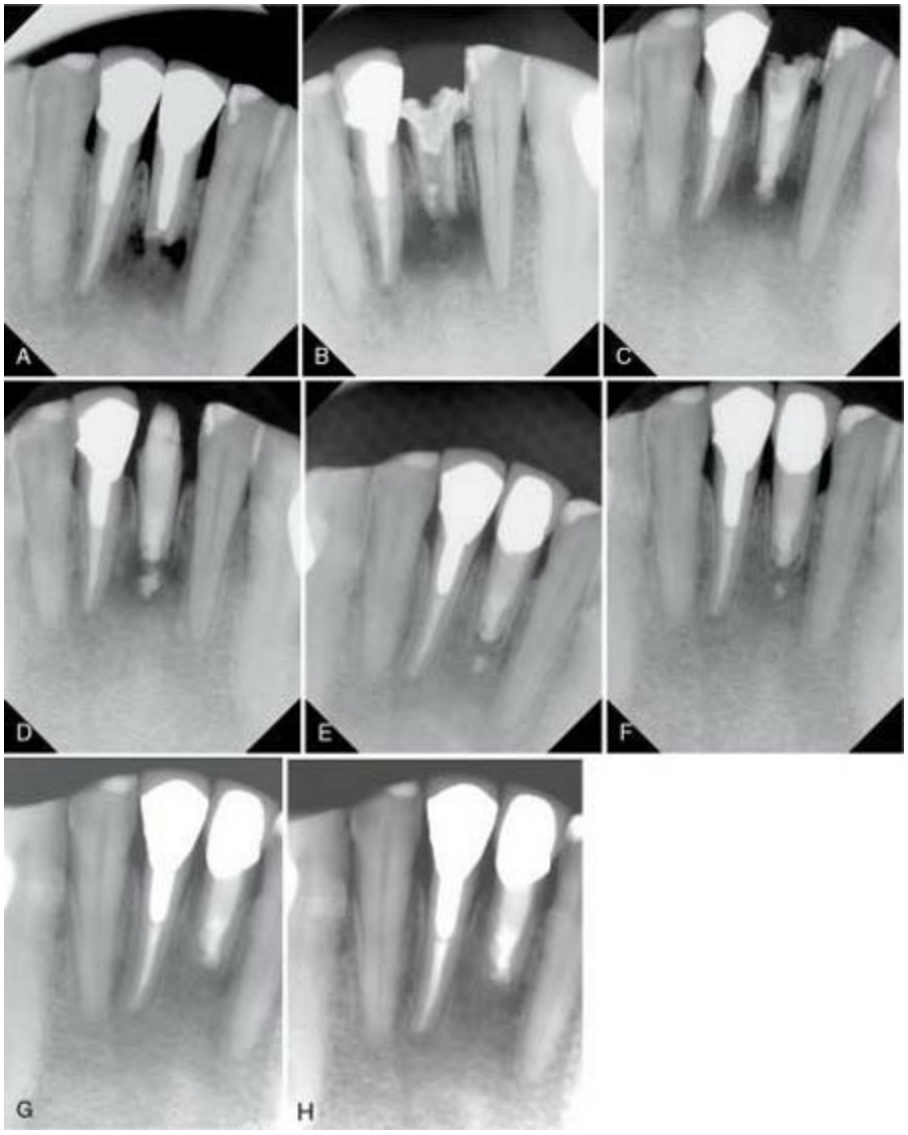

FIG. 20.35 **A,** Preoperative radiograph showing a large periradicular lesion associated with the root of the mandibular left incisor. **B,** Interoperative radiograph showing the cast post and the root filling in the canal was mechanically removed. Calcium hydroxide was placed as an intracanal dressing on the first appointment. **C,** Postoperative radiograph on the second appointment showing the canal was obturated with mineral trioxide aggregate (MTA), which resulted in overextension of the material into the periradicular tissues. **D,** Six-month postoperative radiograph showing the significantly reduced size of the lesion. **E,** One-year postoperative radiograph showing a nice periradicular healing with hard tissues formed on the radicular end of the MTA filling, resulting in a longer root aside from a residual of the MTA root filling extruded into the periradicular tissues. **F,** Three-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with the extruded MTA filling reduced in size. **G,** Twelve-year postoperative radiograph showing the tooth has

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maintained healthy periradicular tissues with the extruded MTA filling completely dissolved in the bone. **H,** Sixteen-year postoperative radiograph showing the tooth has maintained healthy periradicular tissues with healthy lamina dura surrounding the root.

Set of eight radiographs marked A through H shows the restoration of less-density tissue at the root tip of incisor that half root.

When gutta-percha is overextended into the periradicular space, removal can be attempted by inserting a new Hedström file, usually a #15 or #20, into the radicular obturation, using a gentle clockwise rotation to a depth of 0.5 to 1 mm beyond the radicular constriction. The file is then slowly and firmly withdrawn with no rotation, thereby engaging and removing the overextended material.[207] This technique works frequently, but care must be taken not to force the instrument, or further extrusion of the gutta-percha or separation of the file may result. The overextended radicular portion of the gutta-percha should not be softened with heat or solvent, because this application can decrease the likelihood of the Hedström file engaging the obturation, hindering its retrieval.[318] In addition, care should be taken not to overextend the file into the area of the IAN canal, which could cause irreversible damage to the associated nerves.

The use of rotary NiTi files that are specifically designed for gutta-percha removal with active tips and more aggressive cutting may not be superior to conventional files.[348,349] Hand instrumentation may be associated with longer times to remove root filling materials than rotary/reciprocation systems. However, no iatrogenic errors were reported with hand instrumentation.[269,270,271] Hand instruments used for the removal of root fillings include H and K-files, Micro-debriders (DENTSPLY Maillefer, Ballaigues, Switzerland), EGPR-L/R/U/D (Den-Mat Holdings LLC, Lompoc, CA), and the TFRK gutta-percha removal instrument (TFRK-GPR, DELabs). Single-file systems such as the self-adjusting file (SAF) (ReDent-Nova, Ra'anana, Israel), Hyflex EDM "one file" (EDM) (Coltene/Whaledent, Alstatten, Switzerland), WaveOne Gold (WOG) (Dentsply Sirona, Ballaigues, Switzerland), RECIPROC blue (VDW, Munich, Germany), and XP Shaper (XP) (FKG, La Chaux de Fonds, Switzerland) have improved mechanical properties that can potentially make them suitable for gutta-percha removal. The XP is a minimal taper file that can expand beyond its

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core size to adapt to the anatomy of the root canal space and may be more efficient in removing root filling materials than other single-file systems as it provides more space for the debris to be displaced.[16,304] The XP has been reported to provide a shorter operation time and showed the optimum results without any added untoward events when operated at a higher speed (3000 rpm) when compared to other single-file systems.[16] A full rotation motion may also be a more suitable cutting motion in root filling removal than a reciprocating motion.[16] Rotary retreatment files may be more advantageous in the amount of debris extrusion when compared with manual techniques using Hedström files with chloroform,[124,337] although all instrumentation techniques resulted in some radicular extrusion.[146,310] The literature shows more debris extrusion produced by instruments used with a reciprocating movement than instruments used with a continuous rotary movement.[44,45,51,336,340] Likewise, the rotary XP shaper instrument has been reported to extrude a significantly smaller amount of debris compared with reciprocating Reciproc Blue instruments because of its expanding snake-like structure, which may be ideal to mechanically remove root fillings extruded into periradicular tissues (Fig. 20.36). Furthermore, a combination of both rotary instruments for initial quick removal of gutta-percha and hand instruments to refine and complete the cleaning of the canal, especially in the radicular third of the root, shows the better protocol for obtaining clean canal walls during endodontic retreatment.[28,151] It has been reported that the use of a solvent after mechanical retreatment procedures may enhance root filling removal.[50] The use of a solvent, such as chloroform, may help to dissolve the gutta-percha root fillings remaining in both the periradicular tissues and the root canal system after mechanical removal attempts using a micro-hand instrument or an efficient rotary single-file system such as the XP (Fig. 20.37 *D–G*). In addition, chloroform has been reported to be capable of reducing the intracanal levels of *Enterococcus faecalis*.[79,209] However, when chloroform is used during the early stages of gutta-percha removal, more filling material will remain in the canal and some of it may be extruded beyond the radicular foramen.[146,198] Although the carcinogenicity of chloroform in humans is suspect, chloroform is regarded as a safe and effective endodontic solvent when used carefully.[56,204]

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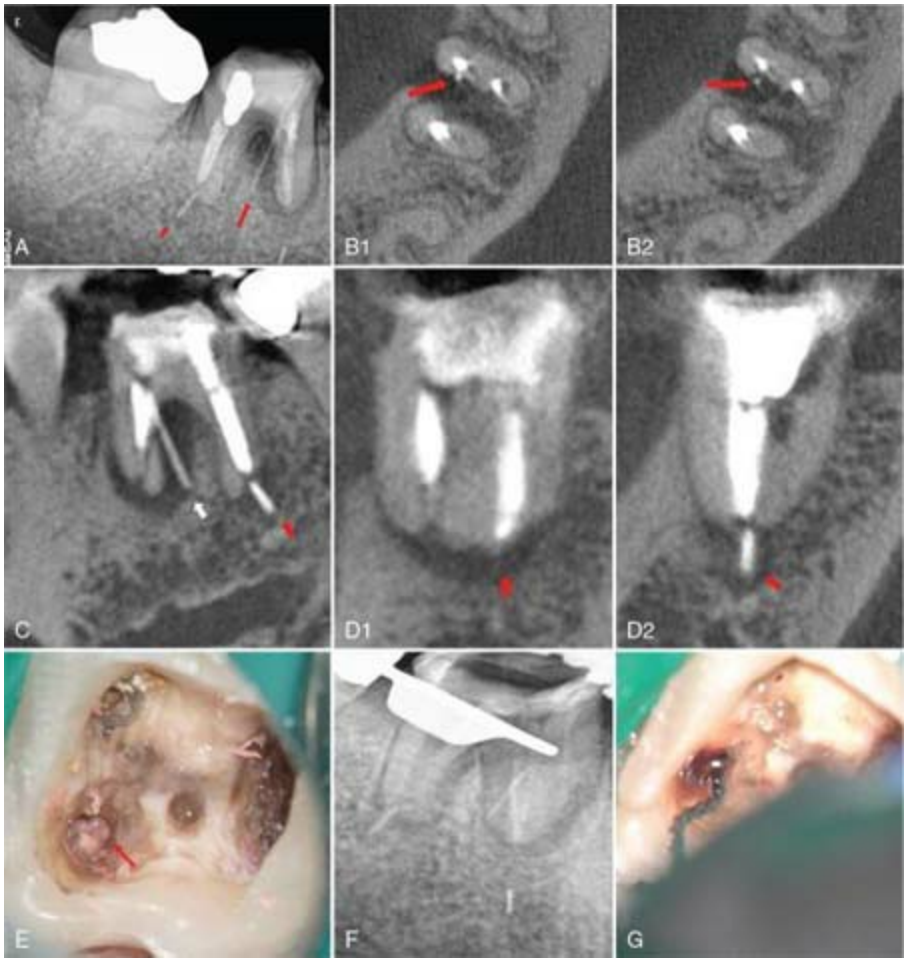

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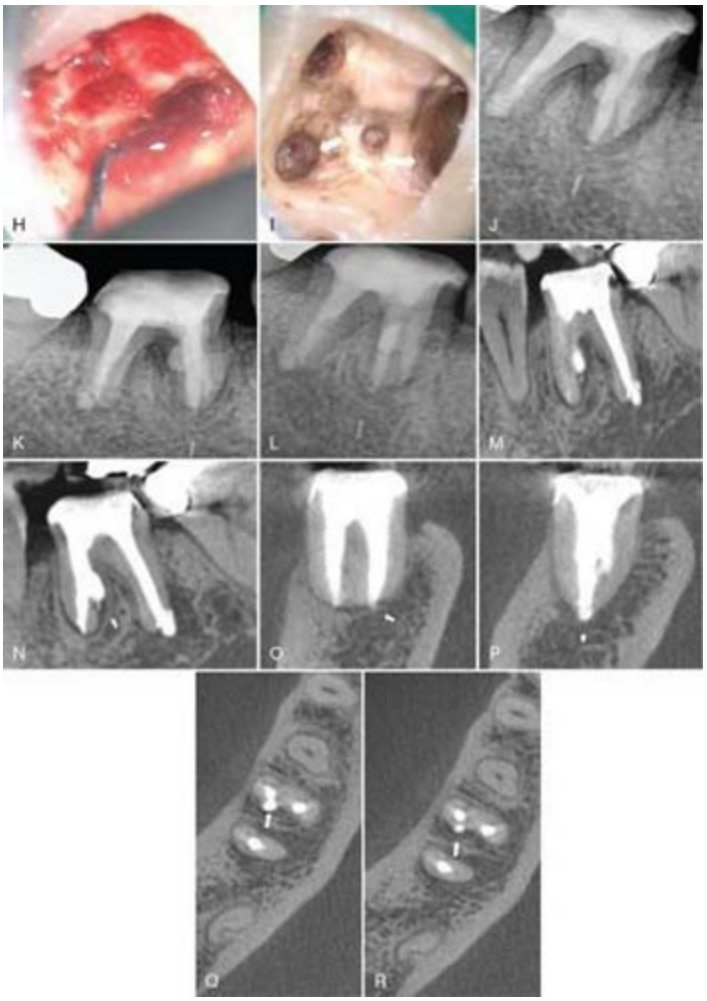

FIG. 20.36 **A,** Preoperative radiograph of the mandibular right first molar showing extruded root filling materials into periapical tissues *(red arrows)*. **B1** and **B2,** Cone-beam computed tomography (CBCT) images in axial view showing the perforation site from the mesiobuccal canal into the furcation *(red arrow)*. **C,** CBCT image in sagittal view showing extruded root fillings from the distal root *(red arrow)* and the perforation *(white arrow)*. **D1** and **D2,** CBCT images in coronal view showing the mesial root without extruded root fillings *(red arrow)* (**D1**) and the distal root with extruded root fillings *(red arrow)* (**D2**). **E,** Magnification of the MB canal with perforation *(red arrow)*. **F,** Interoperative radiograph showing removal of the intracanal root fillings. **G,** Magnification of the MB canal showing removal of the extruded root fillings from the perforation with an XP-Endo Shaper. **H,** Magnification of the distal canal showing removal of the extruded root fillings from the distal root end with an XP-Endo Shaper. **I,** Magnification of the root canals after the removal of extruded root fillings *(white arrows)*. **J,** Postoperative radiograph showing all the root canals including the perforation were successfully filled with mineral trioxide aggregate (MTA). **K,** Three-month postoperative radiograph showing reduced periapical lesions. **L,** Ten-month postoperative

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radiograph showing nice periapical healing. **M,** Ten-month postoperative CBCT image in sagittal view showing furcal healing. **N,** Ten-month postoperative CBCT image in sagittal view showing nice periapical healing around the perforation site *(white arrow)*. **O,** Ten-month postoperative CBCT image in coronal view showing nice periapical healing associated with the mesial root *(white arrow)*. **P,** Ten-month postoperative CBCT image in coronal view showing nice periapical healing associated with the distal root *(white arrow)*. **Q** and **R,** Ten-month postoperative CBCT image in axial view showing periapical healing associated with the perforation site *(white arrows)*.

Set of seven radiographs and two photographs marked A through G shows the steps involved in the removal of extra canals extending beyond the root canals of the first molar of lower jaw.

Set of nine radiographs and two photographs marked H through R shows the healing of damaged tissues after the removal of extended root canal in the right first molar of lower jaw.

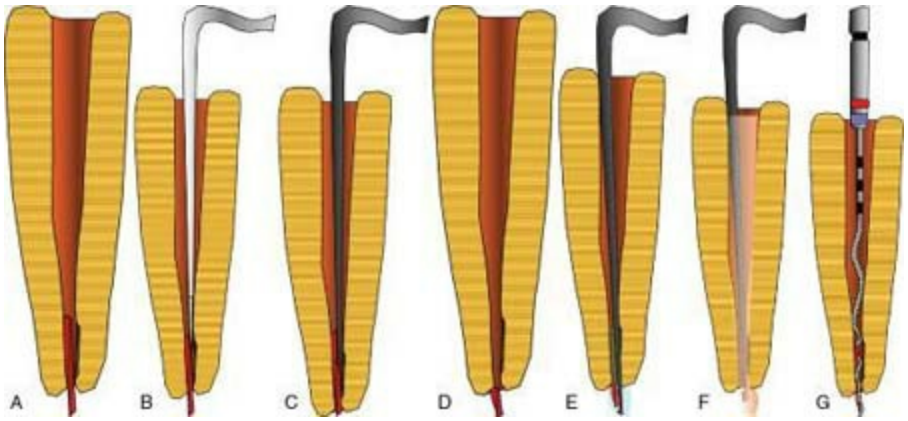

FIG. 20.37 Diagrams showing removal of gutta-percha filling materials extruded beyond the radicular foramen. **A,** Solid gutta-percha filling extruded beyond the radicular foramen. **B,** Small space is created between the canal wall and the gutta-percha filling with a thin ultrasonic tip (such as TFRK-6/12 and TFRK-S ultrasonic tips). **C,** Gutta-percha removal hand instrument, such as the TFRK-GPR and the EGPR, is brought into the space with the tiny projection pressed against the gutta-percha filling to pull it out. **D,** Extruded gutta-percha root filling portion separated from the coronal portion within the canal. **E,** Chloroform or some other solvent is filled in the canal to agitate it beyond the widened radicular foramen with a gutta-percha removal hand instrument or expandable rotary files to dissolve the extruded portion. **F,** Gutta-percha filling material is diluted, dissolved with the hand instrument in push-and-pull motions, and is irrigated out with a

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minimally invasive irrigant such as saline. **G,** Extruded gutta-percha filling material can also be braided out with those expandable rotary files with caution not to break them or not to damage the inferior alveolar nerve if close to it.

Set of seven illustrations marked A through G shows the changes in extruded gutta-percha extended beyond the root tip of tooth.

Unlike rotary instruments, the EGPR-L/R/U/D hand instruments and the GPR instrument are 30 mm in length, allowing them to remove obturation materials not only from the radicular third of the canal but also from the periradicular tissues through the radicular foramen. The micro-debriders are designed to be used in conjunction with a DOM. They have a Hedström cutting configuration with a standard.02 taper in sizes 20 and 30 with 16 mm of cutting flutes, which may not be long enough to reach the gutta-percha filling material beyond the radicular foramen. Another option to reach the root fillings extruded into the radicular tissues is to use an Endo Handle (Vento Endo, Logan, UT) which is designed to be used under the DOM with a pre-bent 30-mm hand file attached to the locking chamber.

First, a small hole must be created between the canal wall and the gutta-percha filling with a small diameter ultrasonic tip to facilitate the root filling removal with the hand gutta-percha removal instrument placed into the hole. It is ideal to remove the root filling in one large piece from the periradicular tissues. Care should be taken not to push the root filling materials in a radicular direction with those micro-instruments especially for a canal with large radicular foramen. Then the gutta-percha filling is scraped and taken out from the canal wall with the micro-instrument.

The EGPR instrument has a tiny projection on one side of the tip for scraping and engaging the gutta-percha filling left on the canal wall. The overall diameter of the tip is 0.3 mm and the length of the projection is 0.1 mm. This hand instrument works like a scraper on the canal wall. When the radicular portion of the EGPR comes down to the bottom of the hole created, the projection is directed to the gutta-percha filling so that it can be hooked on the projection pressed against it. Then the gutta-percha filling should come apart in large pieces from the canal wall (see Fig. 20.37, *A–C*).

On the other hand, the TFRK-GPR instrument has a tiny conical harpoon-shaped tip that is sharp and small enough to sneak into a narrow space on the

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canal wall and hook the gutta-percha on the rim on the return stroke. The tip portion of this instrument has a.40 taper with the minimum tip diameter of 0.1 mm and the maximum diameter of 0.3 mm with a 0.2-mm diameter shank supporting the tip portion. The 0.1-mm gap between the 0.3-mm and the 0.2-mm diameters works practically as a tiny circular projection over the shank portion that can hook a mass of gutta-percha fillings. The conical tip with the maximum diameter of the shank portion being 0.6 mm makes it possible to place the instrument into the canal beyond the curve just like a Hedström file. The gutta-percha filling can also be hooked by pressing the projecting rim against it, and then should be removed in large pieces from the canal wall.

When a small amount of gutta-percha filling remains on the inside curve or in the radicular third of the canal after the mechanical removal attempts, chloroform should be filled in the canal and the GPR instrument can be used to agitate it in short push-and-pull motions in the canal, or even outside the canal if root filling is extruded beyond the radicular foramen, to actively dissolve the residual root filling. The mean percent total values of residual material ranged from 43.9% to 0.02%, with the vast majority of studies reporting values less than 10%.[275]

Management of tissue damage caused by extrusion of root canal filling materials

When root canal filling material extends into the periradicular spaces, tissue damage may occur. The majority of these cases are managed by nonsurgical methods such as analgesics, cold packs, corticosteroids, and antibiotics.[90] Dexamethasone is a corticosteroid that has been widely used in dentistry, and it seems to decrease periradicular inflammation caused by a foreign body[210] with minimal side effects.[65] If the tissue damage is extended to the IAN or maxillary sinus, then a different approach may be necessary.

Sinus perforation

There is a close proximity of maxillary premolars and molars to the sinus, and the apices of these teeth can be less than 2 mm from the floor of the sinus.[78] Pathologic destruction of the sinus floor may predispose a maxillary sinus communication,[294] producing an oroantral communication,[98,190,278,355]

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also known as an OAC. In general, exposure of the sinus lumen is well tolerated,[155,238] with no overt repercussions on healing. One investigator[355] found no significant difference in the healing of the mucosa in patients with and without intraoperative perforations and regeneration of the membrane, complete with cilia, in approximately 5 months following complete removal.[25] However, introduction of foreign materials during the access or apical resection/manipulation can initiate thickening of the sinus mucosa with symptoms of maxillary sinusitis.[84]

Conventional radiographs appear to be of limited value in predicting the potential for OAC, with the position of the root tip, periapical lesion, or outline of the lesion not statistically significant in that respect.[224] In contrast, a CBCT survey of the intended surgical field is much more informative, providing accurate measurements of the osseous thickness, distance to the sinus floor, and presence of pathosis.[33]

Maxillary sinus complications secondary to the extrusion of root canal filling material need to be managed differently. Approximately 10% of all patients with chronic sinusitis are found to have an aspergilloma.[120,193] It has been suggested that root canal treated teeth with overextension of the root canal sealer into the maxillary sinus might be the main cause for aspergillosis in healthy patients.[21,22] Surgical intervention is usually the most predictable method for removing extruded root canal filling material and allowing for adequate healing secondary to IAN or sinus complications.[333]

Inferior alveolar nerve injury

Sensory nerves are extremely sensitive to physical, radiation, chemical, thermal and ischemic damage. Despite their capacity to heal, if the injury causes sufficient damage, then persistent posttraumatic sensory neuropathy will result. The presenting symptoms of sensory neuropathy include; a neuropathic area within the dermatome where the surgery or injury took place,[259] allodynia (pain to touch or cold), hyperpathia (continued pain or altered sensation after stimulus removed), hyperalgesia (increased pain to a painful stimulus for example exaggerated response to sharp blunt discrimination test despite ongoing numbness). Additional clinical symptoms may include ongoing, spontaneous and/ or elicited altered sensation

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(paraesthesia or dysesthesia), a tingling or formication (ants crawling on the skin), burning or shooting pain or electric shock. The sensory deficit to mechanical function indicates damage to larger myelinated fibers, whereas sensor deficit to noxious and thermal stimuli indicates damage to small diameter afferent fibers of the spinothalamic tract.[256]

Clinicians must use appropriate medical language and not refer to sensory neuropathy as 'paraesthesia' which is a clinical sign and not a diagnosis. Generally, neurologists refer to either hypoesthetic (predominantly anesthetic [numb] with reduced mechanosensory function) or hyperesthetic (ongoing, spontaneous and/or elicited pain or dysesthesia) sensory neuropathies. Posttraumatic neuropathic pain (PTNP) is a recognized neurologic diagnosis (ICOP).[154a]

Iatrogenic trigeminal nerve injuries remain significant and complex clinical problems. The impact on the patient of posttraumatic sensory neuropathy can be functionally and psychologically severe, particularly when painful and iatrogenic. The trigeminal nerve underpins our very existence, providing sensory protection to our eyes, nose, mouth and brain. Thus the affective and emotional reaction to the threat or experience of trigeminal pain is thus proportionally immense. Altered sensation and pain in the orofacial region may interfere with speaking, eating, kissing, shaving, applying make-up, tooth brushing and drinking—in fact, just about every social interaction we take for granted. Thus these injuries have a significant negative effect on the patient's quality of life and the iatrogenesis of these injuries lead to significant psychological effects.[308] The management of patients with posttraumatic sensory neuropathy is complex and 'curative' treatment is unavailable, thus prevention of these injuries is paramount. Trigeminal nerve injury is the most challenging consequence of contemporary dental surgical procedures with significant medical-legal repercussions (see also Chapter 27).[46] An approximate estimation places the incidence if IAN injury at 5%, with approximately one third of these patients afflicted with neuropathies, including neuropathic pain.[262] Common causes of injury to the IAN are listed in Box 20.2.[258]

Box 20.2

Common Causes of Damage to the Inferior Alveolar

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Nerve

1. Local anesthetic injections
2. Third molar surgery
3. Implants
4. Endodontics (extrusion of the materials used in endodontic treatment, overinstrumentation, elevated temperatures)
5. Ablative surgery
6. Trauma
7. Orthognathic surgery

The highest incidence of reported damage to the inferior alveolar and lingual nerves is associated with third molar extractions.[18,140,141] Local anesthesia was the second most common cause of nerve injury, and its exact mechanism can be confusing. It may be physical (needle damage, epineural/perineural hemorrhage) or chemical (local anesthetic contents). Reports have implicated certain local anesthetics of high concentrations (prilocaine 4% and articaine 4%) with IAN neuropathies (see also Chapter 6).[17,131,142,248] A relatively small percentage of IAN injury cases (8%) are associated with an endodontic procedure.[261]

IAN damage has been suggested to occur in up to 1% of mandibular premolars that receive root canal treatment.[87] In one retrospective study examining cases of paraesthesia related to endodontic treatment of mandibular premolars, the incidence was 0.96% (8/832).[172] In a survey of 2338 patients, 7% sustained chronic neuropathic pain after a single endodontic procedure.[170] Most reports of endodontic-related nerve injuries are case reports with very little analysis of the clinical impact for the patient, risk factors, or management issues. To date, there are four case series reports on inferior alveolar nerve injuries (IANIs) related to root canal treatment (RCT) including 61 cases,[246] eight cases,[172] four cases,[292] and one case series reported on the treatment of 11 cases.[121] These case series predominantly focus on the outcomes surgical exploration and irrigation of damage nerves.

The mechanisms of sensory neuropathy are complex, and they are likely not only due to mechanical damage including perforation or compression. Pogrel[247] concluded that the severity of the injury was proportional to the

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process by which the injury was created, and these processes included scalpel blade wounding/transection, crushing, and stretching damages. These would be analogous to the dissection and reflection of the flap, improper retraction location/application, and postoperative edema. However, endodontics is complicated with use of chemicals and fillers that provide additional risk of nerve injury. It is likely that chemical (pH of dental materials), ischemia, hemorrhage (the iron in hemoglobin irritates nerve tissue), and thermal factors may compound the nerve injury resulting in persistent neuropathy. Compression of peripheral sensory nerves over 6 hours can evoke nerve fiber atrophy.[298] Ischemia alone without direct nerve damage will cause sufficient neural inflammation and damage to cause permanent nerve injury within only 6 hours.[230] The IAN may be damaged when an increase in temperature proximal to the IAN is greater than 10°C.[87] The type of sealer cement used may influence the symptoms of IAN damage.[216]

The three primary factors responsible for IAN damage during endodontic treatment are as follows:

1. Chemical toxicity and mechanical pressure produced by leakage of sealers into areas close to the mandibular canal are other potential mechanisms for nerve injury related to endodontic procedures, with the IAN and mental nerve being most affected. Cytotoxic materials employed either in the preparation (e.g., irrigation liquids, intracanal medicaments)[2,109] or the obturation of the canal space[277] as the materials most commonly associated with these complications are those containing paraformaldehyde.[3] In addition, most endodontic materials have very high pH levels, resulting in immediate nerve damage likely to be permanent (Fig. 20.38).[37]
2. Mechanical factors: repeated insertions of endodontic instruments through the apex into the IAN and surrounding tissues[70]
3. Thermal factors: inappropriate/prolonged heat application[89]

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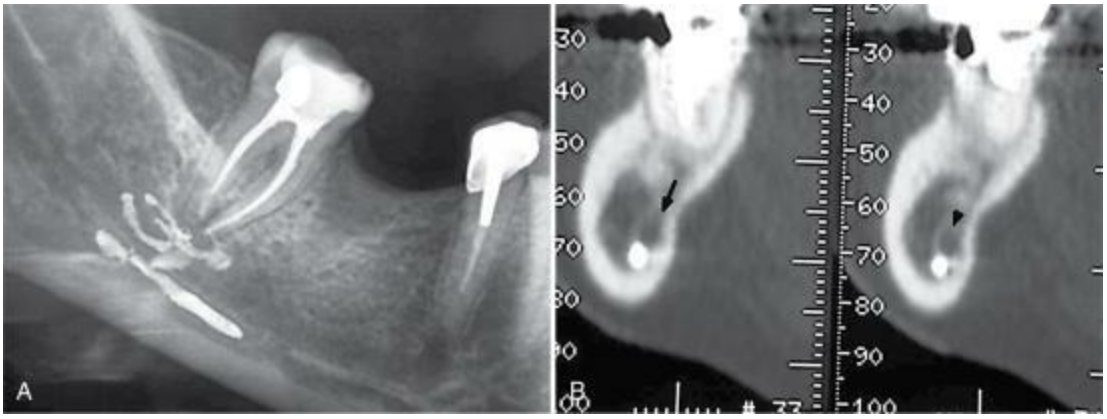

FIG. 20.38 **A,** Panoramic radiograph demonstrating significant overfilling and extension in the radicular vacuoles and overlying the mandibular canal. **B,** A coronal CT section demonstrating that the extruded material is lateral and below the mandibular bundle *(arrows).* The patient's sensory complications were temporary.

Set of three radiographs marked A and B shows panoramic and coronal view of lower jaw where the tooth has extra filling material below the root of tooth

Source: (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

Risk assessment for the prevention of endodontic nerve injury

Preoperative assessment is crucial in prevention of nerve injury related to endodontics (Box 20.3). Prevention is the key to successful treatment. Damage, either directly to the IAN or to the vessel(s) that nourish it, will result in sensory events that may be transient or permanent. Pretreatment radiographic assessment using periapical images is mandatory; in critical cases, CBCT may be more appropriate and precise in establishing proximity (Fig. 20.39). Assessment should include the following:

- Proximity of the apex of the tooth to the IDC. Lower molar teeth where root apices are is close proximity to the Inferior dental canal and or mental foramen. Mandibular second molars are most commonly associated with this population, but cases involving treatment of mandibular first molars and premolars have also been

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reported.[172] Careful consideration should also focus on the distance between the root apices and the mandibular canal. For mandibular first molars, this distance varies between 1 and 4 mm, and for mandibular second and third molars, the distance can be less than 1 mm.[334] With mandibular premolars, the proximity of a mental foramen should always be considered.[219] In addition, a study using CBCT has shown that the mesiobuccal root apex of maxillary second molars is frequently in very close proximity with the sinus floor (see Fig. 20.39).[228]

- Tilotta-Yasukawa and colleagues (2006) determined the proximity of the apex of the premolars and molars in relation to the mandibular canal, as well as the relationship between the IAN and its corresponding artery, with the goal of understanding how endodontic filling material spreads through the bone to penetrate the mandibular canal. They observed that the distance between the dental apex and the mandibular canal was more variable (and generally greater) for the first molar than for the second and third molars (1 to 4 mm vs. <1 mm [35 cases in 40 mandibles examined]). The presence of bone between the apices and the IAN canal does not necessarily protect against injury to the nerve. Research has provided an enlightening explanation to this puzzle (Figs. 20.40–20.42).[334] Using fresh cadaver specimens (mandibles), researchers examined the true anatomic relationship among the root apices, IAN, and the artery. They drew the following conclusions:
  - There was never any compact layer of cortical bone surrounding the mandibular nerve sheath. In some cases, a slightly denser cancellous bone was found, but it was replete with multiple perforations that would not afford significant resistance to penetration.[137]
  - The proximity of the IAN canal to the apices of the second molars was < 1 mm, and it was more variable (1 to 4 mm) with first molars. This could explain the higher percentages of paresthesias reported with these teeth.
  - The mandibular artery ran along the superomedial side of the IAN up to the first molar, and then crossed on the upper side

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until the mental foramen. Perforation of this vessel, with the concomitant hemorrhage exerting pressure on the pedicle, could feasibly produce a paresthetic event via the resulting ischemia.[71]

- Only with root apices in direct contact with the IAN canal were extrusions able to penetrate the nerve. If cancellous bone was interspersed between the apices and pedicle, the spread of the extrusion through the trabeculations remained at the periphery of the sheath.
- The low density of trabeculations and presence of numerous vacuoles provided an avenue for the diffusion of extruded irrigation fluids and obturation materials toward the IAN bundle. In a tooth with apical resorption, the foramen is frequently enlarged[363]; these two factors in concert could explain the occurrence of sensory symptoms in the absence of clinical or radiographic signs of overexpression or close proximity to the mandibular nerve.

- Predisposing radiological factors that may indicate a higher risk of an adverse incident during root canal treatment or cause an extrusion of endodontic filler/hypochlorite accident
  - Resorption defects where extent is not identified, such as internal/external communicating with root canal and external surface of the root
  - Suspicion of a perforation communicating with the external root surface
  - Root fracture where there could be a potential communication of the root canal with external root surface
  - Sclerosed root canal
  - Dens invaginatus
  - Presence and the diameter of bone lesions of endodontic origin may also influence the occurrence of paraesthesia, especially when associated with the premolars and lower molars.[176]

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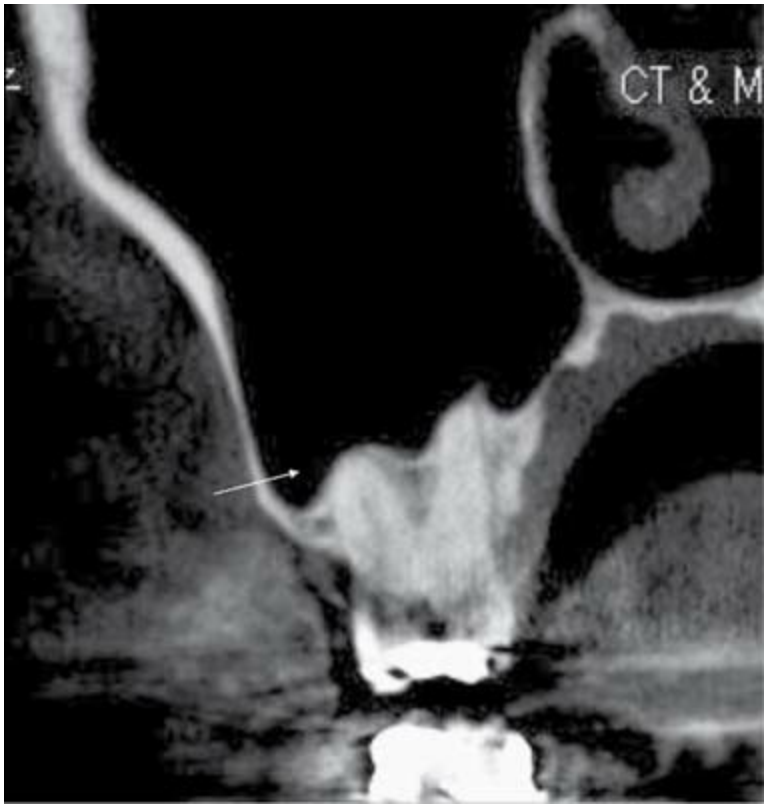

FIG. 20.39 Cone-beam computed tomography (CBCT) image in coronal view showing that the mesiobuccal root apex of a maxillary second molar is in close proximity with the sinus floor.

CBCT image shows an inflamed cavity next to the root tip of second molar in the upper jaw.

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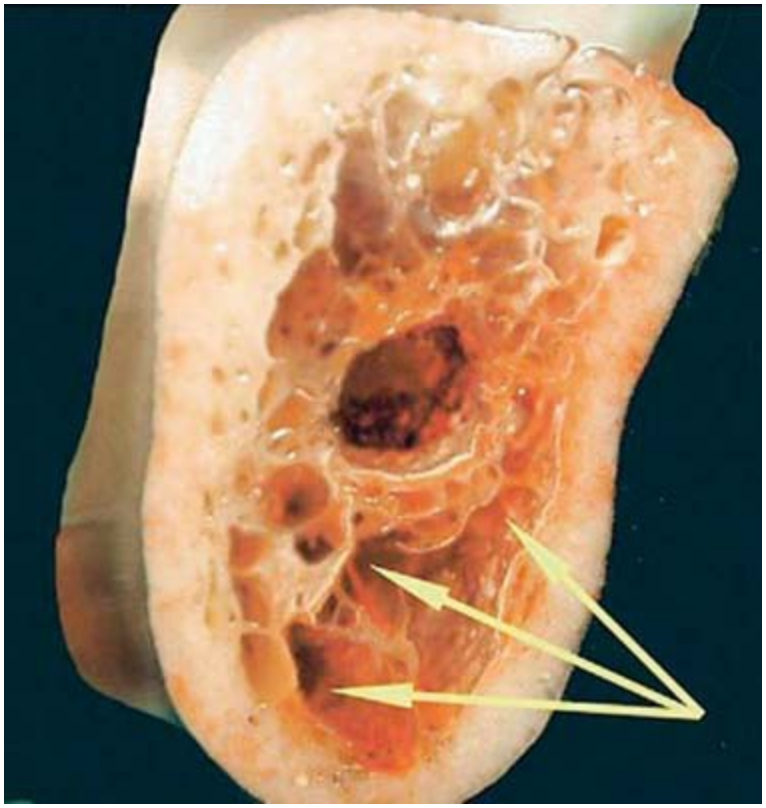

FIG. 20.40 Cross section of mandible in the molar region showing the presence of bone vacuoles *(yellow arrows)*.

Mandible shows soft and perforated tissue forming chambers.

Source: (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

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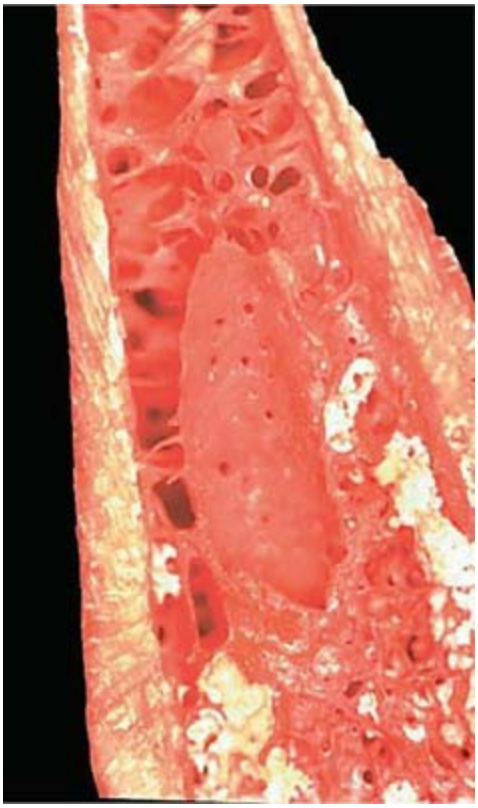

FIG. 20.41 Longitudinal mandible cross section in the molar region showing the spongy bone cover of the neurovascular bundle. Note the multiple fenestrations and honeycombed trabeculations.

Longitudinal section of lower jaw shows an array of hollow cells.

Source: (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

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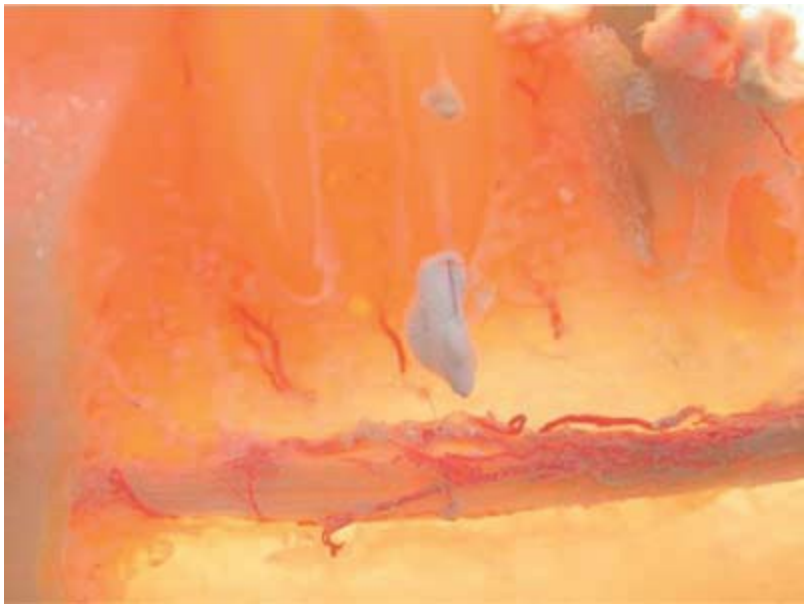

FIG. 20.42 Overfill of the distal root under direct observation. Note the proximity of the extruded material to the artery and nerve bundle (pedicle).

Close-up of extruded material of tooth moving toward the horizontal stalk-like structure at the back of mouth is shown.

Source: (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

Box 20.3

Key Points for Prevention of Nerve Injury Related to Endodontics

- Aways assess the proximity of the tooth apex to the inferior alveolar nerve (IAN).
- Always assess additional risk factors in root morphology and bone factors contributing to leakage of chemicals adjacent to the IAN.
- Take all the steps to minimize the apical breach during endodontic treatment if any of the previous risk factors exists. This will allow leakage of high pH chemicals into the IAN, rendering permanent chemical damage to the IAN.

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- Consider immediate extraction of the tooth and generous lavage of the socket along with adjunctive medical treatment (described in this chapter) if the patient develops sensory neuropathy within 3 days of endodontic treatment.

Operative techniques to minimize nerve injuries

Surgical endodontic procedures need to be designed to minimize trauma to nerves including the IAN, mental nerve, and mental foramen. Although the mental foramen is most often located apically to the second premolar,[217,241,242,243] it can vary in anterior-posterior locations,[162,220] coronal-apical locations,[95] and number.

Careful presurgical assessment via radiographs and CBCT provides critical information on the size, location, and spatial relation of the planned surgery to nearby trigeminal nerves.[32] Before exiting the mandibular canal, the nerve often loops anteriorly, then superior and posteriorly in the apical portion of the premolar region.[119] If an apical procedure is planned in an area commensurate with this anatomic variant, then CBCT is warranted and strongly advised.

Controlled reflection of the tissue overlying the suspected location of the foramen will diminish the incidence of transection. Cautious reflection of the flap will reveal evidence of tissue “funneling” at its base (Fig. 20.43); this is the telltale sign of the exiting nerve bundle.[221] This exiting tissue, upon discovery, can be marked with a dot of gentian violet or methylene blue dye. By highlighting it in this fashion, the surgeon can avoid unintentional impingement with a retractor. The tissues should be minimally distended and the underside of the flap kept well hydrated.

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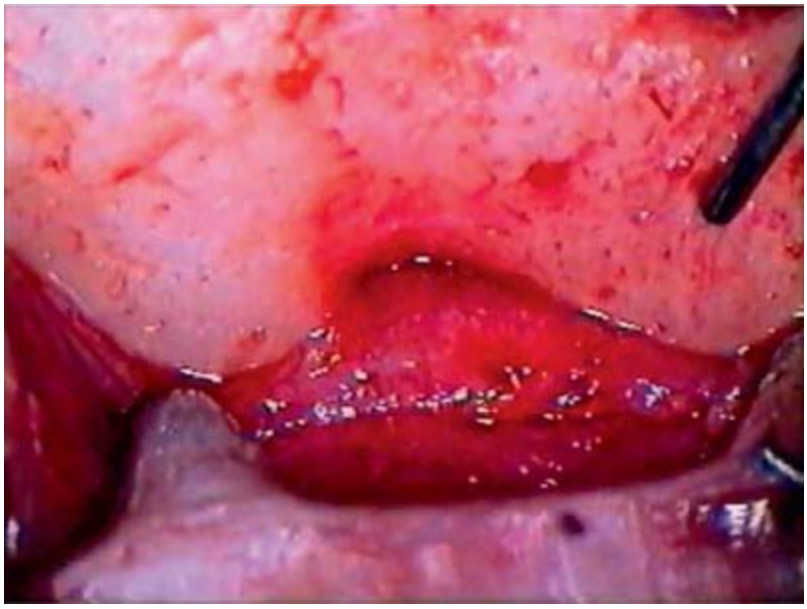

FIG. 20.43 Full-thickness reflection of the flap in the region of the premolar root apices reveals the mental nerve exiting the foramen; note how the tissue "funnels" from the reflected flap to the foramen.

Close-up view of the flap near root tip of premolar shows the mental nerve.

Source: (From Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e47, 2006.)

Prevention of over instrumentation: Every attempt should be undertaken to confine the instrumentation and irrigation processes to within the canal space; damage from extrusions can be chemical or mechanical in origin,[292] and toxic products can easily diffuse through the trabecular spaces and vacuoles surrounding the nerve bundle. This latency in diffusion, and the potential onset of paresthetic symptoms, dictates close monitoring for 72 hours postoperatively in cases where extrusion is apparent.[246]

Prevention of over filling with beyond apex extrusion of filler or treatment chemicals: Any tooth requiring endodontic therapy, which is in close proximity to the IAN canal, should require special attention, including: NaOCl (as irrigation) and calcium hydroxide (as sealant; calcium hydroxide medicaments breach the canal roof, precipitating a vascular bleed resulting in hemoglobin irritation of the nerve due to the iron content).[31,87]

Prevention of thermal injury: Imprudent heat application during

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thermoplastic compaction of gutta-percha may also lead to IAN injury.[31]

The clinician must be aware and record any events that may indicate operative nerve injury, including extreme pain during LA IDB, canal instrumentation, irrigation, medication, or filling; and sudden and profuse hemorrhage arising from the apex of the tooth

Appropriate postoperative periapical radiographs must be taken, in accordance with endodontic clinical guidance, to check for any extrusion of dressing or filling materials into the inferior dental canal or around the mental foramen.

Home check should be undertaken, particularly for high-risk cases, to assess signs of persistent or new neuropathy.

Management of nerve injuries

The priority in management of patients with iatrogenic nerve injuries is for them to understand what has happened, the likely prognosis and possible support, and medical or surgical interventions. Management should be holistic and of the patient with the nerve injury and not just the neuropathy itself.[261] Pain, functional problems, and psychological impact should be assessed using questionnaires.[82] Grötz and colleagues[121] reported on 11 patients with endodontic-associated neuropathy and their management, reporting that the neurologic findings were dominated by hypoaesthesia and dysesthesia, with 50% of patients reporting pain. Initial x-rays showed root filling material in the area of the mandibular canal. Nine cases were treated with apicectomy and decompression of the nerve: in two cases, extraction of the tooth was necessary; only one patient reported persistent pain after surgery.

It is important to recognize that IANI can occur due to local anesthetic block injections and the clinician can often discriminate between endodontic and local anesthesia-caused nerve injuries by careful questioning and careful clinical neurologic assessment. Mapping of the neuropathic area will discriminate between inferior dental block (IDB) and endodontic nerve injury (Figs. 20.44 and 20.45).[49]

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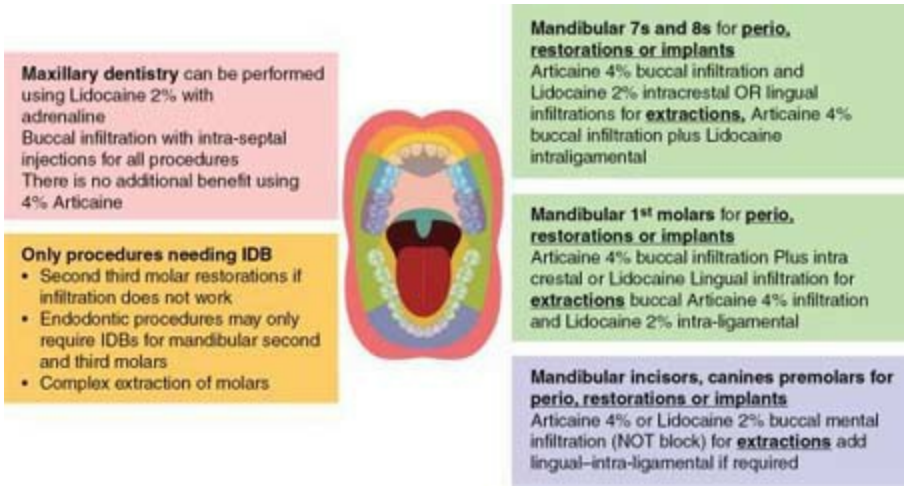

FIG. 20.44 Infiltration dentistry is dependent upon the site and procedure.

Illustration shows upper and lower jaw in open mouth. Text reads: Maxillary dentistry can be performed using Lidocaine 2 percent with adrenaline. Buccal infiltration with intra-septal injections for all procedures. There is no additional benefit using 4 percent Articaine.

Only procedures needing IDB

• Second third molar restorations if infiltration does not work

• Endodontic procedures may only require IDBs for mandibular second and third molars.

• Complex extraction of molars

Mandibular 7s and 8s for perio, restorations or implants

Articaine 4 percent buccal infiltration and Lidocaine 2 percent intracrestal OR lingual infiltrations for extractions, Articaine 4 percent buccal infiltration plus Lidocaine intraligamental.

Mandibular 1st molars for perio, restorations or implants

Articaine 4 percent buccal infiltration Plus intra crestal or Lidocaine Lingual infiltration for extractions buccal Articaine 4 percent infiltration and Lidocaine 2 percent intra-ligamental.

Mandibular incisors, canines premolars for perio, restorations or implants

Articaine 4 percent or Lidocaine 2 percent buccal mental infiltration (NOT block) for extractions add lingual–intra-ligamental if required.

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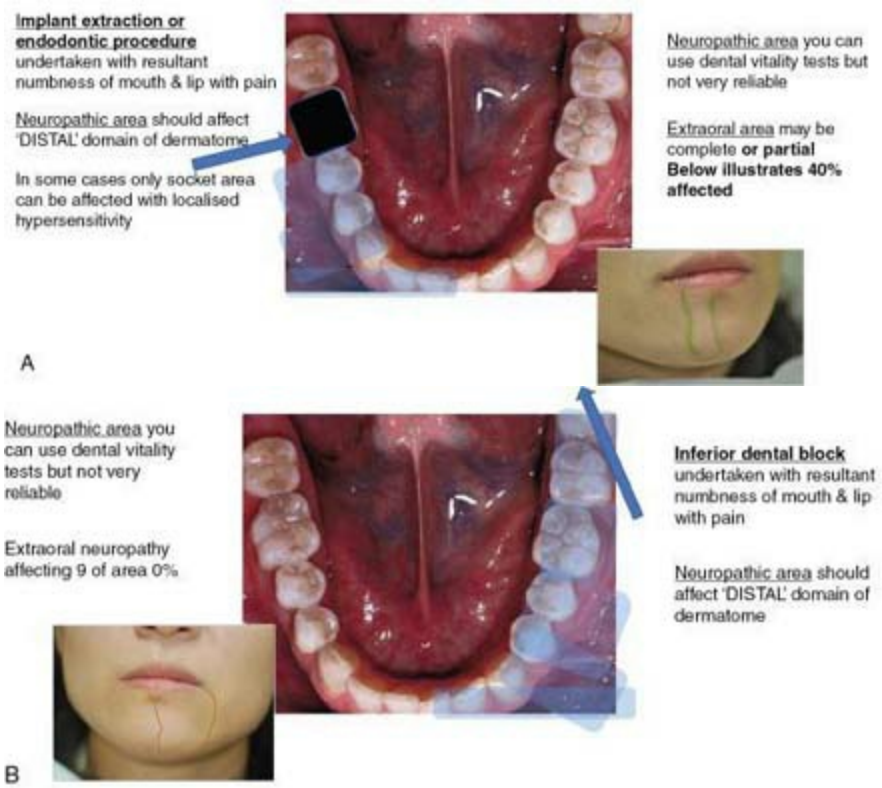

FIG. 20.45 **A,** Nerve injury proximal to endodontically treated tooth. Cause = endo. **B,** Inferior dental block nerve injury initially will cause neuropathy of the whole dermatome.

A) Close-up view of lower jaw shows highlighted teeth in lower right side. Text reads: Implant extraction or endodontic procedure undertaken with resultant numbness of mouth & lip with pain.

Neuropathic area should affect 'DISTAL' domain of dermatome. In some cases only socket area can be affected with localized hypersensitivity. Neuropathic area you can use dental vitality tests but not very reliable. Extraoral area may be complete or partial Below illustrates 40 percent affected. Close-up view of lower mouth highlights the region with U-shaped outline below lower right teeth.

B) Close-up view of lower jaw shows highlighted teeth in lower left side. Text reads: Neuropathic area you can use dental vitality tests but not very reliable. Extraoral neuropathy affecting 9 of area 0 percent. Inferior dental block undertaken with resultant numbness of mouth and lip with pain. Neuropathic area should affect 'DISTAL' domain of dermatome. Close-up view of lower mouth highlights the region with wide U-shaped outline below left right teeth.

Diagnosis is based upon the signs and symptoms described earlier in this section. Posttraumatic sensory neuropathy is a clinical diagnosis and

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knowledge of the preoperative risk along with the posttreatment radiographs should be sufficient in confirming the diagnosis. Further radiographic assessment, especially CBCT, may be useful in revealing the extent of location of extruded materials in relation to the mandibular canal or the maxillary sinus) but is unlikely to improve treatment outcome.[31,103,113] Chemical nerve injury may not be obvious radiographically: If the patient is suffering from neuropathy after the local anesthetic has worn off and the postoperative radiographs confirm that there is no radiopaque material in the canal, chemical nerve injury may be presumed

Transient paresthesia may occur in cases where a protracted surgical procedure, inadvertent stretching, or excessive postoperative edema has occurred. Appropriate reevaluation appointments include neurosensory testing and mapping the outline of the sensory deficit. Return of sensation within the first few weeks indicates a neuropraxia and may indicate prognosis but cannot predict full recovery. It is not possible to distinguish between temporary or permanent nerve injuries early postoperatively, thus treatment must be based upon the patient's symptom severity, the extent of neuropathy, and the evidence of injury (recent treatment and radiographic overfill). Timing of intervention is paramount and ideally high-risk patients should be followed up for 24 to 60 hours post endo to check for onset of symptoms of neuropathy.

Assessment

The patient must be assessed for pain, functional limitations, and psychological impact. Further confirmation of the neuropathic area and mechanosensory testing is important to establish the extent and symptoms of the neuropathy. The patient must be assessed holistically, including their history of the event and if it is related to the initiation of pain.[261] Ensure that the pain history excludes preexisting neuropathic pain, including the following:

- Severe pain during procedure (funny bone pain)
- High-level postsurgical pain (indicative of nerve injury)
- Ongoing pain, altered sensation, and/or numbness
- Functional problems

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- Psychological issues. Important questions about the mechanism and duration of the neuropathy will drive the timing and type of management.[260]

Necessary investigations include:

- Radiological: LCPA; CBCT necessary posttrauma
- Neurosensory to confirm that the presence of a neuropathy and distribution correlates with potential nerve injury
- Diagnostic local anesthesia blocks may be useful in evaluating the potential of some peripheral pain management strategies when medical management is unsuccessful.

Recent developments of medical resonance neurography may enhance the ability of the clinician to assess the trigeminal nerve injury early postoperatively, allowing for earlier surgical intervention, if required.[375]

Immediate intervention

If nerve section or severe injury is known or suspect, likely due to scalpel or drill, then immediate repair must be undertaken. If the transected ends are closely approximated following the injury, either naturally or surgically, proliferating axons will attempt to bridge the gap and, if they find a perineural tube to populate, gradually restore function. However, in cases where the gap is beyond a critical length, a neuroma may develop at the proximal end. Neuromas consist of dense fibrous tissue intermingled with nerve fascicles, with a disproportionate proliferation of unmyelinated fibers. Treatment, if initiated within 3 months of the initial event, often entails resection of the neuroma and repair by tension-free anastomosis or nerve grafting.

Early intervention

If the patient exhibits neuropathic signs during instrumentation (severe neuralgia) or after the local anesthesia has worn off, posttreatment radiographs should be reevaluated to observe the possibility of any obturation material extending into the IAN canal. Devine et al.[72] reported that there may

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be delayed development of sensory neuropathy, usually 2 to 3 days, likely due to chemical leakage through the treated tooth apex, resulting in chemical nerve injury which will not be apparent on a posttrreatment radiograph. Three months after the IAN injury, permanent central and peripheral changes occur within the nervous system subsequent to injury, which are unlikely to respond to surgical treatment intervention.[367]

There is a strong correlation between duration, origin, significance of the injury, and the prognosis for resolution of the paresthesia. The longer the irritation persists, either chemical of mechanical, the more fibers are involved in a degenerative process and the more likely the paresthesia will become permanent.[284] If injury to the nerve is demonstrated by postoperative neuropathy and posttreatment radiographs (not for chemical leakage) confirm the presence of a significant extrusion or overinstrumentation into the IAN canal, management entails the surgical removal of the offending material as soon as possible if the patient is symptomatic.[246,258,262]

Management should be performed by immediate removal of endodontic materials within 24 to 48 hours via periapical surgery, tooth extraction, or surgical debridement, whichever will be the most effective option with the least potential damage to the IAN.[75,178,246,258] If the patient wishes to retain the treated tooth and overfill is present in the inferior dental canal (IDC), then immediate mandibular buccal corticotomy under general anesthesia may be advised, with removal of overfill and hours of neural lavage with saline.[245a]

However, in spite of the best efforts to eliminate the causative agent, the symptoms may persist because of the associated chemical damage to the nerve. If sensory improvement has not been evident after 2 months, with or without repair during interventional surgery, then the prognosis for recovery is poor, and steps should be taken to alleviate the patient's concerns through counseling and systemic medications for chronic pain.[261]

Counseling and medical therapeutic management are indicated within 48 hours for injuries caused by endodontic treatment or local anesthetic injections.[99]

Watch and wait?

If the patient presents with a minimal neuropathic area which is tolerable and hyposensitive (nonpainful) then intervention may not be justified. Decisions

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about intervention have to be based on the patient's understanding of the risks and benefits. Although the prognosis in the case of the IAN damage caused by resorbable or inert material is favorable, as it is dissolved over time, the toxicity of the material, whether chemical or pH, may cause permanent damage if left in place.[222] In addition, pressure from the material on the IAN may be caused by a hematoma or inflammatory, which can also contribute to temporary or permanent IAN damage.[90,222] The prognosis of the IAN damage depends on the severity of the direct injury to the nerve, the toxicity of the irritant, the amount and speed of its resorption, and how rapidly the causative agent is removed.[222,226,246] Because the paresthesia can be of both a mechanical and a chemical nature, removal of the excess material might not be sufficient if the nerve fibers have already undergone degeneration from a chemical irritant. Nevertheless, paresthesia caused by an instant irritation of the nerve, like a minor overinstrumentation or swelling from infection or minor inflammation, usually subsides within days. If no signs of healing are observed within 3 to 6 months, the chances of healing are considered much lower, although normal sensory function might still return after this time.[113,334]

If the opportunity for immediate repair or early decompression has been missed, then the patient must be treated therapeutically, which may include medical and psychological interventions.

Psychological interventions may include counseling, mindfulness, acceptance, and commitment as well as cognitive behavioral therapies for patients with nerve injuries, which is very effective (there is limited evidence for success of this treatment for endodontic-related IANIs, but evidence does support psychological therapies for chronic pain and IANIs).[261]

Early medical management to minimize acute surgical neural inflammation using protocols undertaken for other acute sensory nerve injuries (Gatot, Tovi, 1986; Grötz et al., 1998). Steroids may maximize recovery of peripheral nerve injury (evidenced in spinal nerve injuries) using prednisolone (15 mg for 5 days, 10 mg for 5 days, and 5 mg for 5 days) and high-dose nonsteroidal antiinflammatory drugs (NSAIDs) (600 mg ibuprofen) as long as the patient has no contraindications to NSAIDs (gastric or peptic ulceration or allergy to NSAIDs) and a timely referral is made to an appropriately trained oral or maxillofacial microsurgeon. If the opportunity

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for early repair is missed, long-term therapeutic pain management will be necessary.[197a] Vitamin B complex and magnesium supplements may have some beneficial effects for neural recovery. Antibiotics are not recommended for nerve injuries unless infection related neuropathy is suspected but, in these cases, the tooth should be endodontically treated or extracted.

Regarding late medical management of chronic pain associated with endodontic treatment. Oshima[225] reported that 16 out of 271 patients presenting with chronic orofacial pain were diagnosed with chronic neuropathic tooth pain subsequent to endodontic retreatment. Most of these patients were treated for maxillary teeth. Seventy percent of the patients responded to tricyclic antidepressant therapy, which highlights the importance of establishing whether the patient has neuropathic pain. In Renton and Yilmaz's study,[261] all the patients presented too late for surgical decompression or it was not indicated. Thus two patients were managed with oxcarbazepine for neuralgic pain elicited with touch or cold and with topical clonazepam intraorally to manage the severe gingival discomfort. Two patients were prescribed topical 5% lidocaine patches (12 hours on nocte and 12 hours off daily) for debilitating mechanical allodynia in the extraoral dermatome of the IAN, causing pain and functional problems. This is a treatment used successfully for patients with chronic orofacial pain, particularly those with mechanical or cold allodynia of the face.

Recommendations for the treatment of neuropathic pain are reported; Medical guidance is provided by Alonso-Ezpeleta et al.[9] Surgical intervention using direct repair or allograft cadaveric grafts has limited impact on trigeminal neuropathic pain,[376] and other interventions for neuropathic pain have been summarized.[77] Overall, the management of neuropathic pain remains unsatisfactory in that the side effects of medications are significant. Recent reports of Botoxin A and Lidocaine patches may improve the patients' quality of life with a low evidence base, but most importantly, acknowledgement and holistic support for the patient with iatrogenic nerve injuries must not be underestimated, and the prevention of these nerve injuries is paramount.

Cervicofacial subcutaneous emphysema

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Subcutaneous emphysema is defined as the penetration of air or other gases beneath the skin and submucosa, resulting in soft-tissue distention. A specific type, termed *cervicofacial subcutaneous emphysema* (CFSE), is a relatively rare occurrence and may be limited to traumatic, iatrogenic, or spontaneous events.

The first recorded instance was reported by Turnbull in 1900.[342] It details the account of a musician inducing CFSE by playing his bugle immediately after the extraction of a mandibular tooth; the subsequent swelling resolved a few days after he ceased playing the instrument. Indeed, most early reports of CFSE usually follow tooth extractions and were the result of activities the patient engaged in that raised intraoral pressures.[300] With the introduction of air-driven handpieces, there was an increased risk of CFSE.[11,67,370] Although tooth extraction, especially of mandibular third molars, remains the most commonly reported reason for CFSE,[53,67,145,276,366,370] it can also result from restorative treatment,[52,104,158,320,344] periodontal surgery,[99,309] crown and bridge treatments,[116,374] and root canal therapy.[29,149,161,237,323,344,361]

Compressed air, delivered through an air syringe, a high-speed handpiece, or a combination of the two during the operative procedure is associated with 71% of the cases.[139] A more recent study[14] reviewing 47 computed tomography (CT)–documented cases of CFSE found the exclusive cause was the high-speed handpiece in 66% of these patients. Other means of introducing air into the facial spaces include the following:

- Use of the dental laser (Er:YAG) in the gingival pocket: compressed air is used to cool the tip but is of sufficient pressure to dissect the sulcular wound.[208]
- Hydrogen peroxide beyond the canal space: most often reported when the fluid is introduced through a perforation in the canal[29] or overinstrumented apex.[161]
- Lack of rubber dam isolation: a compressed air syringe was implemented to increase visibility in these molar retreatments, with air infiltration through the gingival sulcus.[76,169]
- Barotrauma secondary to Boerhaave syndrome: spontaneous rupture of the distal esophagus as a result of intractable vomiting, causing an increase in intraluminal esophageal pressure.[123]

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The deposition of air beyond the canal space is a function of both the diameter of the canal at the apex[81] and the position of the syringe tip.[307] An air emphysema has the potential to spread along the facial planes in a similar manner (Figs. 20.46 and 20.47). Air entering the parapharyngeal and retropharyngeal spaces can lead to soft-tissue infections,[91] airway compromise,[263] optic nerve damage,[41] and even death.[68,264] The majority of the cases, however, present as a soft, skin-colored swelling without redness that occurs during or shortly after the dental appointment. The cervicofacial swelling is always associated with crepitus (a crunchy feeling to the tissues upon palpation), which may not be palpable before a latency period of several hours. The more serious presentation of pneumomediastinum is characterized by dyspnea with a brassy voice, chest or back pain, and Hamman sign (a bubbling or crunching sound caused by air or movement accompanied by cardiac pulsation).[130] Secondary complications such as pyogenic mediastinitis or necrotizing fasciitis must be considered, and appropriate means taken to evaluate and manage them (blood cultures, broad-spectrum intravenous antibiotics).[76] Differential diagnosis should include an allergic reaction (more pronounced dermal signs prior to cardiac/respiratory symptoms), hematoma (a rapid accumulation of fluid without initial discoloration and without crepitation), and angioneurotic edema (a defined, circumscribed area of edema, often preceded by a burning sensation in the mucous membranes or skin).[99]

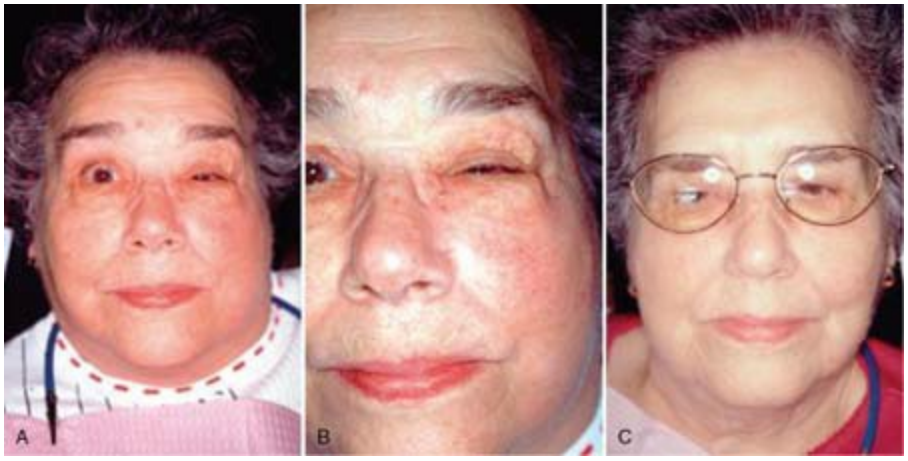

FIG. 20.46 **A,** Facial presentation after the rubber dam was removed. **B,** Close-up view reveals marked distention of the left suborbital region,

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with ptosis of the eye and loss of the nasolabial fold. There was marked crepitus upon palpation, but the patient was pain-free. **C,** She was placed on an antibiotic regime, as per the protocol, 2 days after the incident, the emphysema has resolved and the tissues are normal in color and texture.

A) Close-up view shows the front view of face with swollen under eye and soft tissue on left eye lid.

B) Close-up view shows the left side of patient's face with swollen under eye and soft tissue on left eye lid.

C) Close-up view shows the normal left eye of patient.

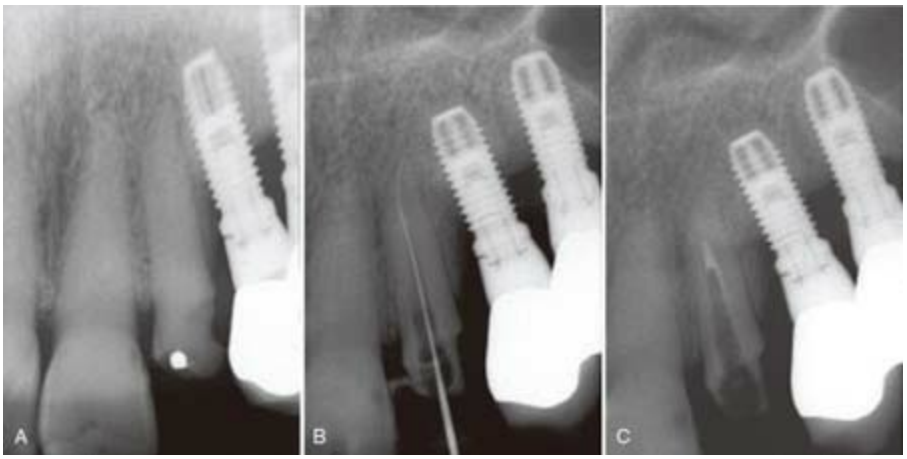

FIG. 20.47 **A,** Preoperative radiograph tooth #10[158]; dystrophic calcification has obliterated the pulp chamber and the coronal half of the canal space. **B,** A #10 file inserted to estimated working length; the file is actually through a facial perforation of the root at the radicular extent of the ultrasonic excavation. Repeated drying of the canal space using an air syringe forced air through the perforation site and precipitated the emphysema seen in Fig. 20.26, *A* and *B*. **C,** The radicular perforation site was sealed with mineral trioxide aggregate (MTA) before the canal space was obturated. The tooth has remained asymptomatic since the incident.

Three radiographs are marked A through C.

A) The central tooth has a radiopaque circular spot and broken edge on one side of the crown.

B) The tooth has radiolucent patch extending from the crown to below the chamber and a file is properly inserted into the tooth.

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C) The tooth has radiolucent crown and root canal. The root tip is filled.

Treatment is largely empirical and is dependent on the extent of the CFSE. Antibiotics are usually prescribed due to the nonsterility of the air supply and the possibility of necrotic debris or microorganisms forced into surrounding tissues.[263] Administration of 100% oxygen via a non-rebreather mask is reported to hasten the resolution of the emphysema because the oxygen, which replaces the trapped air, is more readily absorbed at the site.[169] More severe presentations warrant hospitalization with intravenous antibiotics, where the extent and regression of the CFSE can be closely monitored using CT scans and complications can be managed more effectively.[36] Resolution, barring any severe complications, is characteristically rapid and uneventful, with the patients returning to their normal appearance within 4 to 7 days. A reevaluation of the cause of the emphysema should be examined and corrected before treatment resumes, especially in instances of a perforation.

From an endodontic perspective, minimizing compressed air at the site, from either a high-speed handpiece or an air syringe, is key to preventing this consequence. Isolation with a rubber dam, the use of paper points or high-volume aspiration for drying canal spaces, and avoiding the use of hydrogen peroxide during the procedure will also reduce the risk of introducing or entrapping air beneath the overlying tissues. For endodontic microsurgery, the use of a rear-exhausting high-speed handpiece for root resection as well as ultrasonics for root end preparation would decrease the likelihood of precipitating a CFSE during root end procedures.[19]

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References

1. Abbasipour F, Rastqar A, Bakhtiar H, et al: *The nociceptive and anti-nociceptive effects of white mineral trioxide aggregate*, Int Endod J 2009;42: 794- doi:19549151.
2. Ahlgren FK, Johannessen AC, Hellem S. *Displaced calcium hydroxide paste causing inferior alveolar nerve paraesthesia: report of a case*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 734- doi:14676765.
3. Ahonen M, Tjaderhane L. *Endodontic-related paresthesia: a case report and literature review*, J Endod 2011;37: 1460- doi:21924203.
4. Alapati SB, Brantley WA, Svec TA, et al: *SEM observations of nickel-titanium rotary endodontic instruments that fractured during clinical use*, J Endod 2005;31: 40- doi:15614004.
5. Albrecht LJ, Baumgartner JC, Marshall JG. *Evaluation of apical radicular debris removal using various sizes and tapers of ProFile GT files*, J Endod 2004;30: 425- doi:15167472.
6. Al-Fouzan KS. *Incidence of rotary ProFile instrument fracture and the potential for bypassing in vivo*, Int Endod J 2003;36: 864- doi:14641426.
7. Al-Omari MA, Dummer PM. *Canal blockage and debris extrusion with eight preparation techniques*, J Endod 1995;21: 154- doi:7561660.
8. Alomairy KH. *Evaluating two techniques on removal of fractured rotary nickel-titanium endodontic instruments from root canals: an in vitro study*, J Endod 2009;35: 559- 19345805.

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www.konkur.in

9. Alonso-Ezpeleta O, Martín PJ, López-López J, et al: *Pregabalin in the treatment of inferior alveolar nerve paraesthesia following overfilling of endodontic sealer*, J Clin Exp Dent 2014;6: e197- e202.
10. American Association of Endodontists. Glossary of endodontic terms ed 7 2003; American Association of Endodontists Chicago.
11. American Association of Oral and Maxillofacial Surgeons, Council on Dental Materials, Instruments and Equipment. *Air-driven handpieces and air emphysema*, J Am Dent Assoc 1992;123: 108.
12. Aminoshariae A, Kulild JC, Syed A. *Cone-beam computed tomography compared with intraoral radiographic lesions in endodontic outcome studies: a systematic review*, J Endod 2018;44: 1626-1631.
13. Aminoshariae A, Su A, Kulild JC. *Determination of the location of the mental foramen: a critical review*, J Endod 2014;40: 471-475.
14. Arai I, Aoki T, Yamazaki H, et al: *Pneumomediastinum and subcutaneous emphysema after dental extraction detected incidentally by regular medical checkup: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107: e33- doi:19201622.
15. Augsburge RA, Peters DD. *Radiographic evaluation of extruded obturation materials*, J Endod 1990;16: 492- doi:2084203.
16. Azim AA, Wang HH, Tarrosh M, et al: *Comparison between single-file rotary systems: part 1—efficiency, effectiveness, and adverse effects in endodontic retreatment*, J Endod 2018;44: 1720.
17. Bartling R, Freeman K, Kraut RA. *The incidence of altered sensation of the mental nerve after mandibular*

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www.konkur.in

implant placement, J Oral Maxillofac Surg 1999;57: 1408- doi:10596660.

18. Bataineh AB. *Sensory nerve impairment following mandibular third molar surgery*, J Oral Maxillofac Surg 2001;59: 1012- discussion 7. doi:11526568.
19. Battrum DE, Gutmann JL. *Implications, prevention and management of subcutaneous emphysema during endodontic treatment*, Endod Dent Traumatol 1995;11: 109- doi:7641625.
20. Baumgartner JC, Mader CL. *A scanning electron microscopic evaluation of four root canal irrigation regimens*, J Endod 1987;13: 147- doi:3106553.
21. Beck-Mannagetta J, Necek D, Grasserbauer M. *Solitary aspergillosis of maxillary sinus, a complication of dental treatment*, Lancet 1983;2: 1260.
22. Beck-Mannagetta J, Necek D, Grasserbauer M. *Zahnaerztliche Aspekte der solitaeren Kieferhoehlen-Aspergillose*, Z Stomatol 1986;83: 283- doi:3465133.
23. Becker GL, Cohen S, Borer R. *The sequelae of accidentally injecting sodium hypochlorite beyond the root apex: report of a case*, Oral Surg Oral Med Oral Pathol 1974;38: 633- doi:4547330.
24. Becking AG. *Complications in the use of sodium hypochlorite during endodontic treatment: report of three cases*, Oral Surg Oral Med Oral Pathol 1991;71: 346- doi:2011360.
25. Benninger MS, Sebek BA, Levine HL. *Mucosal regeneration of the maxillary sinus after surgery*, Otolaryngol Head Neck Surg 1989;101: 33- doi:2502761.
26. Bergenholtz G, Lekholm U, Milthon R, et al: *Influence of apical overinstrumentation and overfilling on re-treated root canals*, J Endod 1979;5: 310- doi:297752.

Telegram: @dental_k

www.konkur.in

27. Berutti E, Negro AR, Lendini M, et al: *Influence of manual preflaring and torque on the failure rate of ProTaper rotary instruments*, J Endod 2004;30: 228-230.
28. Betti LV, Bramante CM. *Quantec SC rotary instruments versus hand files for gutta-percha removal in root canal retreatment*, Int Endod J 2001;34: 514- doi:11601768.
29. Bhat KS. *Tissue emphysema caused by hydrogen peroxide*, Oral Surg Oral Med Oral Pathol 1974;38: 304- doi:4528827.
30. Bjørndal L, Amaloo C, Markvart M, et al: *Maxillary sinus impaction of a core carrier causing sustained apical periodontitis, sinusitis, and nasal stenosis: a 3-year follow-up*, J Endod 2016;42: 1851.
31. Blanas N, Kienle F, Sandor GK. *Inferior alveolar nerve injury caused by thermoplastic gutta-percha overextension*, J Can Dent Assoc 2004;70: 384- doi:15175118.
32. Bornstein MM, Lauber R, Sendi P, et al: *Comparison of periapical radiography and limited cone-beam computed tomography in mandibular molars for analysis of anatomical landmarks before apical surgery*, J Endod 2011;37: 151- doi:21238794.
33. Bornstein MM, Wasmer J, Sendi P, et al: *Characteristics and dimensions of the Schneiderian membrane and apical bone in maxillary molars referred for apical surgery: a comparative radiographic analysis using limited cone beam computed tomography*, J Endod 2012;38: 51- doi:22152620.
34. Boutsioukis C, Verhaagen B, Versluis M, et al: *Evaluation of irrigant flow in the root canal using different needle types by an unsteady computational fluid dynamics model*, J Endod 2010;36: 875- doi:20416437.

Telegram: @dental_k

www.konkur.in

35. Bowden JR, Ethunandan M, Brennan PA. *Life-threatening airway obstruction secondary to hypochlorite extrusion during root canal treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 402- doi:16504876.
36. Breznick DA, Saporito JL. *Iatrogenic retropharyngeal emphysema with impending airway obstruction*, Arch Otolaryngol Head Neck Surg 1989;115: 1367- doi:2803720.
37. Brodin P, Røed A, Aars H, et al: *Neurotoxic effects of root filling materials on rat phrenic nerve in vitro*, J Dent Res 1982;61: 1020-1023.
38. Brown DC, Moore BK, Brown CE, et al: *An in vitro study of apical extrusion of sodium hypochlorite during endodontic canal preparation*, J Endod 1995;12: 587.
39. Bryant ST, Thompson SA, al-Omari MA, et al: *Shaping ability of ProFile rotary nickel-titanium instruments with ISO sized tips in simulated root canals: part 1*, Int Endod J 1998;31: 275- doi:9823117.
40. Buckley M, Sprangberg LSW. *The prevalence and technical quality of endodontic treatment in an American subpopulation*, Oral Pathol Oral Radiol Endod 1995;79: 92.
41. Buckley MJ, Turvey TA, Schumann SP, et al: *Orbital emphysema causing vision loss after a dental extraction*, J Am Dent Assoc 1990;120: 421- discussion. doi:4232181013.
42. Budd JC, Gekelman D, White JM. *Temperature rise of the post and on the root surface during ultrasonic post removal*, Int Endod J 2005;38: 705- doi:16164684.
43. Budd CS, Weller RN, Kulild JC. *A comparison of thermoplasticized injectable gutta-percha obturation techniques*, J Endod 1991;17: 260- doi:1940750.

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www.konkur.in

44. Bürklein S, Schäfer E. *Apically extruded debris with reciprocating single-file and full-sequence rotary instrumentation systems*, J Endod 2012;38: 850-852.
45. Bürklein S, Benten S, Schäfer E. *Quantitative evaluation of apically extruded debris with different single-file systems: Reciproc, F360 and OneShape versus Mtwo*, Int Endod J 2014;47: 405-409.
46. Caissie R, Goulet J, Fortin M, et al: *Iatrogenic paresthesia in the third division of the trigeminal nerve: 12 years of clinical experience*, J Can Dent Assoc 2005;71: 185- doi:15763037.
47. Candeiro GTM, Correia FC, Duarte MAH, et al: *Evaluation of radiopacity, ph, release of calcium ions, and flow of a bioceramic root canal Sealer*, J Endod 2012;38: 842- doi:22595123.
48. Caputo BV, Noro Filho GA, de Andrade Salgado DM, et al: *Evaluation of the root canal morphology of molars by using cone-beam computed tomography in a Brazilian population: part I*, J Endod 2016;42: 1604-1607.
49. Carter E, Yilmaz Z, Devine M, et al: *An update on the causes, assessment and management of third division sensory trigeminal neuropathies*, Br Dent J 2016;220: 627-635.
50. Cavenago BC, Ordinola-Zapata R, Duarte MAH, et al: *Efficacy of xylene and passive ultrasonic irrigation on remaining root filling material during retreatment of anatomically complex teeth*, Int Endod J 2014;47: 1078-1083.
51. Caviedes-Bucheli J, Castellanos F, Vasquez N, et al: *The influence of two reciprocating single-file and two rotary-file systems on the apical extrusion of debris and its biological relationship with symptomatic apical*

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www.konkur.in

periodontitis. A systematic review and meta-analysis, Int Endod J 2016;49: 255-270.

52. Chan DC, Myers T, Sharaway M. *A case for rubber dam application—subcutaneous emphysema after class V procedure*, Oper Dent 2007;32: 193- doi:17427830.
53. Chen SC, Lin FY, Chang KJ. *Subcutaneous emphysema and pneumomediastinum after dental extraction*, Am J Emerg Med 1999;17: 678- doi:10597088.
54. Chenail BL, Teplitsky PE. *Orthograde ultrasonic retrieval of root canal obstructions*, J Endod 1987;13: 186- doi:3471842.
55. Cheung GS, Peng B, Bian Z, et al: *Defects in ProTaper S1 instruments after clinical use: fractographic examination*, Int Endod J 2005;38: 802- doi:16218972.
56. Chutich MJ, Kaminski EJ, Miller DA, et al: *Risk assessment of the toxicity of solvents of gutta-percha used in endodontic retreatment*, J Endod 1998;24: 213- doi:9641120.
57. Cintra LTA, Benetti F, de Azevedo Queiroz ÍO, et al: *Cytotoxicity, biocompatibility, and biomineralization of the new high-plasticity MTA material*, J Endod 2017;43: 774.
58. Clegg MS, Vertucci FJ, Walker C, et al: *The effect of exposure to irrigant solutions on apical dentin biofilms in vitro*, J Endod 2006;32: 434- doi:16631843.
59. Cohen S, Burns RC. Pathways of the pulp ed 3 1984; CV Mosby St. Louis 291-292.
60. Cohen S, Burns RC. Pathways of the pulp ed 5 1991; Mosby St. Louis.
61. Cohen S, Burns RC. Pathways of the pulp ed 8 2002; Mosby St Louis 94, 242-252 530, 870, 910.
62. Cohen S, Hargreaves KM. Pathways of the pulp ed 9

Telegram: @dental_k

www.konkur.in

2006; Mosby St Louis 992-994.

63. Crump MC, Natkin E. *Relationship of broken root canal instruments to endodontic case prognosis: a clinical investigation*, J Am Dent Assoc 1970;80: 1341- doi:5266127.
64. Cuje J, Bargholz C, Hulsmann M. *The outcome of retained instrument removal in a specialist practice*, Int Endod J 2010;43: 545- doi:20456518.
65. Dan AE, Thygesen TH, Pinholt EM. *Corticosteroid administration in oral and orthognathic surgery: a systematic review of the literature and meta-analysis*, J Oral Maxillofac Surg 2010;68: 2207- doi:20591548.
66. Daugherty DW, Gound TG, Comer TL. *Comparison of fracture rate, deformation rate, and efficiency between rotary endodontic instruments driven at 150 rpm and 350 rpm*, J Endod 2001;27: 93- doi:11491646.
67. Davies DE. *Pneumomediastinum after dental surgery*, Anaesth Intensive Care 2001;29: 638- doi:11771612.
68. Davies JM, Campbell LA. *Fatal air embolism during dental implant surgery: a report of three cases*, Can J Anaesth 1990;37: 112- doi:2295094.
69. De-Deus G, Moreira EJ, Lopes HP, et al: *Extended cyclic fatigue life of F2 Pro-Taper instruments used in reciprocating movement*, Int Endod J 2010;43: 1063- doi:21080616.
70. Dempf R, Hausamen JE. *Lesions of the inferior alveolar nerve arising from endodontic treatment*, Aust Endod J 2000;26: 67- doi:11359285.
71. Denio D, Torabinejad M, Bakland LK. *Anatomical relationship of the mandibular canal to its surrounding structures in mature mandibles*, J Endod 1992;18: 161- doi:1402570.

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www.konkur.in

72. Devine M, Modgill O, Renton T. *Mandibular division trigeminal nerve injuries following primary endodontic treatment. A case series,* Aust Endod J 2017;43: 56-65.
73. Dhingra A, Ruhal N, Miglani A. *Evaluation of single file systems Reciproc, OneShape, and WaveOne using cone beam computed tomography–an in vitro study*, J Clin Diagn Res 2015;9: ZC30.
74. DiVito EE, Rassoulian SA. *Ex vivo scanning electron microscopy evaluation of cleaning efficacy following in vivo endodontic treatment: a report of 2 cases*, Dentistry 2017;7: 419.
75. Dorn S, Garther A. *Case selection and treatment planning*, Cohen S Burns RC Pathways of the pulp ed 7 1998; St Louis Mosby 60.
76. Durukan P, Salt O, Ozkan S, et al: *Cervicofacial emphysema and pneumomediastinum after a high-speed air drill endodontic treatment procedure,* Am J Emerg Med 2012;30: 2095.e3.
77. Dworkin RH, O'Connor AB, Kent J, et al: *International association for the study of pain neuropathic pain special interest group interventional management of neuropathic pain: NeuPSIG recommendations*, Pain 2013;154: 2249-2261.
78. Eberhardt JA, Torabinejad M, Christiansen EL. *A computed tomographic study of the distances between the maxillary sinus floor and the apices of the maxillary posterior teeth*, Oral Surg Oral Med Oral Pathol 1992;73: 345- doi:1545967.
79. Edgar SW, Marshall JG, Baumgartner JC. *The antimicrobial effect of chloroform on Enterococcus faecalis after gutta-percha removal*, J Endod 2006;32: 1185- doi:17174679.

Telegram: @dental_k

www.konkur.in

80. Ehrich DG, Brian JD Jr, Walker WA. *Sodium hypochlorite accident: inadvertent injection into the maxillary sinus*, J Endod 1993;19: 180- doi:8326264.
81. Eleazer PD, Eleazer KR. *Air pressures developed beyond the apex from drying root canals with pressurized air*, J Endod 1998;24: 833- doi:10023265.
82. Elias LA, Yilmaz Z, Smith JG, et al: *PainDETECT: a suitable screening tool for neuropathic pain in patients with painful post-traumatic trigeminal nerve injuries?*, Int J Oral Maxillofac Surg 2014;43: 120-126.
83. Engstrom B, Segerstad L, Ramstrom G, et al: *Correlation of positive cultures with the prognosis for root canal treatment*, Odontol Revy 1964;15: 257-270.
84. Ericson S, Finne K, Persson G. *Results of apicoectomy of maxillary canines, premolars and molars with special reference to oroantral communication as a prognostic factor*, Int J Oral Surg 1974;3: 386- doi:4217314.
85. Eriksson AR, Albrektsson T. *Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit*, J Prosthet Dent 1983;50: 101- doi:6576145.
86. Erisen R, Yucel T, Kucukay S. *Endomethasone root canal filling material in the mandibular canal: a case report*, Oral Surg Oral Med Oral Pathol 1989;68: 343- doi:2771377.
87. Escoda-Francoli J, Canalda-Sahli C, Soler A, et al: *Inferior alveolar nerve damage because of overextended endodontic material: a problem of sealer cement biocompatibility?*, J Endod 2007;33: 1484- doi:18037065.
88. Ettrich CA, Labossiere PE, Pitts DL, et al: *An investigation of the heat induced during ultrasonic post removal*, J Endod 2007;33: 1222- doi:17889694.

Telegram: @dental_k

www.konkur.in

89. Fanibunda K, Whitworth J, Steele J. *The management of thermomechanically compacted gutta percha extrusion in the inferior dental canal*, Br Dent J 1998;184: 330- doi:9599885.
90. Farren ST, Sadoff RS, Penna KJ. *Sodium hypochlorite chemical burn: case report*, N Y State Dent J 2008;74: 61- doi:18402381.
91. Feinstone T. *Infected subcutaneous emphysema: report of case*, J Am Dent Assoc 1971;83: 1309- doi:5286735.
92. Feldman G, Solomon C, Notaro P, et al: *Retrieving broken endodontic instruments*, J Am Dent Assoc 1974;88: 588- doi:4521266.
93. Felippe MC, Felippe WT, Marques MM, et al: *The effect of the renewal of calcium hydroxide paste on the apexification and periapical healing of teeth with incomplete root formation*, Int Endod J 2005;38: 436- doi:15946263.
94. Ferraz CC, Gomes NV, Gomes BP, et al: *Apical extrusion of debris and irrigants using two hand and three engine-driven instrumentation techniques*, Int Endod J 2001;34: 354- doi:11482718.
95. Fishel D, Buchner A, Hershkowith A, et al: *Roentgenologic study of the mental foramen*, Oral Surg Oral Med Oral Pathol 1976;41: 682- doi:1063970.
96. Fors UGH, Berg JO. *A method for the removal of separated endodontic instruments from root canals*, J Endod 1983;9: 156- doi:6574203.
97. Fox J, Moodnik RM, Greenfield E, et al: *Filling root canals with files: radiographic evaluation of 304 cases*, N Y State Dent J 1972;38: 154- doi:4500382.
98. Freedman A, Horowitz I. *Complications after apicoectomy in maxillary premolar and molar teeth*, Int J

Telegram: @dental_k

www.konkur.in

Oral Maxillofac Surg 1989;28: 192.
99. Fruhauf J, Weinke R, Pilger U, et al: *Soft tissue cervicofacial emphysema after dental treatment: report of 2 cases with emphasis on the differential diagnosis of angioedema*, Arch Dermatol 2005;141: 1437- doi:16301391.
100. Fu M, Zhang Z, Hou B. *Removal of broken files from root canals by using ultrasonic techniques combined with dental microscope: a retrospective analysis of treatment outcome*, J Endod 2011;37: 619- doi:21496659.
101. Gambarini G. *Cyclic fatigue of nickel-titanium rotary instruments after clinical use with low-and high-torque endodontic motors*, J Endod 2001;27: 772- doi:11771588.
102. Gambarini G. *Rational for the use of low-torque endodontic motors in root canal instrumentation*, Endod Dent Traumal 2000;16: 95.
103. Gambarini G, Plotino G, Grande NM, et al: *Differential diagnosis of endodonticrelated inferior alveolar nerve paraesthesia with cone beam computed tomography: a case report*, Int Endod J 2011;44: 176- doi:21083573.
104. Gamboa VCA, Vega Pizarro A, Arriagada AA. *Subcutaneous emphysema secondary to dental treatment: case report*, Medicina Oral, Patologia Oral y Cirugia Bucal 2007;12: E76- doi:17195836.
105. Gandolfi MG, Siboni F, Primus CM, et al: *Ion release, porosity, solubility, and bioactivity of MTA plus tricalcium silicate*, J Endod 2014;40: 1632- doi:25260736.
106. Gao Y, Shotton V, Wilkinson K, et al: *Effects of raw material and rotational speed on the cyclic fatigue of ProFile Vortex rotary instruments*, J Endod 2010;36: 1205- doi:20630300.
107. Gatot A, Arbelle J, Leiberman A, et al: *Effects of sodium*

Telegram: @dental_k

www.konkur.in

hypochlorite on soft tissues after its inadvertent injection beyond the root apex, J Endod 1991;17: 573- doi:1812208.

108. George S, Basrani B, Kishen A. *Possibilities of gutta-percha-centered infection in endodontically treated teeth: an in vitro study*, J Endod 2010;36: 1241- doi:20630308.
109. Gernhardt CR, Eppendorf K, Kozlowski A, et al: *Toxicity of concentrated sodium hypochlorite used as an endodontic irrigant*, Int Endod J 2004;37: 272- doi:15056354.
110. Gettleman BH, Spriggs KA, ElDeeb ME, et al: *Removal of canal obstructions with the endo extractor*, J Endod 1991;17: 608- doi:1820424.
111. Giacomino CM, Wealleans JA, Kuhn N, et al: *Comparative biocompatibility and osteogenic potential of two bioceramic sealers*, J Endod 2019;45: 51.
112. Giardino L, Pontieri F, Savoldi E, et al: *Aspergillus mycetoma of the maxillary sinus secondary to overfilling of a root canal*, J Endod 2006;32: 692- doi:16793483.
113. Giuliani M, Lajolo C, Deli G, et al: *Inferior alveolar nerve paresthesia caused by endodontic pathosis: a case report and review of the literature*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 670- doi:11740484.
114. Givol N, Rosen E, Bjorndal L, et al: *Medico-legal aspects of altered sensation following endodontic treatment: a retrospective case series*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 126-131.
115. Gluskin AH, Ruddle CJ, Zinman EJ. *Thermal injury through intraradicular heat transfer using ultrasonic devices: precautions and practical preventive strategies*, J Am Dent Assoc 2005;136: 1286- doi:16196235.
116. Goorhuis H, Rothrock SG. *Cervicofacial and thoracic*

Telegram: @dental_k

www.konkur.in

barotrauma following a minor dental procedure, Pediatr Emerg Care 1993;9: 29- doi:8488142.

117. Gorduysus M, Avcu N, Gorduysus O, et al: *Cytotoxic effects of four different endodontic materials in human periodontal ligament fibroblasts*, J Endod 2007;33: 1450- doi:18037057.
118. Greene KJ, Krell KV. *Clinical factors associated with ledged canals in maxillary and mandibular molars*, Oral Surg Oral Med Oral Pathol 1990;70: 490- doi:2216387.
119. Greenstein G, Tarnow D. *The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review*, J Periodontol 2006;77: 1933- doi:17209776.
120. Grigorin D, Brambule J, Delacretaz J. *La sinusite maxillaire fungique*, Dermatologica 1979;159: 180.
121. Grötz KA, Al-Nawas B, de Aguiar EG, et al: *Treatment of injuries to the inferior alveolar nerve after endodontic procedures*, Clin Oral Investig 1998;2: 73-76.
122. Guerreiro-Tanomaru JM, De Faria-Jnior NB, Duarte MAH, et al: *Comparative analysis of enterococcus faecalis biofilm formation on different substrates*, J Endod 2013;39: 346- doi:23402505.
123. Gulati A, Baldwin A, Intosh IM, et al: *Pneumomediastinum, bilateral pneumothorax, pleural effusion, and surgical emphysema after routine apicectomy caused by vomiting*, Br J Oral Maxillofac Surg 2008;46: 136- doi:17257717.
124. Gursoy UK, Bostanci V, Kosger HH. *Palatal mucosa necrosis because of accidental sodium hypochlorite injection instead of anaesthetic solution*, International Endod J 2006;39: 157.
125. Gutmann JL, Dumsha TC, Lovdahl PE, et al: Problem

Telegram: @dental_k

www.konkur.in

solving in endodontics ed 3 1997; Mosby St. Louis 96-100 117.

126. Gutmann JL, Rakusin H. *Perspectives on root canal obturation with thermoplasticized injectable gutta-percha*, Int Endod J 1987;20: 261- doi:3481786.
127. Haikel Y, Serfaty R, Bateman G, et al: *Dynamic and cyclic fatigue of engine-driven rotary nickel-titanium endodontic instruments*, J Endod 1999;25: 434- doi:10530246.
128. Hamid HR, Gluskin AH, Peters OA, et al: *Rotary versus reciprocation root canal preparation: initial clinical quality assessment in a novice clinician cohort*, J Endod 2018;44: 1257.
129. Hammad M, Qualtrough A, Silikas N. *Three-dimensional evaluation of effectiveness of hand and rotary instrumentation for retreatment of canals filled with different materials*, J Endod 2008;34: 1370- doi:18928849.
130. Hamman L. *Spontaneous mediastinal emphysema*, Bull Johns Hopkins Hosp 1961;54: 46.
131. Handschel J, Figgener L, Joos U. *[Forensic evaluation of injuries to nerves and jaw bone after wisdom tooth extraction from the viewpoint of current jurisprudence]*, Mund Kiefer Gesichtschir 2001;5: 44- doi:11272387.
132. Hapcook CP Sr. *Dental malpractice claims: percentages and procedures*, J Am Dent Assoc 2006;137: 1444- doi:17012726.
133. Harty FJ, Parkins BJ, Wengraf AM. *Success rate in root canal therapy: a retrospective study of conventional cases*, Br Dent J 1970;128: 65- doi:5270224.
134. Hashem AA. *Ultrasonic vibration: temperature rise on external root surface during broken instrument removal*, J Endod 2007;33: 1070- doi:17931935.

Telegram: @dental_k

www.konkur.in

135. Hasheminia SM, Farhad A, Sheikhi M, et al: *Cone-beam computed tomographic analysis of canal transportation and centering ability of single-file systems*, J Endod 2018;44: 1788-1791.
136. Hauman CH, Chandler NP, Tong DC. *Endodontic implications of the maxillary sinus: a review*, Int Endod J 2002;35: 127- doi:11843967.
137. Heasman PA. *Variation in the position of the inferior dental canal and its significance to restorative dentistry*, J Dent 1988;16: 36- doi:3164005.
138. Heling I, Rotstein I, Dinur T, et al: *Bactericidal and cytotoxic effects of sodium hypochlorite and sodium dichloroisocyanurate solutions in vitro*, J Endod 2001;27: 278- doi:11485267.
139. Heyman SN, Babayof I. *Emphysematous complications in dentistry, 1960-1993: an illustrative case and review of the literature*, Quintessence Int 1995;26: 535- doi:8602428.
140. Hillerup S. *Iatrogenic injury to oral branches of the trigeminal nerve: records of 449 cases*, Clin Oral Investig 2007;11: 133- doi:17186310.
141. Hillerup S. *Iatrogenic injury to the inferior alveolar nerve: etiology, signs and symptoms, and observations on recovery*, Int J Oral Maxillofac Surg 2008;37: 704- doi:18501561.
142. Hillerup S, Jensen R. *Nerve injury caused by mandibular block analgesia*, Int J Oral Maxillofac Surg 2006;35: 437- doi:16343853.
143. Himel V, Ahmed K, Wood D, et al: *An evaluation of nitinol and stainless steel files used by students during a laboratory proficiency exam*, Oral Surg 1995;79: 232.
144. Hinrichs RE, Walker WA, Schindler WG. *A comparison*

Telegram: @dental_k

www.konkur.in

of amounts of apically extruded debris using handpiece-driven nickel-titanium instrument systems, J Endod 1998;24: 102- doi:9641140.

145. Horowitz I, Hirshberg A, Freedman A. *Pneumomediastinum and subcutaneous emphysema following surgical extraction of mandibular third molars: three case reports*, Oral Surg Oral Med Oral Pathol 1987;63: 25- doi:3543796.
146. Huang X, Ling J, Gu L. *Quantitative evaluation of debris extruded apically by using ProTaper Universal Tulsa Rotare System in endodontic retreatment*, J Endod 2007;33: 1102- doi:17931943.
147. Hülsmann M. *Methods for removing metal obstructions from the root canal*, Endod Dent Traumatol 1993;9: 223- doi:8143573.
148. Hülsmann M. *Removal of fractured root canal instruments using the canal finder system*, Dtsch Zahnarztl Z 1990;45: 229- doi:2257833.
149. Hülsmann M, Hahn W. *Complications during root canal irrigation: literature review and case reports*, Int Endod J 2000;33: 186- doi:11307434.
150. Hülsmann M, Peters OA, Dummer PMH. *Mechanical preparation of root canals: shaping goals, techniques and means*, Endod Topics 2005;10: 30.
151. Hülsmann M, Stotz S. *Efficacy, cleaning ability and safety of different devices for gutta-percha removal in root canal retreatment*, Int Endod J 1997;30: 227- doi:9477808.
152. Ingle JI. PDQ endodontics 2005; BC Decker London 220.
153. Ingle JI, Bakland LK: *Endodontics*, ed 5, London, 2002, BC Decker, pp 412, 482–489, 525–538, 695, 729, 769, 776.
154. Ingle JI, Luebke RG, Zidell JD, et al: *Obturation of the*

Telegram: @dental_k

www.konkur.in

radicular space, Ingle JI Taintor JF Endodontics ed 3 1985; Lea & Febiger Philadelphia 223-307.

154a *International Classification of Orofacial Pain (ICOP)*, Cephalalgia 2020;40: 129-221.

155. Ioannides C, Borstlap WA. *Apicoectomy on molars: a clinical and radiographical study*, Int J Oral Surg 1983;12: 73- doi:6409827.

156. Jafarzadeh H, Abbott PV. *Ledge formation: review of a great challenge in endodontics*, J Endod 2007;33: 1155- doi:17889681.

157. Jiang LM, Verhaagen B, Versluis M, et al: *The influence of the orientation of an ultrasonic file on the cleaning efficacy of ultrasonic activated irrigation*, J Endod 2010;36: 1372- doi:20647099.

158. Josephson GD, Wambach BA, Noordzji JP. *Subcutaneous cervicofacial and mediastinal emphysema after dental instrumentation*, Otolaryngol Head Neck Surg 2001;124: 170- doi:11226951.

159. Kapalas A, Lambrianidis T. *Factors associated with root canal ledging during instrumentation*, Endod Dent Traumatol 2000;16: 229- doi:11202887.

160. Karabucak B, Bunes A, Chehoud C, et al: *Prevalence of apical periodontitis in endodontically treated premolars and molars with untreated canal: a cone-beam computed tomography study*, J Endod 2016;42: 538-541.

161. Kaufman AY. *Facial emphysema caused by hydrogen peroxide irrigation: report of a case*, J Endod 1981;7: 470- doi:6945389.

162. Kekere-Ekun TA. *Antero-posterior location of the mental foramen in Nigerians*, Afr Dent J 1989;3: 2- doi:2640130.

163. Kerbl FM, DeVilliers P, Litaker M, et al: *Physical effects of sodium hypochlorite on bone: an ex vivo study*, J Endod

Telegram: @dental_k

www.konkur.in

2012;38: 357- doi:22341074.

164. Kfir A, Tsesis I, Yakirevich E, et al: *The efficacy of five techniques for removing root filling material: microscopic versus radiographic evaluation*, Int Endod J 2012;45: 35- doi:21899565.
165. Khademi A, Yazdizadeh M, Feizianfard M. *Determination of the minimum instrumentation size for penetration of irrigants to the apical third of root canal systems*, J Endod 2006;32: 417- doi:16631839.
166. Khongkhunthian P, Reichart PA. *Aspergillosis of the maxillary sinus as a complication of overfilling root canal material into the sinus: report of two cases*, J Endod 2001;27: 476- doi:11504001.
167. Kim EC, Lee BC, Chang HS, et al: *Evaluation of the radiopacity and cytotoxicity of Portland cements containing bismuth oxide*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: e54- doi:18155604.
168. Kim JY, Cheung GS, Park SH, et al: *Effect from cyclic fatigue of nickel-titanium rotary files on torsional resistance*, J Endod 2012;38: 527- doi:22414843.
169. Kim Y, Kim MR, Kim SJ. *Iatrogenic pneumomediastinum with extensive subcutaneous emphysema after endodontic treatment: report of 2 cases*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: e114- doi:19969477.
170. Klasser GD, Kugelmann AM, Villines D, et al: *The prevalence of persistent pain after nonsurgical root canal treatment*, Quintessence Int 2011;42: 259-269.
171. Kleier DJ, Averbach RE, Mehdipour O. *The sodium hypochlorite accident: experience of diplomates of the American Board of Endodontics*, J Endod 2008;34: 1346- doi:18928844.

Telegram: @dental_k

www.konkur.in

172. Knowles KI, Jergenson MA, Howard JH. *Paresthesia associated with endodontic treatment of mandibular premolars*, J Endod 2003;29: 768- doi:14651287.
173. Kobayashi A. *Asymptomatic aspergillosis of the maxillary sinus associated with foreign body of endodontic origin: report of a case*, Int J Oral Maxillofac Surg 1995;24: 243- doi:7594762.
174. Koch K. *The microscope: its effect on your practice*, Dent Clin North Am 1997;41: 619- doi:9248695.
175. Kopp W, Fotter R, Steiner H, et al: *Aspergillosis of the paranasal sinuses*, Radiology 1985;156: 715- doi:4023231.
176. Köseoğlu BG, Tanrikulu S, Sübay RK, et al: *Anesthesia following overfilling of a root canal sealer into the mandibular canal: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 803-806.
177. Kosti E, Zinelis S, Lambrianidis T, et al: *A comparative study of crack development in stainless-steel Hedström files used with step-back or crown-down techniques*, J Endod 2004;30: 38- doi:14760906.
178. Kothari P, Hanson N, Cannell H. *Bilateral mandibular nerve damage following root canal therapy*, Br Dent J 1996;180: 189- doi:8867623.
179. Kramkowski TR, Bahcall J. *An in vitro comparison of torsional stress and cyclic fatigue resistance of ProFile GT and ProFile GT Series X rotary nickel-titanium files*, J Endod 2009;35: 404- doi:19249605.
180. Krasner P, Rankow HJ. *Anatomy of the pulp-chamber floor*, J Endod 2004;30: 5- doi:14760900.
181. Kremeier K, Pontius O, Klaiber B, et al: *Nonsurgical endodontic management of a double tooth: a case report*, Int Endod J 2007;40: 908- doi:17935498.

Telegram: @dental_k

www.konkur.in

182. Krishnan U, Monsour P, Thaha K, et al: *A limited field cone-beam computed tomography-based evaluation of the mental foramen, accessory mental foramina, anterior loop, lateral lingual foramen, and lateral lingual canal*, J Endod 2018;44: 946-951.
183. Kulild JC, Peters DD. *Incidence and configuration of canal systems in the mesiobuccal root of maxillary first and second molars*, J Endod 1990;16: 311- doi:2081944.
184. Kytridou V, Gutmann JL, Nunn MH. *Adaptation and sealability of two contemporary obturation techniques in the absence of the dentinal smear layer*, Int Endod J 1999;32: 464- doi:10709495.
185. Lambrianidis T. Ledge formation, Iatrogenic complications during endodontic treatment 1996; Univ Studio Press Thessaloniki, Greece.
186. Lee J, Lorenzo D, Rawlins T, et al: *Sodium hypochlorite extrusion: an atypical case of massive soft tissue necrosis*, J Oral Maxillofac Surg 2011;69: 1776- doi:21211884.
187. Lee JS, Lee JH, Lee JH, et al: *Efficacy of early treatment with infliximab in pediatric Crohn's disease*, World J Gastroenterol 2010;16: 1776- doi:20380012.
188. Legent F, Billet J, Beauvillain C, et al: *The role of dental canal fillings in the development of aspergillus sinusitis: a report of 85 cases*, Arch Otorhinolaryngol 1989;246: 318- doi:2686596.
189. Levy G. *[Canal finder system 89: improvements and indications after 4 years of experimentation and use]*, Rev Odontostomatol (Paris) 1990;19: 327- doi:2237106.
190. Lin L, Chance K, Shovlin F, et al: *Oroantral communication in periapical surgery of maxillary posterior teeth*, J Endod 1985;11: 40- doi:3855960.
191. Ling JQ, Wei X, Gao Y. *Evaluation of the use of dental*

Telegram: @dental_k

www.konkur.in

operating microscope and ultrasonic instruments in the management of blocked canals, Zhonghua Kou Qiang Yi Xue Za Zhi 2003;38: 324- review article 1162. doi:14680574.

192. Liu R, Kaiwar A, Shemesh H, et al: *Incidence of apical root cracks and apical dentinal detachments after canal preparation with hand and rotary files at different instrumentation lengths*, J Endod 2013;39: 129- doi:23228272.
193. Loidolt D, Mangge H, Wilders-Trushing M, et al: *In vivo and in vitro suppression of lymphocyte function in aspergillus sinusitis*, Arch Otorhinolaryngol 1989;246: 321- doi:2686597.
194. Lopes HP, Ferreira AA, Elias CN, et al: *Influence of rotational speed on the cyclic fatigue of rotary nickel-titanium endodontic instruments*, J Endod 2009;35: 1013- doi:19567325.
195. Lopes HP, Moreira EJ, Elias CN, et al: *Cyclic fatigue of ProTaper instruments*, J Endod 2007;33: 55- doi:17185132.
196. Lovato KF, Sedgley CM. *Antibacterial activity of EndoSequence Root Repair Material and ProRoot MTA against clinical isolates of Enterococcus faecalis*, J Endod 2011;37: 1542- doi:22000459.
197. Low KM, Dula K, Bürgin W, et al: *Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery*, J Endod 2008;34: 557.

197a Lukáš R, Zýková I, Barsa P, et al: *Soucˇasný pohled na užívání methylprednisolonu v lécˇbeˇ akutního poškození míchy [Current role of methylprednisolone in the treatment of acute spinal cord injury]*, Acta Chir Orthop

Telegram: @dental_k

www.konkur.in

Traumatol Cech 2011;78: 305.
198. Ma J, Al-Ashaw AJ, Shen Y, et al: *Efficacy of ProTaper Universal Rotary Retreatment System for gutta-percha removal from oval root canals: a micro-computed tomography study*, J Endod 2012;38: 1516- doi:23063227.
199. Madarati A, Watts DC, Qualtrough AE. *Opinions and attitudes of endodontists and general dental practitioners in the UK towards the intracanal fracture of endodontic instruments: part 2*, Int Endod J 2008;41: 1079- doi:19133097.
200. Madarati AA, Qualtrough AJ, Watts DC. *Efficiency of a newly designed ultrasonic unit and tips in reducing temperature rise on root surface during the removal of fractured files*, J Endod 2009;35: 896- doi:19482194.
201. Madarati AA, Hunter MJ, Dummer PMH. *Management of intracanal separated instruments*, J Endod 2013;39: 569- doi:23611371.
202. Maia Filho M, de Castro Rizzi C, Bandeca Coelho M, et al: *Shaping ability of Reciproc, UnicOne, and ProTaper Universal in simulated root canals*, ScientificWorldJournal 2015;2015: 690854.
203. Manisal Y, Yücel T, Eriüen R. *Overfilling of the root*, Oral Surg Oral Med Oral Pathol 1989;68: 773- doi:2594328.
204. McDonald MN, Vire DE. *Chloroform in the endodontic operatory*, J Endod 1992;18: 301- doi:1402589.
205. McKendry DJ. *Comparison of balanced forces, endosonic and step-back filing instrumentation techniques: quantification of extruded apical debris*, J Endod 1990;16: 24- doi:2388013.
206. Mehdipour O, Kleier DJ, Averbach RE. *Anatomy of sodium hypochlorite accidents*, Compend Contin Educ

Telegram: @dental_k

www.konkur.in

Dent 2007;28: 544- doi:548, 550 18018389.
207. Metzger Z, Ben-Amar A. *Removal of overextended gutta-percha root canal fillings in endodontic failure cases*, J Endod 1995;21: 287- doi:7673835.
208. Mitsunaga S, Iwai T, Aoki N, et al: *Cervicofacial subcutaneous and mediastinal emphysema caused by air cooling spray of dental laser*, Oral Surg Oral Med Oral Pathol Oral Radiol 2013;115: e13- doi:22762919.
209. Molander A, Reit C, Dahen G, et al: *Microbiological status of root-filled teeth with apical periodontitis*, Int Endod J 1998;31: 1- doi:9823122.
210. Morse DR. *Endodontic-related inferior alveolar nerve and mental foramen paresthesia*, Compend Contin Educ Dent 1997;18: 963- doi:9533307.
211. Naenni N, Thoma K, Zehnder M. *Soft tissue dissolution capacity of currently used and potential endodontic irrigants*, J Endod 2004;30: 785- doi:15505511.
212. Nagai O, Tani N, Kayaba Y, et al: *Ultrasonic removal of separated instruments in root canals*, Int Endod J 1986;19: 298- doi:3466866.
213. Nair PN. *On the causes of persistent apical periodontitis: a review*, Int Endod J 2006;39: 249- doi:16584489.
214. Nair PNR, Henry S, Cano V, et al: *Microbial status of apical root canal system of human mandibular first molars with primary apicalr periodontitis after "one-visit" endodontic treatment*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99: 231- doi:15660098.
215. Namazikhah MS, Mokhlis HR, Alasmakh K. *Comparison between a hand stainless steel K file and a rotary NiTi 0.04 taper*, J Calif Dent Assoc 2000;28: 421- doi:11324127.
216. Neaverth E. *Disabling complications following*

Telegram: @dental_k

www.konkur.in

inadvertent overextension of a root canal filling material, J Endod 1989;15: 135- doi:2607283.

217. Neiva RF, Gapski R, Wang HL. *Morphometric analysis of implant-related anatomy in Caucasian skulls*, J Periodontol 2004;75: 1061- doi:15455732.
218. Nevares G, Cunha RS, Zuolo ML, et al: *Success rates for removing or bypassing fractured instruments: a prospective clinical study*, J Endod 2012;38: 442- doi:22414826.
219. Ngeow WC. *Is there a "safety zone" in the mandibular premolar region where damage to the mental nerve can be avoided if periapical extrusion occurs?*, J Can Dent Assoc 2010;76: a61- doi:20579448.
220. Ngeow WC, Yuzawati Y. *The location of the mental foramen in a selected Malay population*, J Oral Sci 2003;45: 171- doi:14650583.
221. Niemczyk SP. *Essentials of endodontic microsurgery*, Dent Clin North Am 2010;54: 375- doi:20433983.
222. Nitzan DW, Stabholz A, Azaz B. *Concepts of accidental overfilling and overinstrumentation in the mandibular canal during root canal treatment*, J Endod 1983;9: 81- doi:6590769.
223. Noiri Y, Ehara A, Kawahara T, et al: *Participation of bacterial biofilms in refractory and chronic periapical periodontitis*, J Endod 2002;28: 679- doi:12398163.
224. Oberli K, Bornstein MM, von Arx T. *Periapical surgery and the maxillary sinus: radiographic parameters for clinical outcome*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 848- doi:17197213.
225. Oshima K, Ishii T, Ogura Y, et al: *Clinical investigation of patients who develop neuropathic tooth pain after endodonticprocedures*, J Endod 2009;35: 958-961.

Telegram: @dental_k

www.konkur.in

226. Ozkan BT, Celik S, Durmus E. *Paresthesia of the mental nerve stem from periapical infection of mandibular canine tooth: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105: e28.
227. Özyürek T, Yılmaz K, Uslu G. *Shaping ability of Reciproc, WaveOne GOLD, and HyFlex EDM single-file systems in simulated S-shaped canals*, J Endod 2017;43: 805-809.
228. Pagin O, Centurion BS, Rubira-Bullen LRF, et al: *Maxillary sinus and posterior teeth: accessing close relationship by cone-beam computed tomographic scanning in a Brazilian population*, J Endod 2013;39: 748- doi:23683273.
229. Parashos P, Gordon I, Messer HH. *Factors influencing defects of rotary nickel titanium endodontic instruments after clinical use*, J Endod 2004;30: 722- doi:15448468.
230. Park YT, Kim SG, Moon SY. *Indirect compressive injury to the inferior alveolar nerve caused by dental implant placement*, J Oral Maxillofac Surg 2012;70: e258- e259.
231. Pashley EL, Birdsong NL, Bowman K, et al: *Cytotoxic effects of NaOCl on vital tissue*, J Endod 1985;11: 525- doi:3867719.
232. Patel S, Dawood A, Ford TP, et al: *The potential applications of cone beam computed tomography in the management of endodontic problems*, Int Endod J 2007;40: 818- doi:17697108.
233. Patino PV, Biedma BM, Liebana CR, et al: *The influence of a manual glide path on the separation rate of NiTi rotary instruments*, J Endod 2005;31: 114- doi:15671822.
234. Patiño PV, Biedma BM, Liébana CR, et al: *The influence of a manual glide path on the separation rate of NiTi rotary instruments*, J Endod 2005;31: 114-116.

Telegram: @dental_k

www.konkur.in

235. Pedull E, Grande NM, Plotino G, et al: *Influence of continuous or reciprocating motion on cyclic fatigue resistance of 4 different nickel-titanium rotary instruments*, J Endod 2013;39: 258- doi:23321241.
236. Pelka M, Petschelt A. *Permanent mimic musculature and nerve damage caused by sodium hypochlorite: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: e80- doi:18602848.
237. Penna KJ, Neshat K. *Cervicofacial subcutaneous emphysema after lower root canal therapy*, N Y State Dent J 2001;67: 28.
238. Persson G. *Periapical surgery of molars*, Int J Oral Surg 1982;11: 96- doi:6809679.
239. Petersson K, Petersson A, Olsson B, et al: *Technical quality of root fillings in an adult Swedish population*, Endod Dent Traumatol 1986;2: 99- doi:3460804.
240. Pettiette MT, Conner D, Trope M. *Procedural errors with the use of nickel-titanium rotary instruments in undergraduate endodontics*, J Endod 2002;28: 259.
241. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 1. Size, orientation, and positional relationship to the mandibular second premolar*, J Endod 1990;16: 221- doi:2074415.
242. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 2. Radiographic position in relation to the mandibular second premolar*, J Endod 1992;18: 271- doi:1402584.
243. Phillips JL, Weller RN, Kulild JC. *The mental foramen: 3. Size and position on panoramic radiographs*, J Endod 1992;18: 383- doi:1431694.
244. Plotino G, Grande NM, Mercadé Bellido M, et al: *Influence of temperature on cyclic fatigue resistance of ProTaper Gold and ProTaper universal rotary files*, J

Telegram: @dental_k

www.konkur.in

Endod 2017;43: 200-202.

245. Poggio C, Dagna A, Ceci M, et al: *Solubility and pH of bioceramic root canal sealers: A comparative study*, J Clin Exp Dent 2017;9: 1189-1194.

245a Pogrel A: Unpublished.

246. Pogrel MA. *Damage to the inferior alveolar nerve as the result of root canal therapy*, J Am Dent Assoc 2007;138: 65- doi:17197403.

247. Pogrel MA, Le H. *Etiology of lingual nerve injuries in the third molar region: a cadaver and histologic study*, J Oral Maxillofac Surg 2006;64: 1790- doi:17113447.

248. Pogrel MA, Thamby S. *Permanent nerve involvement resulting from inferior alveolar nerve blocks*, J Am Dent Assoc 2000;131: 901- doi:10916328.

249. Poveda R, Bagan JV, Diaz Fernandez JM, et al: *Mental nerve paresthesia associated with endodontic paste within the mandibular canal: report of a case*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: e46- doi:17052625.

250. Powell SE, Wong PD, Simon JH. *A comparison of the effect of modified and nonmodified instrument tips on apical canal configuration: part II*, J Endod 1988;14: 224- doi:3251976.

251. Pruett JP, Clement DJ, Carnes DL Jr. *Cyclic fatigue testing of nickel-titanium endodontic instruments*, J Endod 1997;23: 77- doi:9220735.

252. Rahimi M, Parashos P. *A novel technique for the removal of fractured instruments in the apical third of curved root canals*, Int Endod J 2009;42: 264- doi:19228217.

253. Ramirez-Salomon M, Soler-Bientz R, de la Garza-Gonzalez R, et al: *Incidence of lightspeed separation and the potential for bypassing*, J Endod 1997;23: 586-

Telegram: @dental_k

www.konkur.in

doi:9587288.

254. Reddy SA, Hicks ML. *Apical extrusion of debris using two hand and two rotary instrumentation techniques*, J Endod 1998;24: 180- doi:9558583.

255. Reeh ES, Messer HH. *Long-term paresthesia following inadvertent forcing of sodium hypochlorite through perforation in maxillary incisor*, Endod Dent Traumatol 1989;5: 200- doi:2637861.

256. Rehm S, Koroschetz J, Baron R. *An update on neuropathic pain*, Eur Neur Rev 2008;3: 125.

257. Rene N, Owall B. *Dental malpractice in Sweden*, J Law Ethics Dent 1991;4: 16- doi:1816351.

258. Renton T. *Prevention of iatrogenic inferior alveolar nerve injuries in relation to dental procedures*, Dent Update 2010;37: 350, 354-358 passim, doi:20929149.

259. Renton T, Dawood A, Shah A, et al: *Post-implant neuropathy of the trigeminal nerve: a case series*, Br Dent J 2012;212: E17- doi:22677874.

260. Renton T, Thexton A, Crean SJ, et al: *Simplifying the assessment of the recovery from surgical injury to the lingual nerve*, Br Dent J 2006;200: 569-573.

261. Renton T, Yilmaz Z. *Managing iatrogenic trigeminal nerve injury: a case series and review of the literature*, Int J Oral Maxillofac Surg 2012;41: 629- doi:22326447.

262. Renton T, Yilmaz Z. *Profiling of patients presenting with posttraumatic neuropathy of the trigeminal nerve*, J Orofac Pain 2011;25: 333- doi:22247929.

263. Reznick JB, Ardary WC. *Cervicofacial subcutaneous air emphysema after dental extraction*, J Am Dent Assoc 1990;120: 417- doi:2181012.

264. Rickles NH, Joshi BA. *A possible case in a human and an investigation in dogs of death from air embolism during*

Telegram: @dental_k

www.konkur.in

root canal therapy, J Am Dent Assoc 1963;67: 397- doi:14043965.

265. Ricucci D, Langeland K. *Apical limit of root-canal instrumentation and obturation*, Int Endod J 1998;31: 394- doi:15551607.
266. Robinson MJ, McDonald NJ, Mullally PJ. *Apical extrusion of thermoplasticized obturating material in canals instrumented with Profile 0.06 or Profile GT*, J Endod 2004;30: 418- doi:15167470.
267. Roda RS: Personal communication, 2013.
268. Roda RS. *Root perforation repair: surgical and nonsurgical management*, Pract Proced Aesthet Dent 2001;13: 467- quiz 474. doi:11544819.
269. Rödig T, Hausdörfer T, Konietschke F, et al: *Efficacy of D-RaCe and ProTaper Universal Retreatment NiTI instruments and hand files in removing gutta-percha from curved root canals – a micro-computed tomography study*, Int Endod J 2012;54: 580-589.
270. Rödig T, Kupis J, Konietschke F, et al: *Comparison of hand and rotary instrumentation for removing gutta-percha from previously treated curved root canals: a microcomputed tomography study*, Int Endod J 2014;47: 173-182.
271. Rödig T, Reicherts P, Konietschke F, et al: *Efficacy of reciprocating and rotary NiTi instruments for retreatment of curved root canals assessed by micro-CT*, Int Endod J 2014;47: 942-948.
272. Rodriguez et al. *Influence of CBCT on Endodotic retreatment strategies among general dental practitioners and endodontists*, J Endod 2017;43: 1433.
273. Rosen E, Goldberger T, Taschieri S, et al: *The prognosis of altered sensation after extrusion of root canal filling*

Telegram: @dental_k

www.konkur.in

materials: a systematic review of the literature, J Endod 2016;42: 873.

274. Ross IS. *Some effects of heavy metals on fungal cells*, Trans Br Myco Soc 1975;64: 175.
275. Rossi-Fedele G, Ahmed HM. *Assessment of root canal filling removal effectiveness using micro-computed tomography: a systematic review*, J Endod 2017;43: 520.
276. Rossiter JL, Hendrix RA. *Iatrogenic subcutaneous cervicofacial and mediastinal emphysema*, J Otolaryngol 1981;20:.
277. Rowe AH. *Damage to the inferior dental nerve during or following endodontic treatment*, Br Dent J 1983;155: 306- doi:6580031.
278. Rud J, Rud V. *Surgical endodontics of upper molars: relation to the maxillary sinus and operation in acute state of infection*, J Endod 1998;24: 260- doi:9641131.
279. Ruddle CJ. *Micro-endodontic non-surgical retreatment*, Dent Clin North Am 1997;41: 429- doi:9248684.
280. Ruddle CJ. *Nonsurgical retreatment*, Cohen S Burns RC Pathways of the pulp ed 8 2002; CV Mosby St. Louis 875.
281. Ruddle CJ. *Nonsurgical retreatment*, J Endod 2004;30: 827- doi:15564860.
282. Sabala CL, Roane JB, Southard LZ. *Instrumentation of curved canals using a modified tipped instrument: a comparison study*, J Endod 1988;14: 59- doi:3162940.
283. Saber SE, Nagy MM, Schäfer E. *Comparative evaluation of the shaping ability of Wave-One, Reciproc and OneShape single-file systems in severely curved root canals of extracted teeth*, Int Endod J 2015;48: 109-114.
284. Sakkal S, Gagnon A, Lemian L. *[Paresthesia of the mandibular nerve caused by endodontic treatment: a case report]*, J Can Dent Assoc 1994;60: 556- doi:8032999.

Telegram: @dental_k

www.konkur.in

285. Sattapan B, Nervo GJ, Palamara JE, et al: *Defects in rotary nickel-titanium files after clinical use*, J Endod 2000;26: 161- doi:11199711.
286. Saunders JL, Eleazer PD, Zhang P, et al: *Effect of a separated instrument on bacterial penetration of obturated root canals*, J Endod 2004;30: 177- doi:15055438.
287. Schafer E, Dzepina A, Danesh G. *Bending properties of rotary nickel-titanium instruments*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 757- doi:14676769.
288. Schafer E, Schulz-Bongert U, Tulus G. *Comparison of hand stainless steel and nickel titanium rotary instrumentation: a clinical study*, J Endod 2004;30: 432- doi:15167474.
289. Schaeffer MA, White RR, Walton RE. *Determining the optimal obturation length: a meta-analysis of literature*, J Endod 2005;31: 271-274.
290. Schilder H. *Filling root canals in three dimensions*, Dent Clin North Am 1967;11: 723.
291. Schwarze T, Baethge C, Stecher T, et al: *Identification of second canals in the mesiobuccal root of maxillary first and second molars using magnifying loupes or an operating microscope*, Aust Endod J 2002;28: 57- doi:12360670.
292. Scolozzi P, Lombardi T, Jaques B. *Successful inferior alveolar nerve decompression for dysesthesia following endodontic treatment: report of 4 cases treated by mandibular sagittal osteotomy*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 625- doi:15153877.
293. Selbst AG. *Understanding informed consent and its relationship to the incidence of adverse treatment events in conventional endodontic therapy*, J Endod 1990;16: 387-

Telegram: @dental_k

www.konkur.in

doi:2081957.

294. Selden HS. *The endo-antral syndrome: an endodontic complication*, J Am Dent Assoc 1989;119: 397- doi:401 2768704.
295. Seltzer S, Naidorf IJ. *Flare-ups in endodontics. I. Etiological factors*, J Endod 1985;11: 472- doi:3868692.
296. Senges C, Wrbas KT, Altenburger M, et al: *Bacterial and Candida albicans adhesion on different root canal filling materials and sealers*, J Endod 2011;37: 1247- doi:21846541.
297. Setzer FC, Bohme CP. *Influence of combined cyclic fatigue and torsional stress on the fracture point of nickel-titanium rotary instruments*, J Endod 2013;39: 133- doi:23228273.
298. Shimpo T, Gilliatt RW, Kennett RP, et al: *Susceptibility to pressure neuropathy distal to a constricting ligature in the guinea-pig*, J Neurol Neurosurg Psych 1987;50: 1625-1632.
299. Shen Y, Peng B, Cheung GS. *Factors associated with the removal of fractured NiTi instruments from root canal systems*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98: 605- doi:15529134.
300. Shovelton DS. *Surgical emphysema as a complication of dental operations*, Br Dent J 1957;102: 125.
301. Sigurdsson A, Garland RW, Le KT, et al: *Healing of periapical lesions after endodontic treatment with the gentle wave procedure: a prospective multicenter clinical study*, J Endod 2018;44: 510.
302. Sigurdsson A, Garland RW, Le KT, et al: *12-Month healing rates after endodontic therapy using the novel gentle wave system: a prospective multicenter clinical study*, J Endod 2016;42: 1040-1048.

Telegram: @dental_k

www.konkur.in

303. Silva EJ, Rosa TP, Herrera DR, et al: *Evaluation of cytotoxicity and physicochemical properties of calcium silicate-based endodontic sealer MTA Fillapex*, J Endod 2013;39: 274- doi:23321245.
304. Silva E, Vieira VT, Belladonna FG, et al: *Cyclic and torsional fatigue resistance of XPendo Shaper and TRUShape instruments*, J Endod 2018;44: 168-172.
305. Siqueira JF. *Microbial causes of endodontic flare-ups*, Int Endod J 2003;36: 453-12823700.
306. Deleted in page review.
307. Smatt Y, Browaeys H, Genay A, et al: *Iatrogenic pneumomediastinum and facial emphysema after endodontic treatment*, Br J Oral Maxillofac Surg 2004;42: 160- doi:15013551.
308. Smith JG, Elias LA, Yilmaz Z, et al: *The psychosocial and affective burden of posttraumatic neuropathy following injuries to the trigeminal nerve*, J Orofac Pain 2013;27: 293-303.
309. Snyder MB, Rosenberg ES. *Subcutaneous emphysema during periodontal surgery: report of a case*, J Periodontol 1997;48: 790.
310. Somma F, Cammarota G, Plotino G. *The effectiveness of manual and mechanical instrumentation for the retreatment of three different root canal filling materials*, J Endod 2008;34: 466- doi:18358899.
311. Song M, Kim HC, Lee W, et al: *Analysis of the cause of failure in nonsurgical endodontic treatment by microscopic inspection during endodontic microsurgery*, J Endod 2011;37: 1516- doi:22000454.
312. Sousa CJ, Loyola AM, Versiani MA, et al: *A comparative histological evaluation of the biocompatibility of materials used in apical surgery*, Int Endod J 2004;37: 738-

Telegram: @dental_k

www.konkur.in

doi:15479256.

313. Souter NJ, Messer HH. *Complications associated with fractured file removal using an ultrasonic technique*, J Endod 2005;31: 450- doi:15917685.
314. Souyave LC, Inglis AT, Alcalay M. *Removal of fractured endodontic instruments using ultrasonics*, Br Dent J 1985;159: 251- doi:3864460.
315. Spangberg L, Langeland K. *Biologic effects of dental materials. 1. Toxicity of root canal filling materials on HeLa cells in vitro*, Oral Surg 1973;35: 402.
316. Spencer HR, Ike V, Brennan PA. *Review: the use of sodium hypochlorite in endodontics—potential complications and their management*, Br Dent J 2007;202: 555- doi:17496870.
317. Spili P, Parashos P, Messer HH. *The impact of instrument fracture on outcome of endodontic treatment*, J Endod 2005;31: 845- doi:16306815.
318. Stabholz A, Friedman S. *Endodontic retreatment—case selection and technique. Part 2: treatment planning for retreatment*, J Endod 1988;14: 607- doi:3270681.
319. Stammberger H, Jakse R, Beaufort F. *Aspergillosis of the paranasal sinuses: x-ray diagnosis, histopathology and clinical aspects*, Ann Otol Rhinol Laryngol 1984;93: 251- doi:6375518.
320. Steelman RJ, Johannes PW. *Subcutaneous emphysema during restorative dentistry*, Int J Paediatr Dent Br Paedod Soc Int Assoc Dent Child 2007;17: 228- doi:17397469.
321. Stock CJ. *Calcium hydroxide: root resorption and perio-endo lesions*, Br Dent J 1985;158: 325-334 doi:3859315.
322. Stropko JJ. *Canal morphology of maxillary molars: clinical observations of canal configurations*, J Endod 1999;25: 446- doi:10530248.

Telegram: @dental_k

www.konkur.in

323. Sujeet K, Shankar S. *Images in clinical medicine. Prevertebral emphysema after a dental procedure*, New Engl J Med 2007;356: 173.
324. Suter B, Lussi A, Sequeira P. *Probability of removing fractured instruments from root canals*, Int Endod J 2005;38: 112- doi:15667633.
325. Sweatman TL, Baumgartner JC, Sakaguchi RL. *Radicular temperatures associated with thermoplastic gutta percha*, J Endod 2001;27: 512- doi:11501588.
326. Tahan E, elik D, Er K, et al: *Effect of unintentionally extruded mineral trioxide aggregate in treatment of tooth with periradicular lesion: a case report*, J Endod 2010;36: 760- doi:20307760.
327. Takahashi CM, Cunha RS, De Martin AS, et al: *In vitro evaluation of the effectiveness of ProTaper Universal rotary retreatment system for gutta-percha removal with or without a solvent*, J Endod 2009;35: 1580- doi:19840652.
328. Tambe VH, Nagmode PS, Abraham S, et al: *Comparison of canal transportation and centering ability of rotary protaper, one shape system and wave one system using cone beam computed tomography: an in vitro study*, J Conserv Dent 2014;17: 561.
329. Tanalp J, Kaptan F, Sert S, et al: *Quantitative evaluation of the amount of apically extruded debris using 3 different rotary instrumentation systems*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 252.
330. Terauchi Y. *Separated file removal*, Dentistry Today 2012;31: 110.
331. Terauchi Y, O'Leary L, Kikuchi I, et al: *Evaluation of the efficiency of a new file removal system in comparison with two conventional systems*, J Endod 2007;33: 585-

Telegram: @dental_k

www.konkur.in

doi:17437878.

332. Terauchi Y, O'Leary L, Yoshioka T, et al: *Comparison of the time required to create secondary fracture of separated file fragments using ultrasonic vibration under various canal conditions*, J Endod 2013;39: 1300- doi:24041396.
333. Thompson SA, Dummer PMH. *Shaping ability of Quantec Series 2000 rotary nickel-titanium instruments in simulated root canals: part 2*, Int Endod J 1998;31: 268- doi:9823116.
334. Tilotta-Yasukawa F, Millot S, El Haddioui A, et al: *Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: e47- doi:16997095.
335. Tinaz AC, Alacam T, Uzun O, et al: *The effect of disruption of apical constriction on periapical extrusion*, J Endod 2005;31: 533- doi:15980716.
336. Tinoco JM, De-Deus G, Tinoco EM, et al: *Apical extrusion of bacteria when using reciprocating single-file and rotary multifile instrumentation systems*, Int Endod J 2014;47: 560-566.
337. Topğuoƒülu HS, Aktı A, Tuncay Ö, et al: *Evaluation of debris extruded apically during the removal of root canal filling material using ProTaper, D-RaCe, and R-Endo rotary nickel-titanium retreatment instruments and hand files*, J Endod 2014;40: 2066.
338. Torabinejad M, Lemon RR. *Procedural accidents*, Walton RE Torabinejad M Principles and practice of endodontics ed 3 2002; WB Saunders Philadelphia 310.
339. Torabinejad M, Walton RE. Principles and practice of endodontics ed 4 2009; Saunders St. Louis.
340. Toyoğlu M, Altunbaş D. *Influence of different kinematics*

Telegram: @dental_k

www.konkur.in

on apical extrusion of irrigant and debris during canal preparation using K3XF instruments, J Endod 2017;43: 1565-1568.

341. Tronstad L, Sunde PT. *The evolving new understanding of endodontic infections*, Endod Topics 2003;6: 57.
342. Turnbull A. *Remarkable coincidence in dental surgery [letter]*, Br Med J 1900;1: 1131.
343. Tzanetakis GN, Kontakiotis EG, Maurikou DV, et al: *Prevalence and management of instrument fracture in the postgraduate endodontic program at the Dental School of Athens: a five-year retrospective clinical study*, J Endod 2008;34: 675- doi:18498887.
344. Uehara M, Okumura T, Asahina I. *Subcutaneous cervical emphysema induced by a dental air syringe: a case report*, Int Dent J 2007;57: 286- doi:17849689.
345. Ullmann CJ, Peters OA. *Effect of cyclic fatigue on static fracture loads in ProTaper nickel-titanium rotary instruments*, J Endod 2005;31: 183- doi:15735465.
346. Uraba S, Ebihara A, Komatsu K, et al: *Ability of cone-beam computed tomography to detect periapical lesions that were not detected by periapical radiography: a retrospective assessment according to tooth group*, J Endod 2016;42: 1186-1190.
347. Van der Borden WG, Wang X, Wu MK, et al: *Area and three dimensional volumetric changes of periapical lesions after root canal treatments*, J Endod 2013;39: 1245-1249.
348. van der Vyver PJ, Paleker F, Vorster M, et al: *Root canal shaping using Nickel Titanium, M-Wire, and Gold Wire: A micro-computed tomographic comparative study of One Shape, ProTaper Next, and WaveOne Gold Instruments in maxillary first molars*, J Endod 2019;45: 62-67.
349. Vandrangi P. *Evaluating penetration depth of treatment*

Telegram: @dental_k

www.konkur.in

fluids into dentinal tubules using the gentle wave system, Dentistry 2016;6: 366.

350. Vasilakis GJ, Vasilakis CM. *Mandibular endodontic-related paresthesia*, General Dent 2004;52: 334.
351. Walton RE. *Current concepts of canal preparation*, Dent Clin North Am 1992;36: 309- doi:1572501.
352. Walton RE, Torabinejad M. Principles and practice of endodontics ed 3 2002; WB Saunders Philadelphia 184, 222-223, 319.
353. Ward JR, Parashos P, Messer HH. *Evaluation of an ultrasonic technique to remove fractured rotary nickel-titanium endodontic instruments from root canals: an experimental study*, J Endod 2003;29: 756- doi:14651285.
354. Ward JR, Parashos P, Messer HH. *Evaluation of an ultrasonic technique to remove fractured rotary nickel-titanium endodontic instruments from root canals: clinical cases*, J Endod 2003;29: 764- doi:14651286.
355. Watzek G, Bernhart T, Ulm C. *Complications of sinus perforations and their management in endodontics*, Dent Clin North Am 1997;41: 563- doi:9248692.
356. Weine F. Endodontic therapy ed 5 1996; Mosby St. Louis 324-330, 545.
357. Weisman MI. *The removal of difficult silver cones*, J Endod 1983;9: 210- doi:6574209.
358. Wilcox LR, Roskelley C, Sutton T. *The relationship of root canal enlargement to finger-spreader induced vertical root fracture*, J Endod 1997;23: 533- doi:9587326.
359. Wong R, Cho F. *Microscopic management of procedural errors*, Dent Clin North Am 1997;41: 455- doi:9248685.
360. Deleted in page review.
361. Wright KJ, Derkson GD, Riding KH. *Tissue-space emphysema, tissue necrosis, and infection following use of*

Telegram: @dental_k

www.konkur.in

compressed air during pulp therapy: case report, Pediatr Dentistry 1991;13: 110.

362. Wu MK, Kast'akova A, Wesselink PRL. *Quality of cold and warm gutta-percha in oval canals in mandibular premolars*, J Endod 1998;24: 223- doi:9641122.
363. Wu MK, Wesselink PR, Walton RE. *Apical terminus location of root canal treatment procedures*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 99- doi:10630950.
364. Xu J, He J, Yang Q, et al: *Accuracy of cone-beam computed tomography in measuring dentin thickness and its potential of predicting the remaining dentin thickness after removing fractured instruments*, J Endod 2017;43: 1522-1527.
365. Yaltirik M, Berberoglu HK, Koray M, et al: *Orbital pain and headache secondary to overfilling of a root canal*, J Endod 2003;29: 771- doi:14651288.
366. Yang SC, Chiu TH, Lin TJ, et al: *Subcutaneous emphysema and pneumomediastinum secondary to dental extraction: a case report and literature review*, Kaohsiung J Med Sci 2006;22: 641- doi:17116627.
367. Yekta SS, Koch F, Grosjean MB, et al: *Analysis of trigeminal nerve disorders after oral and maxillofacial intervention*, Head Face Med 6, 2010;26: 24.
368. Yilmaz F, Koc C, Kamburoglu K, et al: *Evaluation of 3 different retreatment techniques in maxillary molar teeth by using micro-computed tomography*, J Endod 2018;44: 480-484.
369. Yoldas O, Oztunc H, Tinaz C, et al: *Perforation risks associated with the use of Masserann endodontic kit drills in mandibular molars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97: 513- doi:15088037.

Telegram: @dental_k

www.konkur.in

370. Yoshimoto A, Mitamura Y, Nakamura H, et al: *Acute dyspnea during dental extraction*, Respiration 2002;69: 369- doi:12169756.
371. You SY, Bae KS, Baek SH, et al: *Lifespan of one nickel-titanium rotary file with reciprocating motion in curved root canals*, J Endod 2010;36: 1991- doi:21092819.
372. Yum J, Cheung GS, Park JK, et al: *Torsional strength and toughness of nickel-titanium rotary files*, J Endod 2011;37: 382- doi:21329826.
373. Zehnder M. *Root canal irrigants*, J Endod 2006;32: 389- doi:16631834.
374. Zemann W, Feichtinger M, Karcher H. *Cervicofacial and mediastinal emphysema after crown preparation: a rare complication*, Int J Prosthodont 2007;20: 143- doi:17455433.
375. Zuniga JR, Mistry C, Tikhonov I, et al: *Magnetic resonance neurography of traumatic and nontraumatic peripheral trigeminal neuropathies*, J Oral Maxillofac Surg 2018;76: 725-736.
376. Zuniga JR, Renton T. *Managing post-traumatic trigeminal neuropathic pain: is surgery enough?*, J Neurol Neuromed 2016;1: 10-14.
377. Zuolo ML, Walton RE, Imura N. *Histologic evaluation of three endodontic instrument/preparation techniques*, Endod Dent Traumatol 1992;8: 125- doi:1289071.

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21: The role of endodontics after dental traumatic injuries

Bill Kahler*

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A traumatic injury to the tooth results in damage to many dental and periradicular structures, making the management and consequences of these injuries multifactorial. Knowledge of the interrelating healing patterns of these tissues is essential. This chapter concentrates on the role of the dentinopulpal complex in the pathogenesis of disease subsequent to dentinal trauma and how treatment of this complex can contribute to favorable healing after an injury.

Unique aspects of dental trauma

Most dental trauma occurs in the 7- to 12-year-old age group and is mainly due to falls and accidents near home or school.[28,165] It occurs primarily in the anterior region of the mouth, affecting the maxillary more than the mandibular jaw.[33] Serious accidents, such as automobile crashes, can affect any tooth and can occur in all age ranges. In many cases, after a traumatic dental injury, endodontic treatment is provided to caries-free, single-rooted, young permanent teeth. If quick and correct treatment for these teeth is provided after injury, the potential for a successful endodontic outcome is very good.

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Most common types of dental trauma

Crown fractures

Most crown fractures occur in young, caries-free anterior teeth.[109,128] This makes maintaining or regaining pulp vitality essential. Luckily, vital pulp therapy supports a good prognosis in these situations if correct treatment and follow-up procedures are carefully followed.

Crown-root fractures

Crown-root fractures are first treated periodontally to ensure that there is a sufficient and good margin to allow restoration. If the tooth can be maintained from a periodontal point of view, then the pulp is treated as for a crown fracture.

Root fractures

A surprisingly large number of pulps in root-fractured teeth will survive this rather dramatic injury. In almost every case, the apical segment stays vital, and in many cases, the coronal segment stays vital or regains vitality subsequent to the injury. If the coronal segment permanently loses vitality, it should be treated as an immature permanent tooth with nonvital pulp. *The apical segment rarely needs treatment.*

Luxation injuries and avulsion

Luxation injuries and avulsion often result in pulp necrosis and damage to the cemental protective layer of the root. The potential complication of pulp infection in a root that has lost its cemental protective layer makes these injuries potentially catastrophic. Correct emergency and follow-up evaluation, which may include timely endodontic treatment, is critical.

Follow-up after dental trauma

The reader is referred to Chapter 1 for specific descriptions of pulp tests, but a few general statements about pulp tests on traumatized teeth may be helpful in trying to interpret the results.

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For decades, controversy has surrounded the validity of thermal and electric tests on traumatized teeth. Only generalized impressions may be gained from these tests following a traumatic injury. They are in reality sensitivity tests for nerve function and do not indicate the presence or absence of blood circulation within the pulpal space. It is assumed that subsequent to traumatic injury, the conduction capability of the nerve endings and/or sensory receptors is sufficiently deranged to inhibit the nerve impulse from an electrical or thermal stimulus. This makes the traumatized tooth vulnerable to false-negative readings from such tests.[146]

Teeth that give a response at the initial examination cannot be assumed to be healthy and to continue to give a response over time. Teeth that yield no response cannot be assumed to have necrotic pulps because they may give a response at later follow-up visits. It has been demonstrated that it may take as long as 9 months for normal blood flow to return to the coronal pulp of a traumatized fully formed tooth. As circulation is restored, the responsiveness to pulp tests returns.[81]

The transition from a negative response to a positive response at a subsequent test may be considered a sign of a healing pulp. The repetitious finding of responses may be taken as a sign of a healthy pulp. The transition from a response to no response may be taken as an indication that the pulp is probably undergoing degeneration, and therefore an intervention in the form of endodontic treatment might be indicated. The persistence of no response would suggest that the pulp has been irreversibly damaged, but even this is not absolute.[39]

Thermal and electrical pulp tests of all anterior teeth (canine to canine) of the maxillary and mandibular jaws should be performed at the time of the initial examination and carefully recorded to establish a baseline for comparison with subsequent repeated tests in later months. These tests should be repeated at 2 to 4 and 6 to 8 weeks; at 3, 6, and 12 months; and at yearly intervals after the trauma (according to the guidelines of the American Association of Endodontists). The purpose of the tests is to establish a trend as to the physiologic status of the pulps of these teeth. Particularly in traumatized teeth, carbon dioxide snow (CO_2, −78°C) or dichlorodifluoromethane (−40°C) placed on the incisal third of the facial surface gives more accurate responses than a water-ice pencil (Fig. 21.1).[79,80]

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The intense cold seems to penetrate the tooth and covering splints or restorations and reach the deeper areas of the tooth. Neither the dry ice nor the dichlorodifluoromethane spray forms ice water, which could disperse over adjacent teeth or gingiva to give a false-positive response. In trauma evaluation, there is no question that using water-ice pencils should be avoided because of this. Dichlorodifluoromethane spray is a very inexpensive alternative to CO_2 snow; its coldness elicits much more reliable responses than water-ice. The electrical pulp test relies on electrical impulses directly stimulating the nerves of the pulp. These tests have limited value in young teeth but are useful when the dentinal tubules are closed and do not allow dentinal fluid to flow in them. This situation is typical of teeth in elderly patients or in traumatized teeth that are undergoing premature sclerosis. In these situations, the thermal tests that rely on fluid flow in the tubules cannot be used, and the electrical pulp test becomes important.

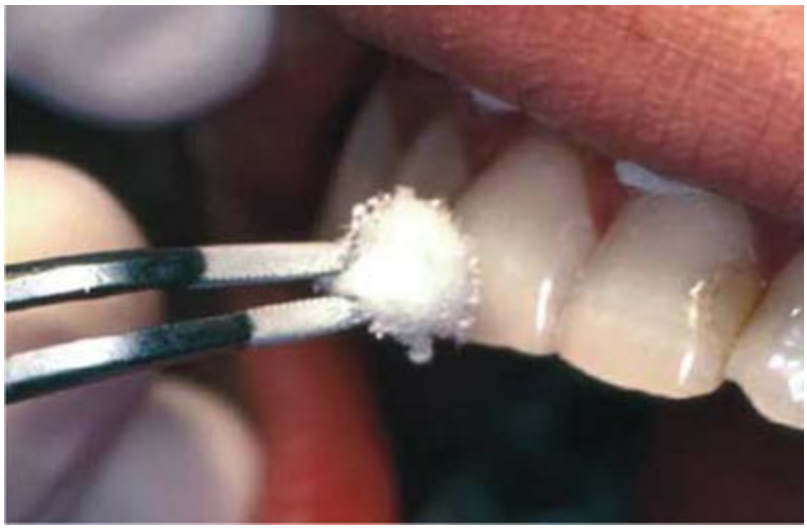

FIG. 21.1 Dichlorodifluoromethane (−40°F [−40°C]) gas is sprayed on a cotton pellet and then placed on the incisal edge of the maxillary incisor.

Close-up view shows gloved hands placing the cotton pellet using forceps on the upper front tooth.

Laser Doppler flowmetry (LDF) was introduced in the early 1970s for measurement of blood flow in the retina.[102] The technique has also been used to assess blood flow in other tissue systems, such as the skin and renal cortex.

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It uses a beam of infrared (780 to 820 nm) or near-infrared (632.8 nm) light that is directed into the tissue by optical fibers. As light enters the tissue, it is scattered by moving red blood cells and stationary tissue cells. Photons that interact with moving red blood cells are scattered, and the frequency shifts according to the Doppler principle. Photons that interact with stationary tissue cells are scattered but not Doppler shifted. A portion of the light is returned to a photodetector, and a signal is produced (Fig. 21.2).

FIG. 21.2 Laser Doppler machine.

DRT4, Laser Doppler Monitor manufactured by moor instruments shows a display at the center with controls on left and right.

Source: (Courtesy Moor Instruments, Devon, United Kingdom.)

Attempts have been made to use LDF technology for pulp vitality diagnosis in traumatized teeth because this would provide a more accurate reading of the vitality status of the pulp.[136,196,197] Studies have shown promising results, indicating that laser Doppler can detect blood flow more consistently and earlier than the standard vitality tests would be expected to render a response. In a study on young dogs, laser Doppler was able to correctly detect blood flow as early as 2 to 3 weeks after avulsion of an immature tooth and at the same time indicate no flow in those that would remain necrotic.[197]

Presently the cost of an LDF machine limits its use in private dental

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offices; these are used primarily in hospitals and teaching institutions.

Radiographic examinations

Radiographic imaging is essential for thorough examination, diagnosis, and management of dentoalveolar trauma. Imaging may reveal root fractures, subgingival crown fractures, tooth displacements, bone fractures, root resorptions, and embedded foreign objects. A single radiograph, even a panoramic image, is insufficient to properly diagnose just about any dental trauma case. In its 2020 guidelines for the management of traumatic dental injuries, and in the current recommendations on its interactive website (www.dentaltraumaguide.org), the International Association of Dental Traumatology (IADT) has recommended taking at least four different radiographs for almost every injury[44a,64,75a]: a direct 90-degree on the axis of the tooth, two with different vertical angulations, and one occlusal film (Fig. 21.3).

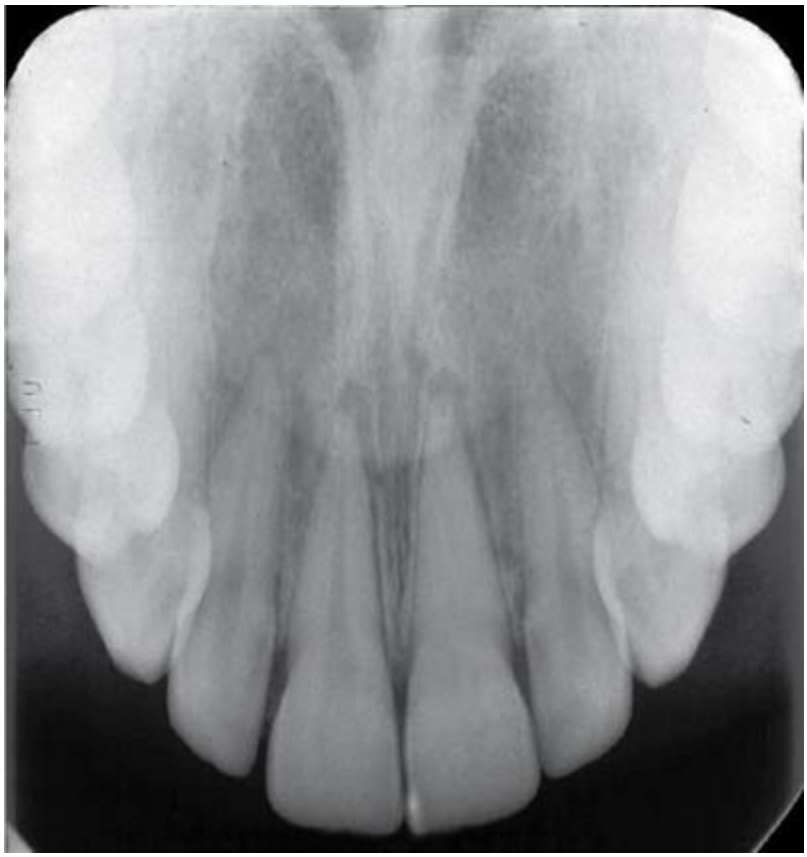

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FIG. 21.3 An occlusal film of a luxation injury of central incisors. Both were diagnosed to be laterally luxated with apical translocation. Note that the left central is completely obliterated and has a history of being luxated some years prior.

Radiograph shows two large oval patches extending from the gum line and roots of incisors.

Multiple radiographs increase the likelihood of diagnosing root fractures, tooth displacement, and other possible injuries. However, two-dimensional (2D) imaging methods have well-known inherent limitations, and their lack of three-dimensional (3D) information may prevent proper diagnosis and adversely affect long-term treatment outcomes. The interpretation of an image can be confounded by a number of factors, including the regional anatomy and superimposition of both the teeth and surrounding dentoalveolar structures. As a result of superimposition, periapical radiographs reveal only limited aspects (i.e., a 2D view) of the true 3D anatomy.[52,142] In addition, there is often geometric distortion of the anatomic structures imaged with conventional radiographic methods.[4] These problems can be overcome by using cone-beam computed tomography (CBCT) imaging techniques, which produce accurate 3D images of the teeth and surrounding dentoalveolar structures and have shown promise in improving the clinician's ability to properly diagnose luxation injuries, alveolar fractures, root fractures, and root resorption (Fig. 21.4).[50,51]

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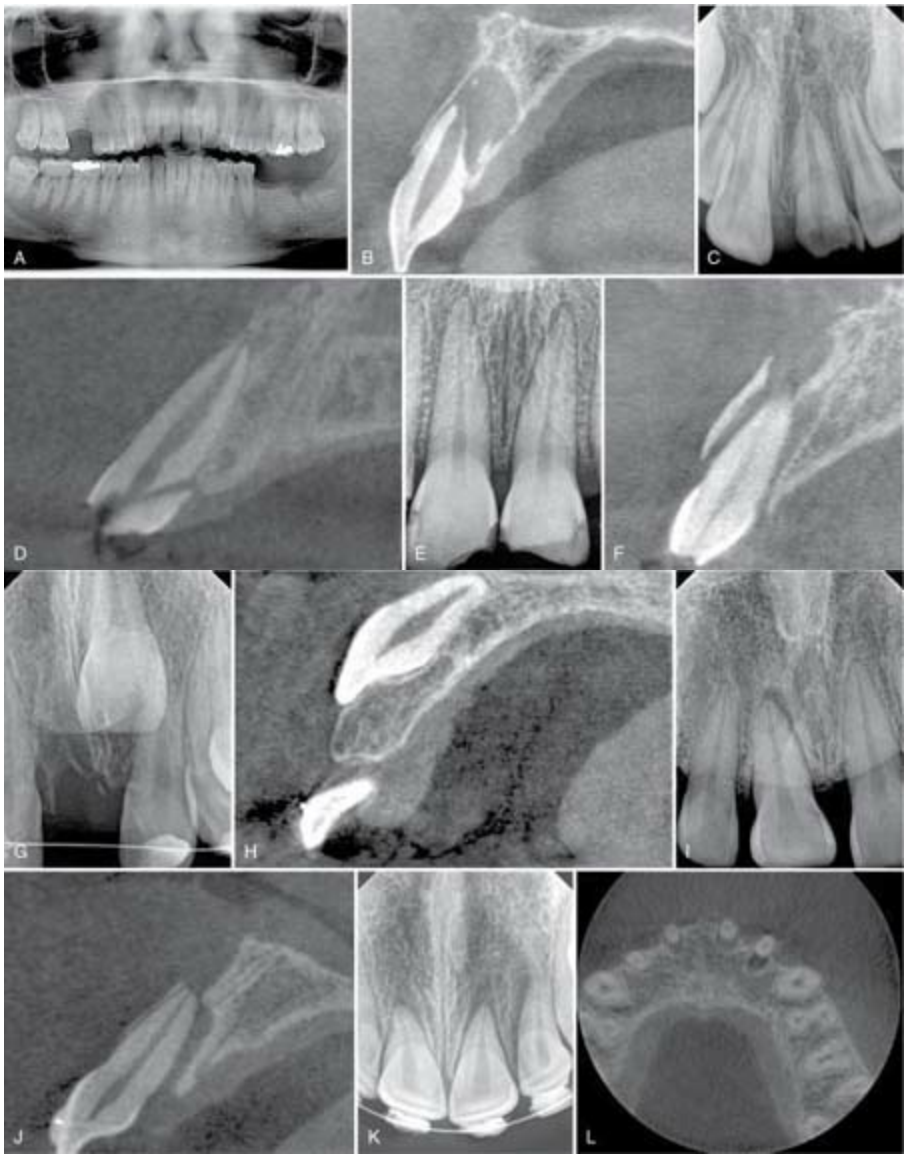

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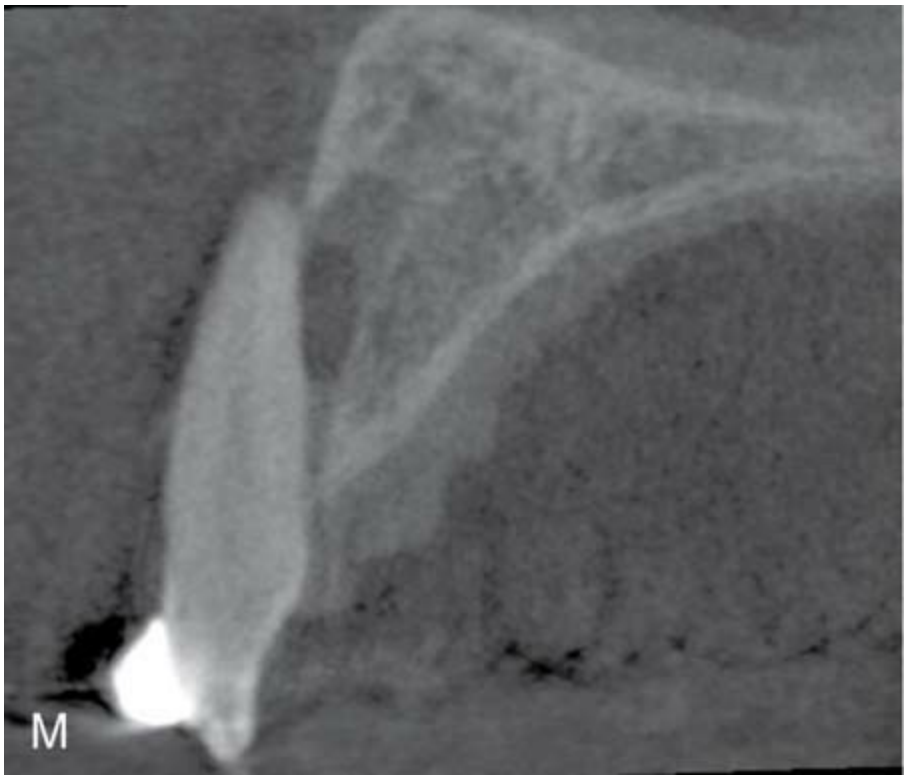

FIG. 21.4 **A,** Panoramic radiograph taken on a patient with past history of dentoalveolar trauma. Tooth #9 appeared to have arrested root development with a periapical radiolucency. **B,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 had arrested root development, a large periapical radiolucency, and extensive root resorption along the palatal surface that was *not* evident on the panoramic image. **C,** Periapical radiograph of tooth #9 with extensive complicated crown fracture. The clinical examination revealed the crown fracture extended subgingivally on the palatal aspect. **D,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that the apical extent of the fractured palatal portion extended just apical to the crest of bone. The correct diagnosis of this injury was complicated crown-root fracture. **E,** Periapical radiograph of teeth #8, #9 revealed crown fractures. **F,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 suffered a lateral luxation displacement injury with extensive concomitant alveolar fracture. The necessary treatment of repositioning and splinting was apparent. **G,** Periapical radiograph of tooth #9 following dentoalveolar trauma. Tooth #8 was avulsed and tooth #9 appeared to be severely intruded. **H,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed the labial position of tooth #9. **I,** Periapical radiograph of tooth #8 revealed a lateral luxation injury. **J,** Sagittal view of tooth #8 from CBCT imaging. The extensive tooth displacement and concomitant alveolar are evident. **K,** Periapical radiograph of teeth #8 to #10, that had recently been splinted in an emergency room. This image revealed that these teeth were not

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properly repositioned before splinting, evidenced by the spaces along each root. **L,** Axial view of maxillary anterior region of same patient from CBCT imaging. This revealed that teeth #8 to #10 are labially displaced. **M,** Sagittal view of tooth #10 of same patient revealed how severe the displacement was. Following consent and local anesthesia, the splint was removed and teeth #8 to #10 were repositioned and resplinted.

Six radiographs marked A through F.

A) It shows radiolucent edge of central incisor along with dark patches at the root apices. One tooth is missing in the upper right quadrant and few teeth are missing in the lower left quadrant.

B) Incisor has a large dark patch at the root apex followed by porous tissue above.

C) Two incisors at the center have space in-between. Tissue above root apices show large marrow spaces with some dark patches.

D) Tooth has fracture in crown, indicated by radiolucent regions.

E) Close-up view of two central incisors. Both have broken crown edges and wedge-shaped radiolucency.

F) Tooth shows radiopaque lateral edges of the crown.

Seven radiographs are marked G through M.

G) It shows tooth little above the root tip of adjacent incisor. Gum line is completely radiolucent.

H) It has displaced tooth along the arch.

I) It shows three incisors with a tooth-like structure at the top. The central incisor has radiolucent curved patch at the root apex.

J) The central incisor with radiolucency at the root apex.

K) Three incisors that appear little smaller and locate slightly below the teeth arch.

L) Teeth arranged in inverted U-shaped structure. The teeth at the center are situated away from their original position.

M) Incisor shows an oval radiolucent patch near the root apex and radiopaque patch at the one side of crown.

In instances of soft-tissue laceration, it is advisable to radiograph the

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injured area before suturing to be sure that no foreign objects have been embedded. A soft-tissue radiograph with a normal-sized film briefly exposed at reduced kilovoltage should reveal the presence of many foreign substances, including tooth fragments (Fig. 21.5).

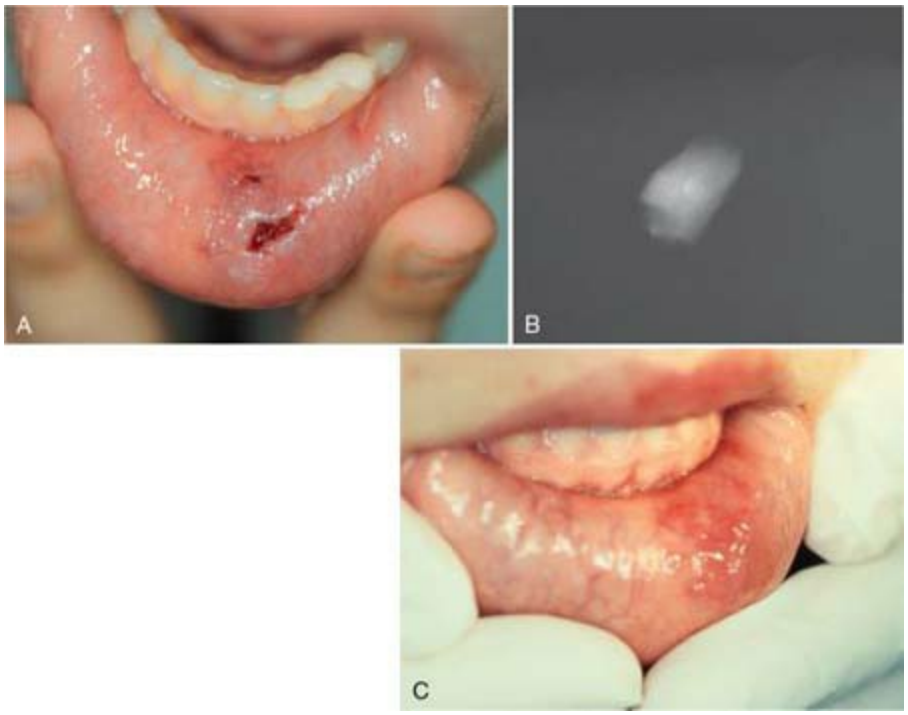

FIG. 21.5 Patient sustained complicated crown fractures on the left lower incisors and later on the canine tooth. **A,** An emergency pulp capping; Band-Aid composite was placed on the teeth. Six days later the patient was referred to the clinic because of a poorly healed laceration on the lip. **B,** Radiograph of the lip revealed a portion of the lateral crown still in the lip. The patient was anesthetized and the crown removed. **C,** Healing was quick and uneventful.

A) Close-up view of lower lip with a deep wound that shows clotted blood surrounded by inflammation.

B) Radiograph shows a radiopaque crown in the dark background.

C) Close-up view of gloved hands stretches the lower lip that shows slight inflamed tissue in place of the wound.

Cone-beam computed tomography and dentoalveolar trauma

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CBCT is accomplished by using a rotating gantry to which an x-ray source and detector are fixed (see Chapter 2). A divergent pyramidal or cone-shaped source of ionizing radiation is directed through the middle of the area of interest onto an area x-ray detector on the opposite side of the patient. The x-ray source and detector rotate around a fixed fulcrum within the region of interest. During the exposure sequence, hundreds of planar projection images are acquired of the field of view (FOV) in an arc of at least 180 degrees. In this single rotation, CBCT provides precise, essentially immediate, and accurate 3D radiographic images. Because CBCT exposure incorporates the entire FOV, only one rotational sequence of the gantry is necessary to acquire enough data for image reconstruction.

CBCT has been suggested as an adjunct imaging tool when the true nature of the dentoalveolar root fracture and dental injuries cannot be confidently diagnosed from a conventional examination and radiographs.[13,50,134,143] However, it has been repeatedly demonstrated that the use of CBCT provides improved diagnostic images in cases of dentoalveolar injury.[37,38,50,51,52,66,71,123,134,142,143] Perhaps the use of CBCT imaging for dentoalveolar injuries could be considered “best practice” and is recommended for most serious injuries (Fig. 21.6).[69]

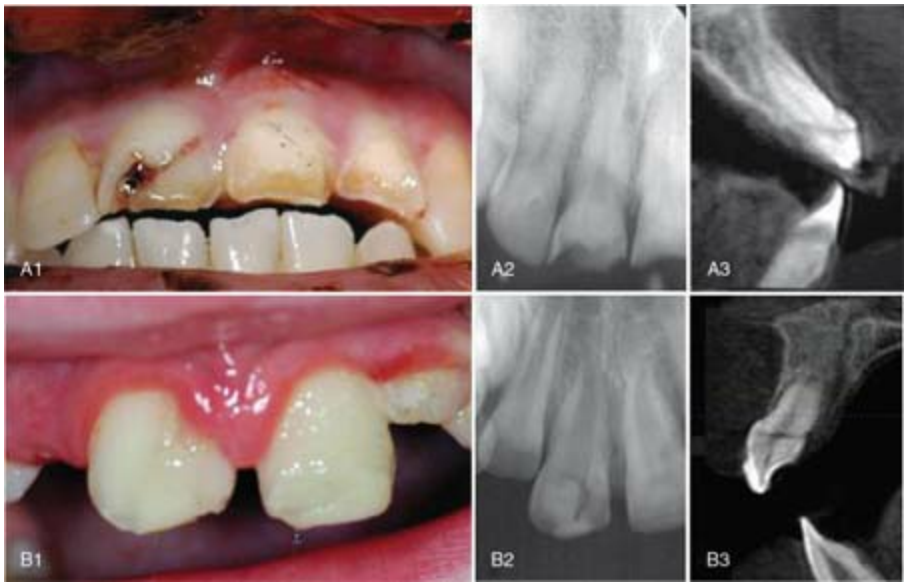

FIG. 21.6 **A1,** A clinical photograph of a maxillary right central incisor that sustained a crown-root fracture. **A2,** A periapical radiograph that failed to show the extent of the crown-root fracture. **A3,** A CBCT image

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revealed the extent of the crown-root fracture. **B1,** A clinical photograph of a right maxillary central incisor that had sustained a crown fracture that was restored with composite resin by the referring dentist. **B2,** A periapical radiograph revealed a vertical fracture in the crown and the coronal third of the root. An area of bone loss just below the alveolar crest on the mesial aspect of the root suggested further injury. **B3,** A CBCT image revealed the further injury as a transverse root fracture.

A1) Close-up view of teeth shows cracks on crowns of front three teeth. The right central incisor has brown filling.

A2) Radiograph shows three teeth where the central tooth has chipped crown.

A3) Close-up view of the chipped crown shows wide empty space in-between.

B1) Close-view of upper jaw shows front incisors with wide space in-between and the surrounding.

B2) Radiograph shows the central tooth with radiolucent root canal and radiolucent triangular fracture on the crown.

B3) Radiograph shows wide black space adjacent to incisor. The crown of incisor is broken from one side.

Source: (Reproduced from Andreasen FM, Kahler B: Diagnosis of acute dental trauma: the importance of standardized documentation: a review, *Dent Traumatol* 31:340, 2015.)

Root resorption

Because the development and progression of root resorption occur without clinical signs or symptoms, their early detection is challenging. Therefore the definitive diagnosis of root resorption depends on its radiographic demonstration, which in turn is limited by the diagnostic accuracy of the imaging device used to determine its presence (see Chapter 18).[143] The radiodensity of the root requires that a significant amount of root substance be removed to cause enough contrast on the radiograph to allow it to be detected. Thus only resorptive defects on the mesial or distal aspects of the root can be predictably detected after some time; the facial and palatal or lingual aspects are much harder to see. To overcome these difficulties, it is essential to take as many different horizontal-angled radiographs as feasible in cases of suspected root resorption. Early detection of small resorption defects has been shown to be poor with conventional dental radiographs, and

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the extent of the resorptive defect is grossly underestimated compared with CBCT.[38,66,71]

The available literature supports the use of CBCT as a diagnostic tool to assess the true nature of teeth diagnosed with root resorption to improve diagnosis and aid management. This should ultimately improve the prognosis of teeth with root resorption that require endodontic management (see Fig. 21.4, *A*, *B*).[143]

Horizontal (transverse) root fractures

There is a significant risk of misdiagnosing the true location of a root fracture in anterior teeth when intraoral radiography is used because of the possibility of the oblique course of the fracture line in the sagittal plane. It has been shown that horizontal root fracture can be detected sooner using CBCT than with periapical views, and the fracture can be assessed in coronal, axial, and cross-sectional views (see Fig. 21.4, *C*, *D*).[117] Compared with conventional radiographs, CBCT increased the accuracy of diagnosing the actual nature of horizontal root fractures.[37,134]

Luxation injuries

As previously mentioned, conventional intraoral radiography provides poor sensitivity in the detection of minimal tooth displacements and root and alveolar fractures.[123] CBCT has significantly improved the ability to accurately diagnose traumatic injuries and has the potential to overcome most of the technical limitations of the plain-film projection (see Fig. 21.4, *E*–*M*).[37,38,50,51,66,71,123,134,143]

Crown fractures

As already mentioned, the primary aim from an endodontic point of view is to maintain pulp vitality after crown fractures.

Crown infraction

Crown infraction can be defined as an incomplete fracture of or a crack in the enamel, without loss of tooth structure.[21]

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Biologic consequences

Crown infractions are injuries that carry little danger of resulting in pulp necrosis. Meticulous follow-up over a 5-year period is the most important endodontic preventive measure in these cases. If, at any follow-up examination, the reaction to sensitivity tests changes, or if, on radiographic assessment, signs of apical or periradicular periodontitis develop or the root appears to have stopped development or is obliterating, endodontic intervention should be considered.

Uncomplicated crown fracture

Uncomplicated crown fracture can be defined as fracture of the enamel only or the enamel and dentin without pulp exposure.[21]

Incidence

Uncomplicated crown fracture is likely to be the most commonly reported dental injury. It is estimated to account for at least one third to one half of all reported dental trauma.

Biologic consequences

Uncomplicated crown fractures are also injuries that have little danger of resulting in pulp necrosis. In fact, the biggest danger to the health of the pulp is through iatrogenic causes during the esthetic restoration of these teeth.

Treatment

There are two key issues in the treatment of crown fractures. First, all exposed dental tubules need to be closed as soon as reasonably possible. If the broken-off piece is not available or if it is not possible to reattach it and there is no time to do a full composite restoration at the time of the emergency appointment, a composite Band-Aid or temporary coverage should be placed on all exposed dentin. This prevents any ingress of bacteria into the tubules and reduces the patient's discomfort. The second issue is the remaining dentin thickness. Several studies have confirmed that if the remaining dentin is more than 0.5 mm thick, the tooth can be restored with the restoration of choice, including etching and bonding, and no special

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attention needs to be given to the pulp.[4,54,70,137,173] However, if the remaining dentin is less than this thickness, a protective layer of hard-setting calcium hydroxide in the deepest part of the dentin exposure does reduce, if not completely prevent, reactive inflammation of the underlying pulp, which is a significantly different reaction compared with the negative reaction to composite bonding systems.[4,54,70,137,173]

Complicated crown fracture

A *complicated crown fracture* involves enamel, dentin, and pulp (Fig. 21.7).[21]

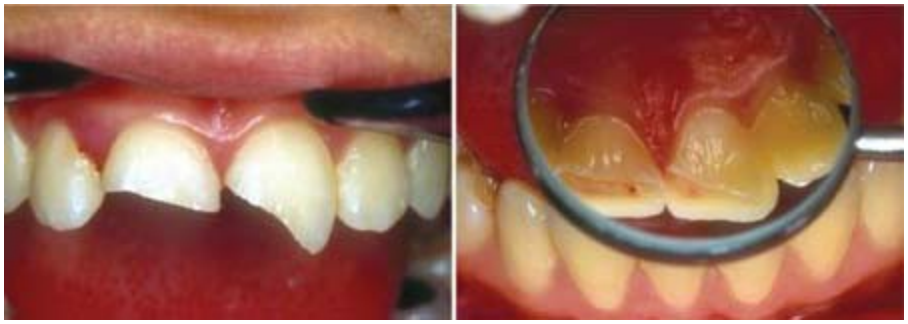

FIG. 21.7 Complicated crown fracture involving enamel, dentin, and pulp.

1) Close-up view shows upper jaw where half-crown of right incisor is broken horizontally. The crown of left incisor is broken diagonally.

2) Close-up view shows horizontal and diagonal cracks under dental mirror in right and left incisor, respectively.

Incidence

Complicated crown fractures occur in 0.9% to 13% of all dental injuries.[47,153,175]

Biologic consequences

A crown fracture involving the pulp, if left untreated, always results in pulp necrosis.[116] However, the manner and time sequence in which the pulp becomes necrotic allow a great deal of potential for successful intervention to

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maintain pulp vitality. The first reaction after the injury is hemorrhage and local inflammation (Fig. 21.8).

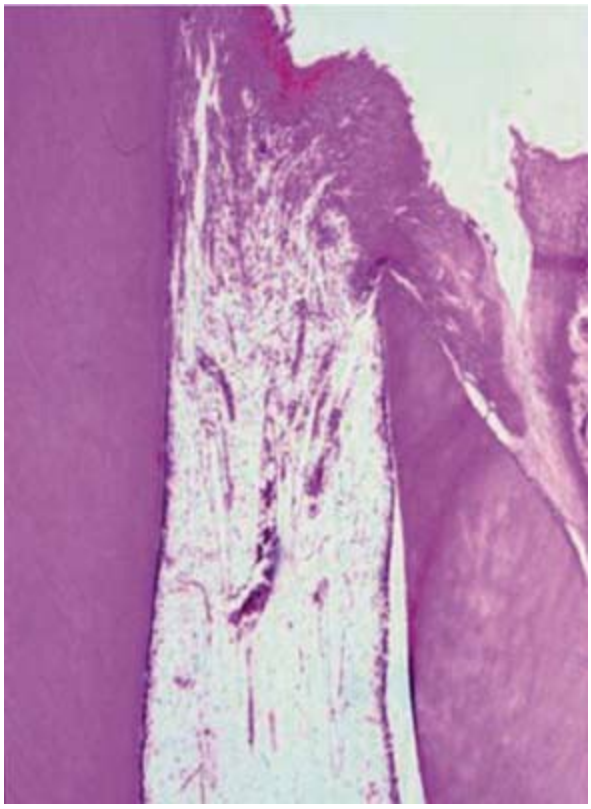

FIG. 21.8 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferated over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

Stained micrograph of the inflamed pulp shows proliferated cells in canal with a tinge of purple.

Subsequent inflammatory changes are usually proliferative but can be destructive over time. A proliferative reaction is favored in traumatic injuries because the fractured surface is usually flat, allowing salivary rinsing with little chance of impaction of contaminated debris. Unless impaction of contaminated debris is obvious, it is expected that in the first 24 hours after the injury, a proliferative response with inflammation extending not more than 2 mm into the pulp will be present (see Fig. 21.8).[57,60,90] In time, the bacterial challenge results in local pulpal necrosis and a slow apical progression of the pulpal inflammation (Fig. 21.9).

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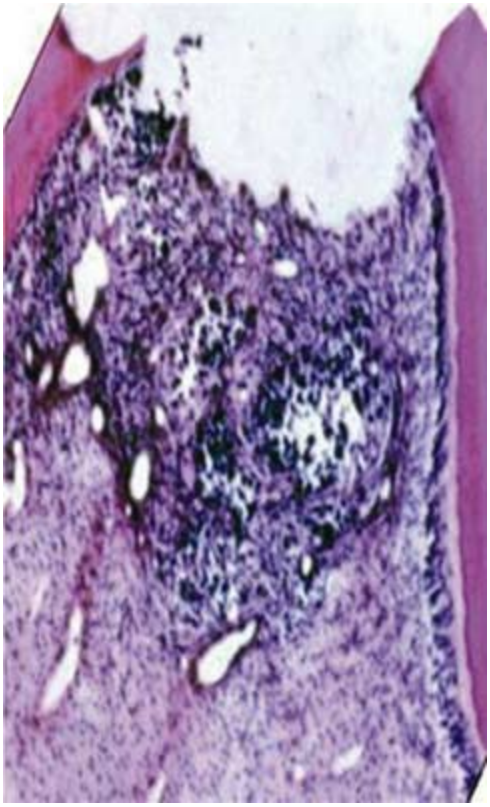

FIG. 21.9 Histologic appearance of the pulp days after a traumatic exposure. Superficial necrosis above a zone of inflamed pulp is seen.

Stained micrograph of partial pulp necrosis shows normal cell nuclei at the bottom. At the top, porous connective tissue has white and dark patches with granules.

Treatment

Treatment options for complicated crown fracture are (1) vital pulp therapy, comprising pulp capping, partial pulpotomy, or full pulpotomy; and (2) pulpectomy. The choice of treatment depends on the stage of development of the tooth, the time between trauma and treatment, concomitant periodontal injury, and the restorative treatment plan.

Stage of development of the tooth

Loss of vitality in an immature tooth can have catastrophic consequences. Root canal treatment on a tooth with a blunderbuss canal is time consuming

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and difficult. It is probably more important that necrosis of an immature tooth leaves it with thin dentinal walls that are susceptible to fracture both during and after the apexification procedure.[118] Every effort must be made to keep the tooth vital, at least until the apex and cervical root have completed their development.

Removal of the pulp in a mature tooth is not as significant as in an immature tooth because a pulpectomy in a mature tooth has an extremely high success rate.[164] However, it has been shown that under optimal conditions, vital pulp therapy (rather than removal) can be carried out successfully on a mature tooth.[133,193] Therefore this form of therapy can be an option under certain circumstances, even though a pulpectomy is the treatment that affords the most predictable success.

In an immature tooth, vital pulp therapy should always be attempted if at all feasible because of the tremendous advantages of maintaining the vital pulp.

Time between trauma and treatment

For 48 hours after a traumatic injury, the initial reaction of the pulp is proliferative, with no more than a 2-mm depth of pulpal inflammation (see Fig. 21.9). After 48 hours, chances of direct bacterial contamination of the pulp increase, with the zone of inflammation progressing apically[60]; as time passes, the likelihood of successfully maintaining a healthy pulp decreases.

Concomitant attachment damage

A periodontal injury compromises the nutritional supply of the pulp. This fact is particularly important in mature teeth, in which the chance of pulp survival is not as good as for immature teeth.[18,68]

Restorative treatment plan

Unlike in an immature tooth, in which the benefits of maintaining vitality of the pulp are so great, pulpectomy is a viable treatment option in a mature tooth. As pointed out, if performed under optimal conditions, vital pulp therapy after traumatic exposures can be successful. If the restorative treatment plan is simple and a composite resin restoration will suffice as the permanent restoration, this treatment option should be given serious

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consideration. If a more complex restoration is to be placed (e.g., a crown or bridge abutment), pulpectomy may be the more predictable treatment method.

Vital pulp therapy: Requirements for success

Vital pulp therapy has an extremely high success rate if the following requirements can be met (also see Chapter 24).

- *Treatment of a noninflamed pulp*. Treatment of a healthy pulp has been shown to be an important requirement for successful therapy.[174,183] Vital pulp therapy of the inflamed pulp yields an inferior success rate,[174,183] so the optimal time for treatment is in the first 24 hours when pulp inflammation is superficial. As time between the injury and therapy increases, pulp removal must be extended apically to ensure that noninflamed pulp has been reached.
- *Bacteria-tight seal*. This requirement can be considered the most critical factor for successful treatment.[174] Challenge by bacteria during the healing phase causes failure (see also Chapter 15).[53] If the exposed pulp is effectively sealed from bacterial leakage, successful healing of the pulp with a hard-tissue barrier will occur independent of the dressing placed on the pulp and after more extended time periods between accident and treatment.[54,88]
- *Pulp dressing*. Calcium hydroxide has traditionally been used for vital pulp therapy. Its main advantage is that it is antibacterial[46,163] and disinfects the superficial pulp. Pure calcium hydroxide causes necrosis of approximately 1.5 mm of pulp tissue, which removes superficial layers of inflamed pulp if present (Fig. 21.10).[135] The high pH (12.5) of calcium hydroxide causes a liquefaction necrosis in the most superficial layers.[156] The toxicity of calcium hydroxide appears to be neutralized as the deeper layers of pulp are affected, causing a coagulative necrosis at the junction of the necrotic and vital pulp, resulting in only mild irritation. This mild irritation initiates an inflammatory response, and in the absence of bacteria,[156] the pulp will heal with a hard-tissue barrier (Fig. 21.11).[155,156] Hard-

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setting calcium hydroxide does not cause necrosis of the superficial layers of pulp; it has also been shown to initiate healing with a hard-tissue barrier.[172,180]

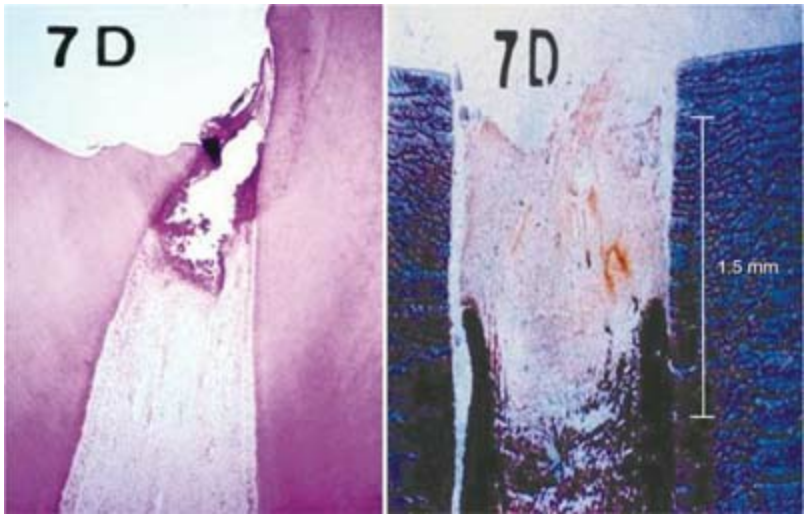

FIG. 21.10 Pulp necrosis of 1.5 mm as a result of the high pH of calcium hydroxide.

1) Stained micrograph after 7 days of traumatic exposure shows a white deposition in the root canal.

2) Micrograph after 7 days shows 1.5 millimeter pulp necrosis as bundle of delicate and proliferated collagen fibers. The bottom area has black deposition.

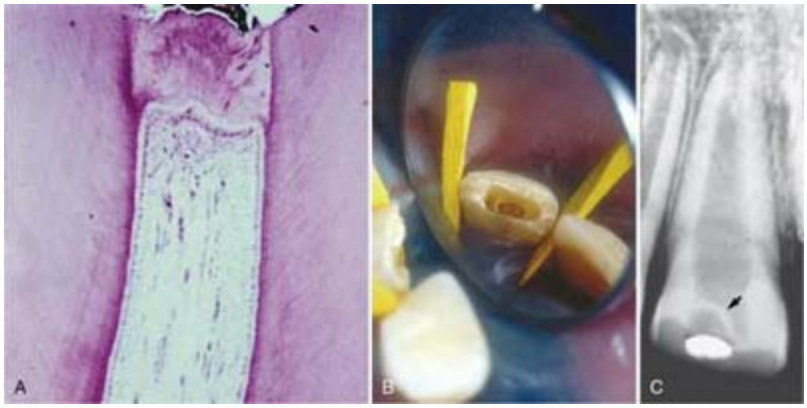

FIG. 21.11 Hard-tissue barrier after calcium hydroxide partial pulpotomy. **A,** Histologic appearance of replacement odontoblasts and a hard-tissue barrier. **B,** Clinical appearance of the barrier on removal of the coronal restoration 3 months after placement of the calcium

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hydroxide. **C,** Radiographic appearance of the hard-tissue barrier *(arrow)*.

A) Stained micrograph shows squarish hard tissue above the root canal.

B) Close-up view of tooth shows cavity with a brown deposition at the center.

C) Radiograph shows tooth with radiolucent root canal and a curved radiopaque line in the crown that also has a radiopaque patch at the tip.

The major disadvantage of calcium hydroxide is that it does not seal the fractured surface. Therefore an additional material must be used to ensure that bacteria do not challenge the pulp, particularly during the critical healing phase.

Many materials, such as zinc oxide eugenol,[30,183] tricalcium phosphate,[98] and composite resin,[29] have been proposed as medicaments for vital pulp therapy. None to this date have afforded the predictability of calcium hydroxide used in conjunction with a well-sealed coronal restoration.[70,122,161,173] In a dog study, for example, pulps capped directly with various adhesive agents showed moderate to severe inflammatory reactions, with progressive extension of tissue necrosis with time and total absence of continuous hard-tissue bridge formation.[122] Application of a calcium hydroxide–based material was characterized by inflammatory cell infiltration, limited tissue necrosis, and partial to complete hard-tissue bridging.[122] In a similar study on human teeth in vivo, it was found that Scotchbond Multi-Purpose Plus (3M, St. Paul, MN) caused inflammatory changes when applied directly to exposed pulp tissue; however, direct capping with Dycal followed by sealing with Scotchbond Multi-Purpose Plus showed favorable results in pulp tissue.[173]

Currently, bioceramic materials are considered the pulp capping agents of choice.[5] Mineral trioxide aggregate (MTA), a first-generation bioceramic material, has been shown to be a good pulp capping agent.[72,139,141,195] It has a high pH, similar to that of calcium hydroxide, when unset[177] and after setting creates an excellent bacteria-tight seal.[178] It is also hard enough to act as a base for a final restoration.[177] Yet MTA does not enjoy the same popularity as calcium hydroxide as a pulp capping agent in the treatment of traumatic exposures due to a prolonged setting time and discoloration of the

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crown because of the bismuth oxide filler in the material.

Recently, newer-generation bioceramic materials (e.g., BioDentine) have come to the market that have similar positive properties as MTA but not the disadvantages described previously. They set quickly enough for a one-visit procedure and do not discolor the tooth.[198] These materials are now considered superior to calcium hydroxide as the capping agent for traumatic pulp exposures.

Treatment methods

Pulp capping

Pulp capping implies placing the dressing directly on the pulp exposure without any removal of the soft tissue.[29]

Indications.

There are few indications for pulp capping when traumatic pulp exposures are treated. The success rate of this procedure (80%),[77,148] compared with partial pulpotomy (95%),[57] indicates that a superficial pulp cap should not be considered after traumatic pulp exposures. The lower success rate is not difficult to understand because superficial inflammation develops soon after the traumatic exposure. If the treatment is at the superficial level, a number of inflamed (rather than healthy) pulps will be treated, lowering the potential for success. In addition, a bacteria-tight coronal seal is much more difficult to attain in superficial pulp capping because there is no depth to the cavity to aid in creating the seal, as there is with a partial pulpotomy.

It must be acknowledged that the studies suggesting that an inflamed pulp is contraindicated for an attempt at pulp capping were performed in the 1970s with amalgam as the standard coronal restoration. From a microleakage perspective, as amalgam leaks and calcium hydroxide washes out in the presence of moisture as the reasons for the poor results, then it may be poor coronal seal, rather than the inflamed pulp, that leads to failure in these cases. More recent studies suggest that with the bioceramic as the pulp cap, the inflamed pulp is not the impediment as previously thought, but rather that the seal seems to be the major factor for success. Thus this material used as a base in these situations may provide more leeway for capping the inflamed

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pulp.

However, in a situation of a traumatic pulp exposure of a tooth (injuries in which most patients are young [large pulps]), the patient typically presents for treatment within 48 hours because of sensitivity and esthetic concerns. Therefore the pulpal inflammation is usually only superficial. In these cases, it may be prudent to remove the superficial layer of inflammation and place a bioceramic agent for pulp capping.

Partial pulpotomy

Partial pulpotomy implies the removal of coronal pulp tissue to the level of healthy pulp. Knowledge of the reaction of the pulp after a traumatic injury makes it possible to accurately determine this level. This procedure is commonly called the *Cvek pulpotomy*.

Indications.

As for pulp capping.

Technique.

Administration of an anesthetic (possibly without a vasoconstrictor), rubber dam placement, and superficial disinfection are performed. A 1- to 2-mm-deep cavity is prepared into the pulp, using a high-speed handpiece with a sterile diamond bur of appropriate size and copious water coolant (Fig. 21.12).[85] A slow-speed bur or spoon excavator should be avoided. If bleeding is excessive, the pulp is amputated deeper until only moderate hemorrhage is seen. Excess blood is carefully removed by rinsing with sterile saline, and the area is dried with a sterile cotton pellet. Use of 5% sodium hypochlorite (NaOCl; bleach) has been recommended to rinse the pulpal wound.[54] The bleach causes chemical amputation of the blood coagulum; removes damaged pulp cells, dentin chips, and other debris; and provides hemorrhage control with minimal damage to the “normal” pulp tissue underneath.

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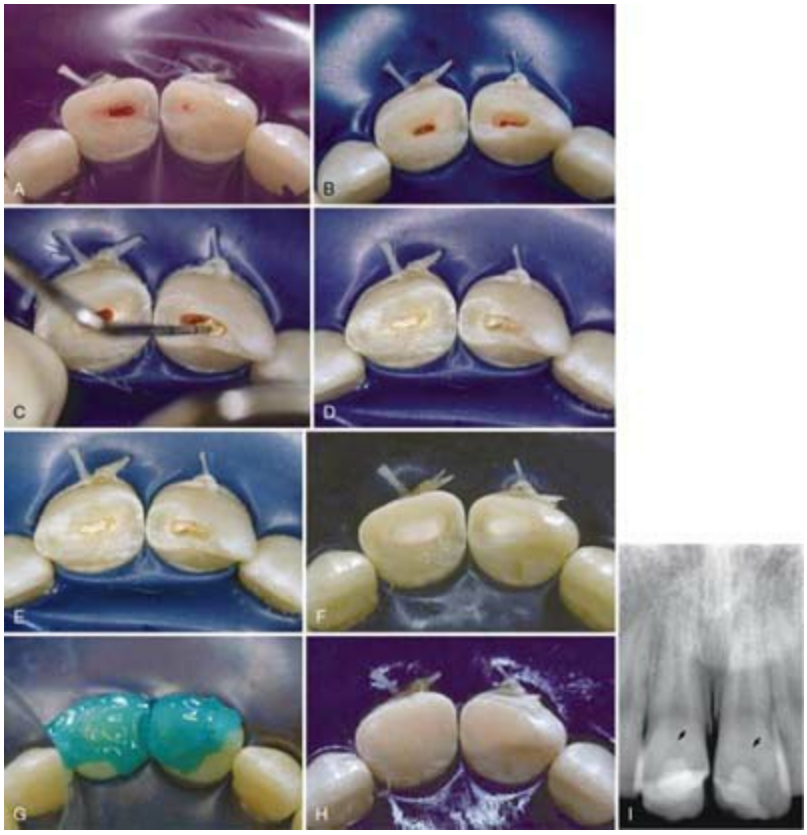

FIG. 21.12 Cvek partial pulpotomy. **A,** The fractured teeth are cleaned and disinfected; a rubber dam is placed. **B,** Cavities are prepared at high speed with a round diamond bur 1 to 2 mm into the pulpal tissue. **C,** Calcium hydroxide on a plugger **(D)** is placed on the soft tissue of the pulp. **E,** Care is taken to avoid smearing the walls of the preparation with the calcium hydroxide. **F,** Cavity preparations are filled with glass ionomer cement. The exposed dentin is etched **(G)** and then covered with composite resin **(H)**. **I,** Radiograph 6 months later shows formation of hard-tissue barriers in both teeth *(arrows)*.

Eight close-up view show four teeth isolated by rubber dam clamps. The close-up views are accompanied by a radiograph.

A) Second tooth on right has a blood stain in the fracture. Third tooth has a fracture with a tiny spot.

B) Both second and third teeth show deep cavities at the center with accumulated fluids.

C) The cavity of third tooth is being filled by using plugger.

D) The cavities of both second and third tooth are partially filled with calcium hydroxide.

E) It is similar to D, except that the image is clearer and the color of the rubber

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clamp is changed.

F) The cavities are completely filled with calcium hydroxide and they appear as smooth surface.

G) Both teeth are covered with plastic material.

H) The plastic covers are removed and teeth have no cavities.

I) Radiograph of both teeth shows normal appearance with partially radiopaque region in the pulp.

Source: (Courtesy Dr. Alessandra Ritter, Chapel Hill, NC.)

Care must be taken not to allow a blood clot to develop because this would compromise the prognosis.[57,155] Calcium hydroxide is carefully placed on the pulp stump. If the pulp size does not permit additional loss of pulp tissue, a commercial hard-setting calcium hydroxide can be used.[159] The prepared cavity is filled with a material with the best chance of a bacteria-tight seal such as a glass ionomer cement to a level flush with the fractured surface. The material in the pulpal cavity and all exposed dentinal tubules are etched and restored with bonded composite resin. Alternatively, after hemostasis has been obtained, the pulp can be capped with MTA, covered with a lining material, and then restored with composite restoration. However, this has been to shown to cause discoloration of the tooth.[35]

Follow-up.

The follow-up is the same as for pulp capping. Emphasis is placed on evidence of maintenance of responses to sensitivity tests and radiographic evidence of continued root development (Fig. 21.13).

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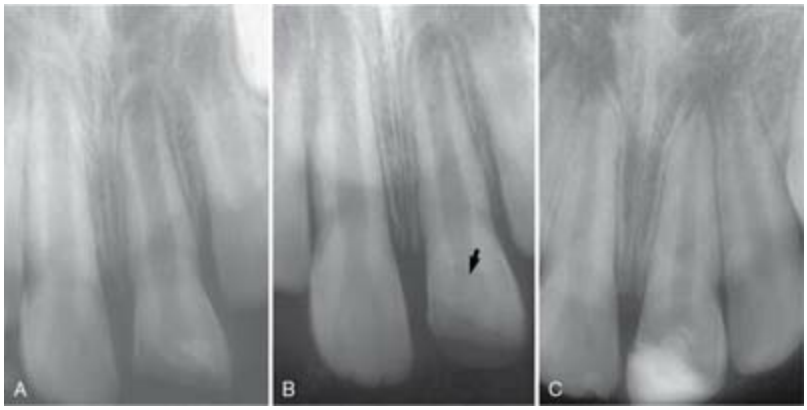

FIG. 21.13 Continued root development after partial pulpotomy. **A,** Radiograph of immature tooth with a complicated crown fracture. **B,** At the time of placement of calcium hydroxide after partial pulpotomy *(arrow)*. **C,** Follow-up radiograph confirming that the pulp maintained vitality and the root continued to develop.

Three radiographs of two teeth are marked A through C.

A) Left tooth has fracture at the edge of crown followed by radiolucency with a radiopaque patch. The canal root canal is wide and radiolucent.

B) Left tooth has hard-density tissue in the pulp.

C) Crown of left tooth shows radiopaque edge at the site of fracture.

Prognosis.

This method affords many advantages over pulp capping. Superficial inflamed pulp is removed during preparation of the pulpal cavity. Calcium hydroxide disinfects dentin and pulp and removes additional pulpal inflammation. Most important, space is provided for a material that can achieve a bacteria-tight seal to allow pulpal healing with hard tissue under optimal conditions. In addition, coronal pulp remains, which allows sensitivity testing to be carried out at the follow-up visits. The prognosis is extremely good (94% to 96%).[57,78]

Full pulpotomy

Full pulpotomy involves removal of the entire coronal pulp to a level of the root orifices. This level of pulp amputation is chosen arbitrarily because of its anatomic convenience. Because inflamed pulp sometimes extends past the canal orifices into the root pulp, many mistakes are made that result in

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treatment of an inflamed rather than a noninflamed pulp.

Indications.

Full pulpotomy is indicated when it is anticipated that the pulp is inflamed to the deeper levels of the coronal pulp. Traumatic exposures after more than 72 hours and carious exposure of a young tooth with a partially developed apex are two examples of cases in which this treatment may be indicated. Because of the likelihood that the dressing will be placed on an inflamed pulp, full pulpotomy is contraindicated in mature teeth. In the immature tooth, benefits outweigh risks for this treatment form only with incompletely formed apices and thin dentinal walls.

Technique.

The procedure begins with administration of an anesthetic, rubber dam placement, and superficial disinfection, as for pulp capping and partial pulpotomy. The coronal pulp is removed as in partial pulpotomy but to the level of the root orifices. A calcium hydroxide dressing, a bacteria-tight seal, and a coronal restoration are carried out as for partial pulpotomy.

Follow-up.

Follow-up is the same as for pulp capping and partial pulpotomy. A major disadvantage of this treatment method is that sensitivity testing is not possible, owing to the loss of coronal pulp, so radiographic follow-up is extremely important to assess for signs of apical periodontitis and to ensure the continuation of root formation.

Prognosis.

Because cervical pulpotomy is performed on pulps that are expected to have deep inflammation and the site of pulp amputation is arbitrary, many more mistakes are made that lead to treatment of the inflamed pulp. Consequently the prognosis, which is in the range of 75%, is poorer than for partial pulpotomy.[82] Because of the inability to evaluate pulp status after full pulpotomy, some authors have recommended pulpectomy routinely after the roots have fully formed (Fig. 21.14). This philosophy is based on the pulpectomy procedure having a success rate in the range of 95%, whereas if

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apical periodontitis develops, the prognosis of root canal treatment drops significantly to approximately 80%.[158,164]

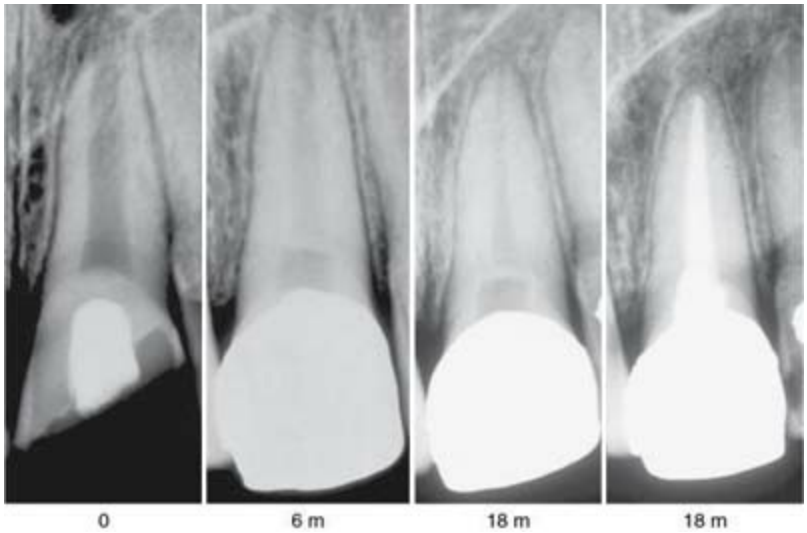

FIG. 21.14 Successful pulpotomy followed by a pulpectomy at 18 months.

Radiograph, marked 0, shows tooth with half-crown fractured diagonally with a radiopaque spot. The root canal is radiolucent.

Radiograph after 6 months shows partial radiopaque crown including fracture site with comparatively radiolucent root canal in tooth. There is a hard-density barrier below the pulp.

Radiograph after 18 months shows more radiopaquicity than that of the radiograph after 6 months.

Another radiograph after 18 months shows filled root canal and crown.

Source: (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

Pulpectomy

Pulpectomy implies removal of the entire pulp to the level of the apical foramen.

Indications.

Pulpectomy is indicated in a complicated crown fracture of mature teeth if conditions are not ideal for vital pulp therapy or if it is foreseeable that

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restoring the tooth would require placement of a post. This procedure is no different from root canal treatment of a vital nontraumatized tooth.

Treatment of the nonvital pulp

Immature tooth: Apexification

Indications.

Apexification should be performed in teeth with open apices and thin dentinal walls in which standard instrumentation techniques cannot create an apical stop to facilitate effective root canal filling.

Biologic consequences.

A nonvital immature tooth presents a number of difficulties for adequate endodontic treatment. The canal is often wider apically than coronally, necessitating the use of a filling technique with softened filling material to mold it to the shape of the apical part of the canal. Because the apex is extremely wide, no barrier exists to stop this softened material from moving into and traumatizing the apical periodontal tissues. In addition, the lack of apical stop and extrusion of material through the canal might result in a canal that is underfilled and susceptible to leakage. An additional problem in immature teeth with thin dentinal walls is their susceptibility to fracture, both during and after treatment.[58]

These problems are overcome by stimulating the formation of a hard-tissue barrier to allow for optimal filling of the canal and by reinforcing the weakened root against fracture, both during and after apexification.[120,176]

Technique

Disinfection of the canal.

In most cases, nonvital teeth are infected,[36,160] so the first phase of treatment is to disinfect the root canal system to ensure periapical healing.[46,61] The canal length is estimated with a parallel preoperative radiograph, and after access to the canals has been prepared, a file is placed to this estimated length. When the length has been confirmed radiographically,

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very light filing (because of the thin dentinal walls) is performed with copious irrigation with 0.5% NaOCl.[62,169] A lower strength of NaOCl is used because of the increased danger of placing the agent through the apex in immature teeth. The increased volume of irrigant used compensates for this lower concentration of NaOCl. An irrigation needle that can passively reach close to the apical length is useful in disinfecting the canals of these immature teeth. The canal is dried with paper points and a creamy mix of calcium hydroxide (toothpaste thickness) spun into the canal with a Lentulo spiral instrument (Fig. 21.15). Additional disinfecting action of calcium hydroxide is effective after its application for at least 1 week,[163] so continuation of treatment can take place any time after 1 week. Further treatment should not be delayed more than 1 month because the calcium hydroxide could be washed out by tissue fluids through the open apex, leaving the canal susceptible to reinfection.

FIG. 21.15 Creamy mix of calcium hydroxide on a Lentulo spiral instrument ready for placement into the canal.

A long filament of calcium hydroxide paste oozes out from Lentulo spiral instrument.

Hard-tissue apical barrier

Traditional method.

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Formation of the hard-tissue barrier at the apex requires a similar environment to that required for hard-tissue formation in vital pulp therapy: a mild inflammatory stimulus to initiate healing and a bacteria-free environment to ensure that inflammation is not progressive.

As with vital pulp therapy, calcium hydroxide is used for this procedure.[58,91,99] Calcium hydroxide is packed against the apical soft tissue with a plugger or thick point to initiate hard-tissue formation. This step is followed by backfilling with calcium hydroxide to completely fill the canal. The calcium hydroxide is meticulously removed from the access cavity to the level of the root orifices, and a well-sealing, temporary filling is placed in the access cavity. A radiograph is taken; the canal should appear to have become calcified, indicating that the entire canal has been filled with the calcium hydroxide (Fig. 21.16). Because calcium hydroxide washout is evaluated by its relative radiodensity in the canal, it is prudent to use a calcium hydroxide mixture without the addition of a radiopaquer, such as barium sulfate. These additives do not wash out as readily as calcium hydroxide, so if they are present in the canal, evaluation of washout is impossible.

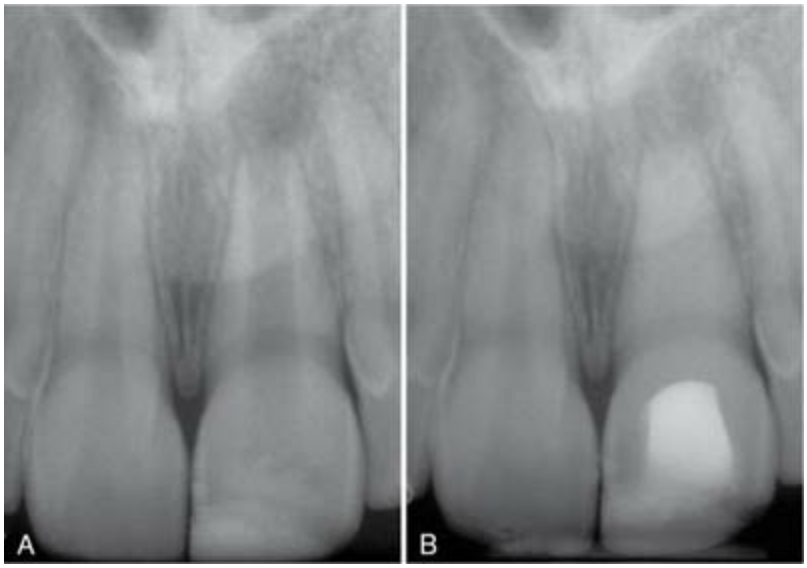

FIG. 21.16 **A** and **B,** Root canal that "disappears" after placement of a thick mix of pure calcium hydroxide and then over time washes out again.

Radiographs of two teeth are marked A and B.

A) The tooth on the left shows wide radiolucent root canal.

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B) The tooth on the left shows partial radiolucent root canal with a radiopaque patch on the crown.

Source: (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

At 3-month intervals, a radiograph is exposed to evaluate whether a hard-tissue barrier has formed and if the calcium hydroxide has washed out of the canal. This is assessed to have occurred if the canal can be seen again radiographically. If no washout is evident, the calcium hydroxide can be left intact for another 3 months. Excessive calcium hydroxide dressing changes should be avoided if at all possible because the initial toxicity of the material is thought to delay healing.[125]

When completion of a hard-tissue barrier is suspected, the calcium hydroxide should be washed out of the canal with NaOCl. A file of a size that can easily reach the apex can be used to gently probe for a stop at the apex. When a hard-tissue barrier is indicated radiographically and can be probed with an instrument, the canal is ready for filling.

Bioceramic barrier.

The creation of a physiologic hard-tissue barrier, although quite predictable, takes anywhere from 3 to 18 months with calcium hydroxide. The disadvantages of this long time period are that the patient is required to present for treatment multiple times and the tooth may fracture during treatment before the thin, weak roots can be strengthened. In addition, one study has indicated that long-term treatment with calcium hydroxide may weaken the roots and make them even more susceptible to fracture.[24] However, this is not conclusive because another more recent study showed that calcium hydroxide did not weaken teeth over time when compared with positive and negative controls and that fracture. Therefore fracture may be more related to the stage of root development rather than calcium hydroxide use.[115]

Bioceramic material has been used to create a hard-tissue barrier quickly after disinfection of the canal (see earlier in the section Traditional Method) (Fig. 21.17). Calcium sulfate is pushed through the apex to provide a resorbable extraradicular barrier against which to pack the bioceramic material. The material is mixed and packed into the apical 4 to 5 mm of the

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canal. Historically, a wet cotton pellet was placed against the bioceramic material and left for at least 6 hours and this is now not considered necessary as long as the bioceramic material does not dry out. The canal is filled with thermoplasticized gutta-percha. The cervical canal is then reinforced with composite resin to below the marginal bone level (described later; also see Fig. 21.17).

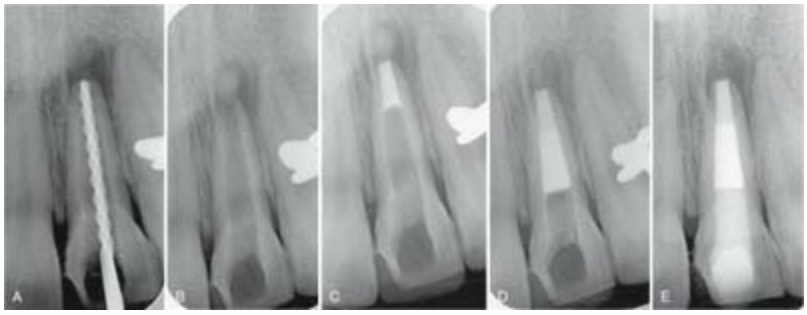

FIG. 21.17 Apexification with mineral trioxide aggregate. **A,** The canal is disinfected with light instrumentation, copious irrigation, and a creamy mix of calcium hydroxide for 1 month. **B,** Calcium sulfate is placed through the apex as a barrier against which the mineral trioxide aggregate (MTA) is placed. **C,** A 4-mm MTA plug is placed at the apex. **D,** The body of the canal is filled with the Resilon obturation system. **E,** A bonded resin is placed below the cementoenamel junction (CEJ) to strengthen the root.

Five radiographs are marked A through E.

A) It shows a spiral instrument being inserted into the root canal of tooth through its crown that has a black large hole.

B) It shows slightly dense tissue at the place of the hole in the tooth. The root has a globular hard tissue at the tip.

C) The one-fourth root canal of tooth is filled at the tip.

D) The half root canal of tooth is filled.

E) Tooth shows filled root canal with radiopaquicity in place of the black hole.

Source: (Courtesy Dr. Marga Ree. Purmerend, Netherlands.)

A number of case reports have been published using this apical barrier technique,[83,101,131] and it has steadily gained popularity with clinicians. Numerous reports attest to the efficacy of this technique.[48,114]

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Filling the root canal.

Because the apical diameter is larger than the coronal diameter of most of these canals, a softened filling technique is indicated in these teeth (see Chapter 9). Care must be taken to avoid excessive lateral force during filling, owing to the thin walls of the root. If the hard-tissue barrier was produced by long-term calcium hydroxide therapy, it consists of irregularly arranged layers of coagulated soft tissue, calcified tissue, and cementum-like tissue (Fig. 21.18). Included also are islands of soft connective tissue, giving the barrier a "Swiss cheese" consistency.[40,56] Because of the irregular nature of the barrier, it is not unusual for cement or softened filling material to be pushed through it into the apical tissues during filling (Fig. 21.19). Formation of the hard-tissue barrier might be some distance short of the radiographic apex because the barrier forms wherever the calcium hydroxide contacts vital tissue. In teeth with wide, open apices, vital tissue can survive and proliferate from the periodontal ligament a few millimeters into the root canal. Filling should be completed to the level of the hard-tissue barrier and not forced toward the radiographic apex.

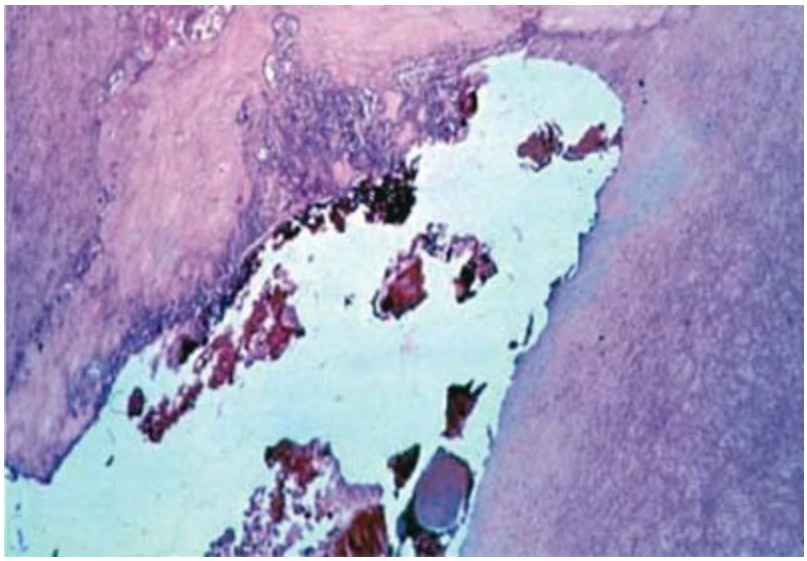

FIG. 21.18 Histologic appearance of hard-tissue barrier after calcium hydroxide apexification. The barrier is composed of cementum and bone with soft-tissue inclusions.

Stained micrograph of tooth shows irregular patches in root along with dense tissues and calcification.

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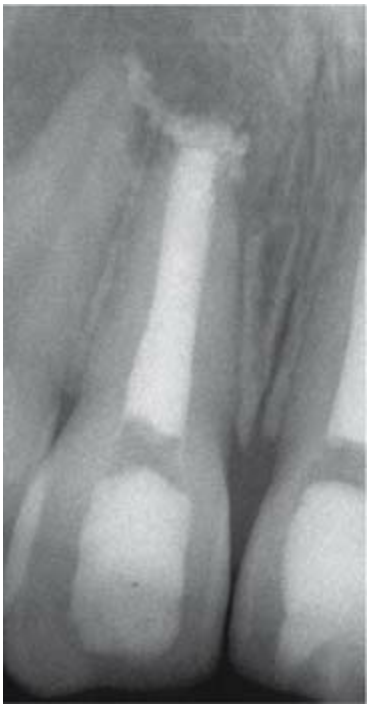

FIG. 21.19 Root filing with a soft technique after calcium hydroxide apexification. Sealer and softened filling material are expressed through the “Swiss cheese” holes in the barrier.

Radiograph shows filled root canal and radiolucent patch in the neck region of tooth.

Reinforcement of thin dentinal walls.

The apexification procedure has become a predictably successful procedure (see the section Prognosis),[76,91] but thin dentinal walls still present a clinical problem. Should secondary injuries occur, teeth with thin dentinal walls are more susceptible to fractures that render them unrestorable.[65,179] It has been reported that approximately 30% of these teeth will fracture during or after endodontic treatment.[120] Consequently, some clinicians have questioned the advisability of the apexification procedure and have opted for more radical treatment procedures, including extraction followed by extensive restorative procedures, such as dental implants. However, this approach cannot be considered in children or young adults because facial growth has not been completed. Studies have shown that intracoronal bonded

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restorations can internally strengthen endodontically treated teeth and increase their resistance to fracture.[84,118] Thus, after root filling, the material should be removed to below the marginal bone level and a bonded resin filling placed (see Fig. 21.17).

Follow-up.

Routine recall evaluation should be performed to determine success in the prevention or treatment of apical periodontitis. Restorative procedures should be assessed to ensure that they in no way promote root fractures.

Prognosis.

Periapical healing and the formation of a hard-tissue barrier occur predictably with long-term calcium hydroxide treatment (79% to 96%).[58,120] However, long-term survival is jeopardized by the fracture potential of the thin dentinal walls of these teeth. It is expected that the newer techniques of internally strengthening the teeth described earlier will increase their long-term survivability.

Pulp revascularization/regenerative endodontic treatment.

Regeneration of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth that was reimplanted within 40 minutes (discussed later). The advantages of pulp revascularization lie in the possibility of further root development and reinforcement of dentinal walls by deposition of hard tissue, thus strengthening the root against fracture (see also Chapter 12). After reimplantation of an avulsed immature tooth, a unique set of circumstances exists that allows regeneration to take place. The young tooth has an open apex and is short, which allows new tissue to grow into the pulp space relatively quickly. The pulp is necrotic but usually neither degenerated nor infected, so it acts as a matrix into which the new tissue can grow. It has been experimentally shown that the apical part of a pulp may remain vital and after reimplantation proliferate coronally, replacing the necrotized portion of the pulp.[30,140,167] Because in most cases the crown of the tooth is intact and caries free, bacterial penetration into the pulp space through cracks[127] and defects will be a slow process. The race between new tissue growth and infection of the pulp space favors the new tissue.[14]

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Traditionally, regeneration of pulp tissue in a necrotic infected tooth with apical periodontitis was thought to be impossible.[138] Regeneration/repair can occur when the canal is effectively disinfected, a matrix into which new tissue can grow is provided, and coronal access is effectively sealed.[30,105] There have been many studies in the past few years aimed at making the procedure more predictable both in healing of apical periodontitis and in promoting pulp and not nonspecific connective tissue (see Chapter 12). A clinical protocol can be found in the AAE "Clinical Considerations for a Regenerative Procedure." However, although there is the potential for further root maturation, a prospective study has shown that increases in root width and length are variable in a cohort of predominantly traumatized teeth (Fig. 21.20).[112]

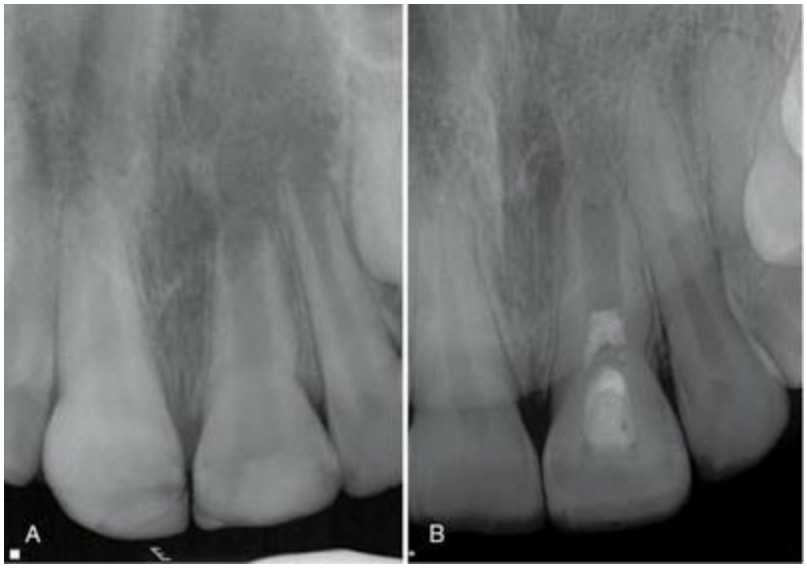

FIG. 21.20 **A,** Preoperative radiograph showing an immature incisor (tooth #9) that sustained an uncomplicated crown fracture where pulp necrosis developed as shown by cessation of root development when compared with tooth #8. **B,** A 6-year review radiograph showing the tooth after treatment with a regenerative endodontic protocol. There were no signs of infection (primary goal). Calcific material at the apex is present indicating apical closure. A quantitative analysis as described by Bose et al.[44] reports a 1.9% increase in root length and a 5% increase in canal wall width (secondary goal). The tooth was responsive to electric pulp testing in a comparable range to tooth #8 (tertiary goal).

Radiograph A shows ninth tooth with wide radiolucent root canal.

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Radiograph B shows two radiopaque patches, one in the crown and the other at the beginning of root.

In regenerative endodontic procedures there are three outcome measures for success. The primary goal is the elimination of symptoms and the evidence of bony healing. The secondary goal is increased root wall thickness and/or increased root length, which is considered desirable but perhaps not essential. The tertiary goal is a positive response to vitality testing (which if achieved, could indicate a more organized vital pulp tissue).[1] It is known that results are variable for these objectives and true regeneration of the pulp/dentin complex is not achieved. Repair derived primarily from the periodontal and osseous tissues has been shown histologically.[34,132]

Disinfection of the canal involved no mechanical preparation, but copious irrigation with NaOCl was performed, followed by placement of an intracanal mixture of antibiotics consisting of equal amounts of ciprofloxacin, metronidazole, and minocycline, to a final concentration of 1 to 5 mg/mL.[1,103] The minocycline in the triantibiotic paste causes unacceptable discoloration of the teeth[113]; this has led to many studies on eliminating or replacing this antibiotic in the paste or eliminating the tripaste. Alternatively, calcium hydroxide can be used as an intracanal medicament rather than an antibiotic paste. Furthermore, Biodentine when used as an intracanal barrier results in less crown discoloration then when MTA is used. Avoiding discoloration of the crown is a patient-centered outcome.

Studies are underway to find a synthetic matrix that can act as a more predictable scaffold for new ingrowth of tissue than the introduction of a blood clot into the canal.[121,194] Studies are also underway on stem cells and growth factors aimed at a more predictable production of odontoblasts and thus pulp cells. It is hoped that with the concept of tissue engineering, namely stem cells, scaffolds, and signaling molecules, that true pulp regeneration is an achievable goal.[121] The procedure described here can be attempted in most cases and has been recommended as treatment of choice for immature teeth with incomplete root formation, although teeth with near or complete root formation may be more suited for conventional endodontic therapy or MTA barrier techniques.[121] If there are no signs of regeneration or continued signs of infection, the more traditional treatment methods for treatment of

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immature teeth can be initiated.

Mature tooth

Conventional endodontic therapy is recommended for mature teeth with closed apex.

Crown-root fracture

Crown-root fracture is a periodontal rather than an endodontic challenge. The tooth must be treated periodontally to enable a well-sealed coronal restoration. This could be accomplished by simple gingivectomy if the extent of the root component of the fracture is large. Alternatively, the tooth could be orthodontically or surgically extruded such that the exposed surface of the root fracture is treatable. Once the feasibility of the coronal restoration is ensured, the particular crown fracture is treated as previously described.

Root fracture

Root fracture implies fracture of the cementum, dentin, and pulp. These injuries are relatively infrequent, occurring in less than 3% of all dental injuries.[199] Immature teeth with vital pulps rarely sustain horizontal root fractures.[10] When a root fractures horizontally, the coronal segment is displaced to a varying degree, but generally the apical segment is not displaced. Because the apical pulpal circulation is not disrupted, pulp necrosis in the apical segment is extremely rare. Permanent pulpal necrosis of the coronal segment, requiring endodontic treatment, occurs in approximately 25% of cases.[11,12,107]

Diagnosis and clinical presentation

The clinical presentation of a root fracture is similar to that of luxation injuries. The extent of displacement of the coronal segment is usually indicative of the location of the fracture and can vary from none, simulating a concussion injury (apical fracture), to severe, simulating an extrusive luxation (cervical fracture).

Radiographic examination for root fractures is extremely important.

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Because root fractures are usually oblique (facial to palatal; Fig. 21.21), one periapical radiograph may easily miss it. It is imperative to take at least three angled radiographs (45 degrees, 90 degrees, and 110 degrees) so that at least at one angulation, the x-ray beam passes directly through the fracture line to make it visible on the radiograph (Fig. 21.22).

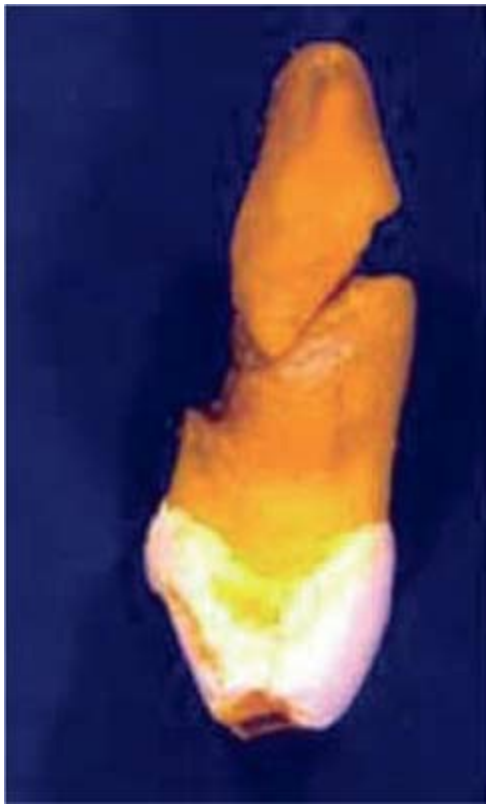

FIG. 21.21 Extracted root after a root fracture. Note the oblique angle of the fracture.

Specimen of tooth shows chipped enamel at the top of crown along with the fracture in the middle of root.

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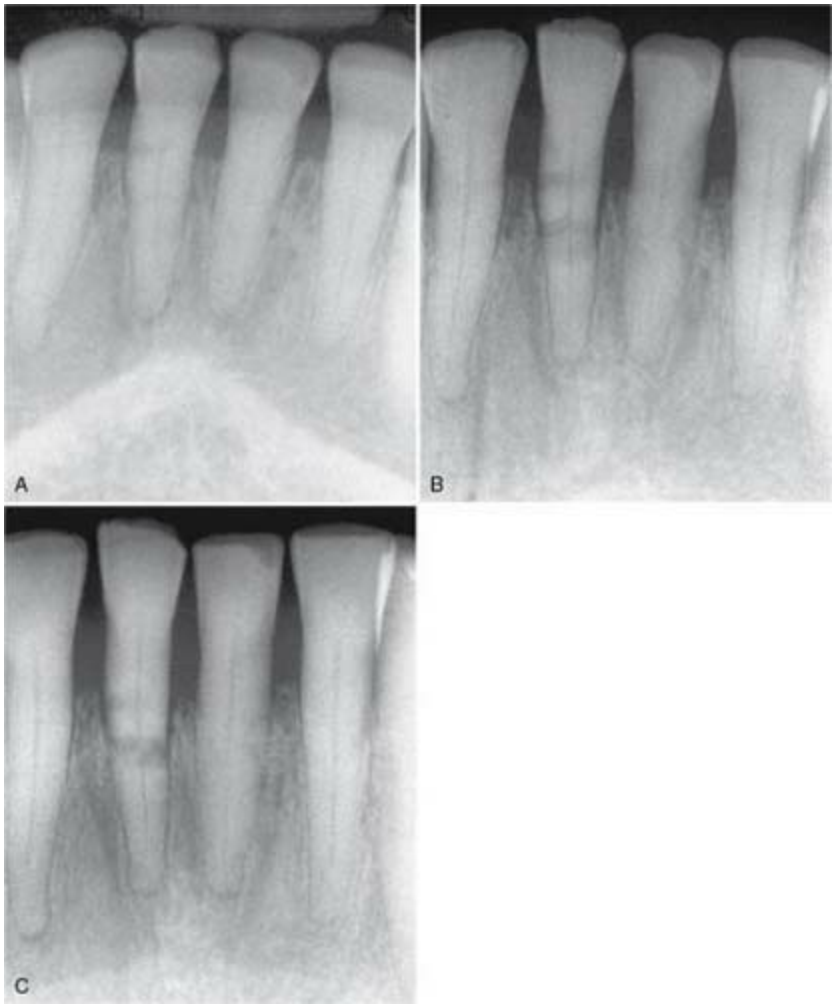

FIG. 21.22 Radiographs showing the importance of different vertical angulations for diagnosis of root fracture. All three radiographs were taken within minutes of each other.

Three radiographs are marked A through C.

A) It shows four incisors with normal anatomy without any fracture mark.

B) Two fracture marks are visible on the right incisor with receding gum line.

C) One light and one wide dark fracture mark is visible on the right incisor.

Treatment

Emergency treatment involves repositioning the segments into close proximity as much as possible. In the case of severe displacement of the coronal segment, its apical extension is frequently lodged in (if not

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perforating through) the cortical bone facial to the tooth. Forcing the crown facially is not possible, and the two segments will not be properly aligned. The only way to accomplish reapproximation of the two segments is to release the coronal segment from the bone by gently pulling it slightly downward with finger pressure or extraction forceps and, once it is loose, rotating it back to its original position (Fig. 21.23). The traditionally recommended[147] splinting protocol has been changed from 2 to 4 months with rigid splinting to a flexible splint to adjacent teeth for 2 to 4 weeks.[23,69,110] If a long time has elapsed between the injury and treatment, repositioning of the segments close to their original position probably will not be possible; this compromises the long-term prognosis for the tooth.

FIG. 21.23 Figure of facial root fracture manipulated back into place.

An arrow points toward the space below the dispositioned root canal. The upper right corner shows an X mark.

Healing patterns

Investigators[26] have described four types of responses to root fractures.

1. *Healing with calcified tissue.* Radiographically, the fracture line is discernible, but the fragments are in close contact (Fig. 21.24, *A*).
2. *Healing with interproximal connective tissue.* Radiographically, the

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fragments appear separated by a narrow radiolucent line, and the fractured edges appear rounded (see Fig. 21.24, *B*).

3. *Healing with interproximal bone and connective tissue*. Radiographically, the fragments are separated by a distinct bony ridge (see Fig. 21.24, *C*).
4. *Interproximal inflammatory tissue without healing*. Radiographically, a widening of the fracture line and/or a developing radiolucency corresponding to the fracture line becomes apparent (see Fig. 21.24, *D*).

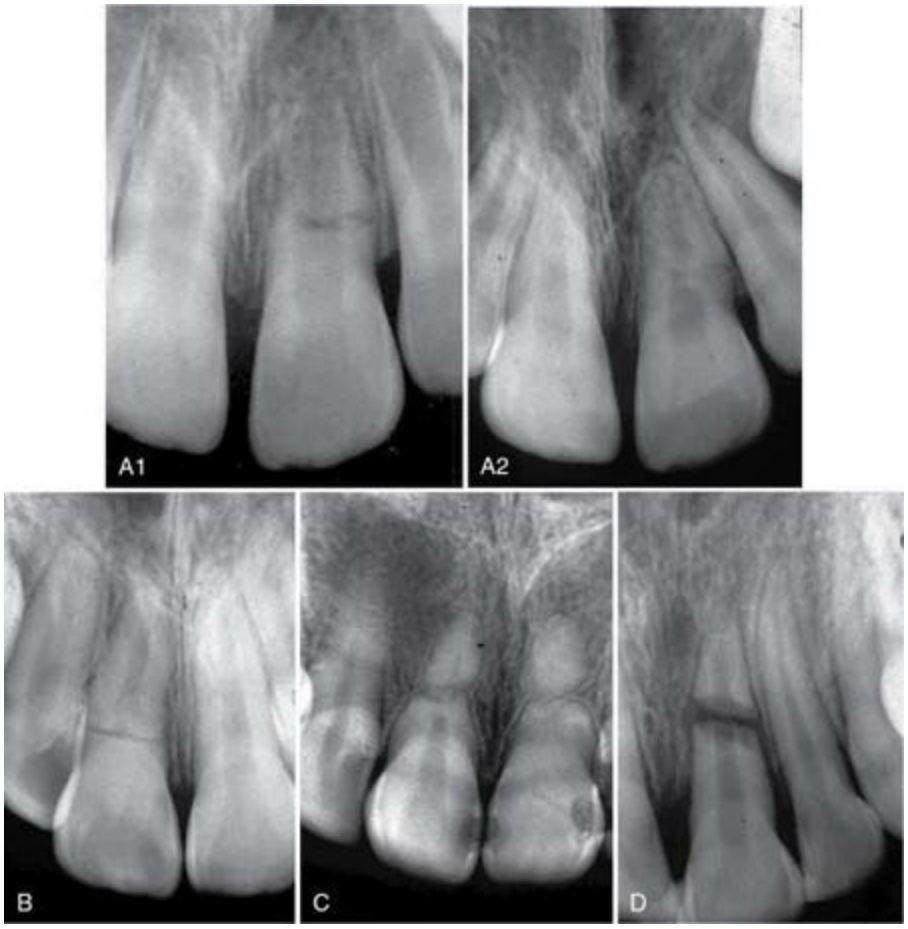

FIG. 21.24 Healing patterns after horizontal root fractures. **A1,** A transverse root fracture in a 7.5-year-old boy. **A2,** Healing with calcified tissue with internal thickening of root dentin into the pulp chamber at a 3-year review. **B,** Healing with interproximal connective tissue. **C,** Healing with bone and connective tissue. Note extensive intracanal calcification of tooth #9. **D,** Interproximal connective tissue without healing.

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Five radiographs are marked A1, A2, B, C, and D.

A1) Ninth tooth has a horizontal radiolucent region in the middle of root with porous tissue in the surrounding.

A2) Ninth tooth has hard-density tissue in the root canal.

B) Tooth has a thin horizontal radiolucent line below the neck region.

C) Tooth has radiolucent patch at the one side of crown and a junction at the middle of root.

D) The root of tooth is broken at the middle.

Source: (Courtesy Professor Geoffrey Heithersay, Adelaide, Australia.)

The first three healing patterns are considered successful. The teeth are usually asymptomatic and respond positively to sensitivity testing. Histologically, healing can be derived from pulpal and periodontal tissues (Fig. 21.25 for healing with calcified tissue; Fig. 21.26 for healing with interproximal connective tissue).[97]

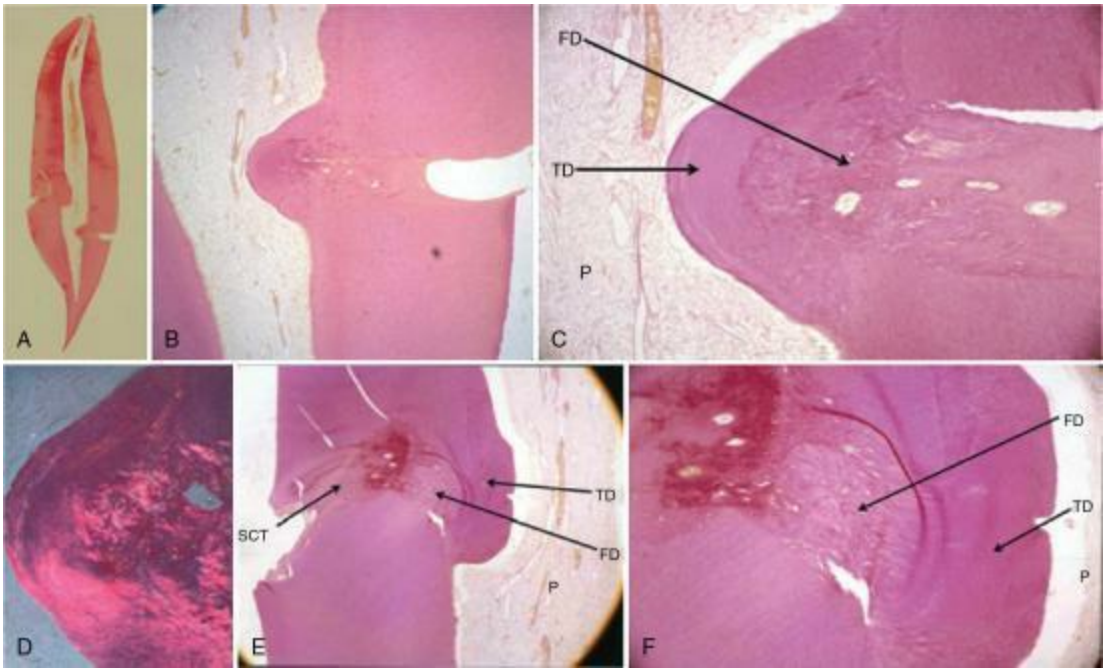

FIG. 21.25 **A,** Sagittal histologic section in a labiopalatal plane showing pulp and hard-tissue deposition between the fractured segments on the labial and to a greater extent on the palatal aspect

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(Van Gieson stain, original magnification ×4). **B,** Labial section showing hard-tissue deposition between approximately half of the fractured dentin surfaces and bulging into the root canal space (Van Gieson stain, original magnification ×10). **C,** Higher-power view of 25A showing fibrillar dentin deposit *(FD)* with some small and larger cell/tissue inclusions and tubular dentin *(TD)* with a sunray orientation bulging into the root canal and lined by pulp (original magnification ×20). **D,** Polarized light view of the pulpal extremity of dentin deposition showing the tubular orientation and deeper irregular hard-tissue formation with cell/tissue inclusions (original magnification ×25). **E,** Palatal section showing hard-tissue deposition between the majority of the fractured dentin surfaces and budging into the root canal. The calcified tissue deposits consist of tubular dentin *(TD)* fibrillar dentin *(FD)* with cell/tissue inclusions, considered to be of pulpal origin *(P)*, and sclerotic calcified tissue *(SCT)*, considered to be of periodontal ligament origin (Van Gieson stain, original magnification ×10). **F,** Higher-power view of the calcified tissue considered to have been pulpally deposited showing further details of the orientation of the dentinal tubules in the tubular dentin layers *(TD)* and the deeper fibrillar dentin layers *(FD)* (original magnification ×20).

Six micrographs are marked A through F.

A) Tooth has stained root canal with two outgrowths along the root line at the center.

B) The magnified view of one of the outgrowth on the dentine surface.

C) The outgrowth is magnified further, in which two labels read, FD and TD. A label in the root canal reads, P.

D) The magnified view of the curved outgrowth shows dark dense-tissue inside.

E) Four labels in and near the fractured site are as follows: SCT, TD, FD, and P.

F) Three labels are FD, TD, and P. FD layers in the tooth are surrounded by TD close to the pulpal region, P.

Source: (Reproduced from Heithersay GS, Kahler B: Healing responses following transverse root fracture: a historical review and case reports showing healing with (a) calcified tissue and (b) dense fibrous connective tissue, *Dent Traumatol* 29:253, 2013.)

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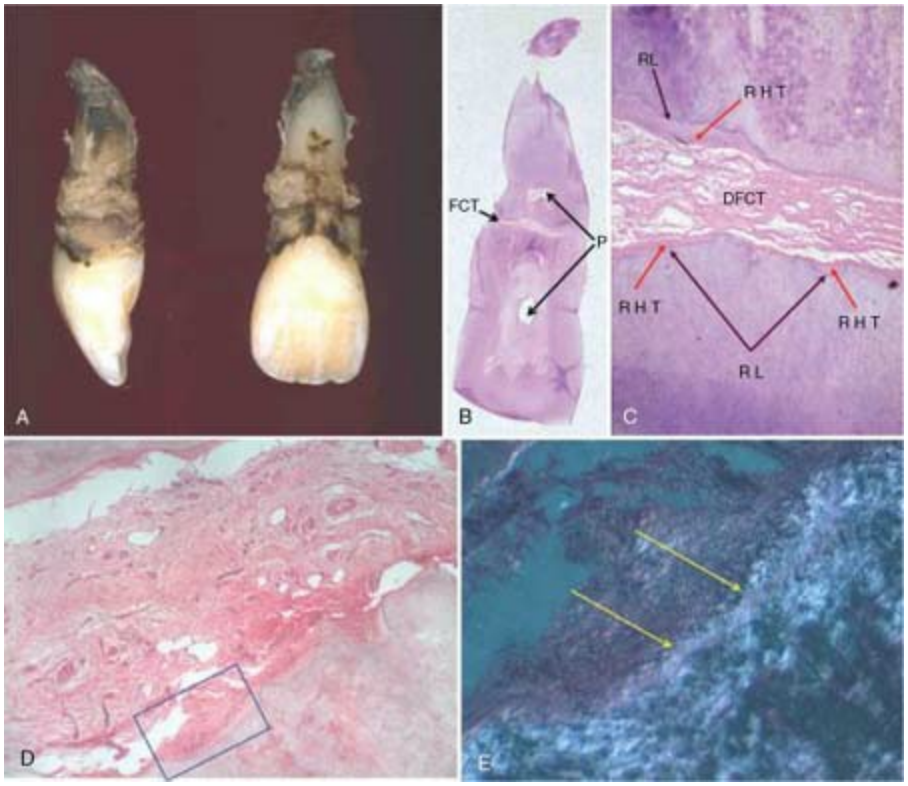

FIG. 21.26 **A,** The intact central incisor after extraction and formalin fixation. **B,** The histologic appearance of the extracted tooth sectioned in a mesiodistal sagittal plane showing the interposition of fibrous connective tissue *(FCT)* calcification of the root canals but evidence of residual pulp tissue *(P)* (H&E, original magnification ×4). **C,** View of the fracture site showing dentin resorption lines *(RL)* repaired with reparative hard tissue *(RHT)* and the interposed dense fibrous connective tissue *(DFCT)* (H&E, original magnification ×10). **D,** Higher-power view showing dense connective tissue, generally with a parallel orientation to the fractured dentin surfaces that show reparative hard tissue laid down on previously resorbed dentin. There is minimal vascularity, but some extravasated erythrocytes are evident (Van Gieson stain, original magnification ×20). **E,** Highlighted section from **(C)** using polarised light to demonstrate Sharpey fiber insertion into the reparative hard tissue *(arrows)* (original magnification ×40).

A) The first view of tooth has a conical crown. The second view of tooth has squared-tapered crown.

B) Stained micrograph shows two pulp residues above and below the FCT at the fracture site in root.

C) Stained micrograph shows the fracture site consisting of DFCT with RHT at margins followed by RL.

D) A rectangular marked area in stained micrograph shows dense connective

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tissues with small spots along with light patches on one side.

E) Two arrows point toward reparative hard tissues at the margins of dense fibrous connective tissue.

Source: (Reproduced from Heithersay GS, Kahler B: Healing responses following transverse root fracture: a historical review and case reports showing healing with (a) calcified tissue and (b) dense fibrous connective tissue, *Dent Traumatol* 29:253, 2013.)

Coronal yellowing is possible because calcific metamorphosis of the coronal segment is not unusual (Fig. 21.27).[108,199]

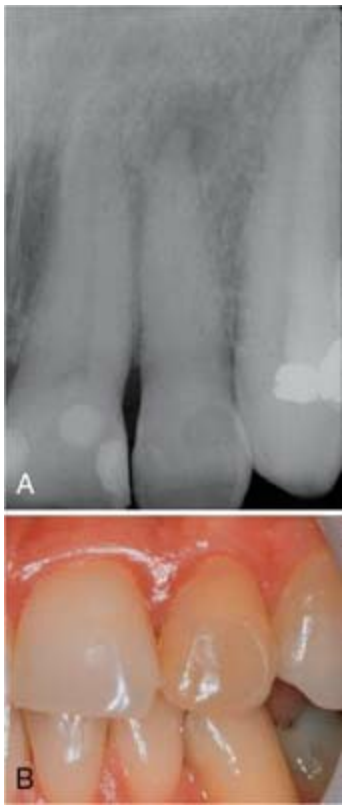

FIG. 21.27 **A,** Pulp canal calcification after a luxation injury. Radiographic appearance of a calcified maxillary lateral incisor; note that despite calcification, it has now become necrotic and infected, evident by the periapical lesion. **B,** The typical yellow appearance of the tooth caused by thickened dentin in the pulp chamber.

A) Radiograph shows white circular patches on the crown of lateral incisor. The central incisor has radiolucent spot on the crown, whereas the incisor on the left has white patches near the neck region.

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B) Close-up view shows reddish-yellow front teeth. The lateral incisor has circular deep patch.

The fourth type of root fracture response is typical when the coronal segment loses its vitality. The infective products in the coronal pulp cause an inflammatory response and typically radiolucencies are at the fracture line (see Fig. 21.24, *D*).[26]

Treatment of complications

Coronal root fractures

Historically it had been thought that fractures in the cervical segment had a poor prognosis, and extraction of the coronal segment was recommended. Research does not support this treatment; in fact, if these coronal segments are adequately splinted, chances of healing do not differ from those for midroot or apical fractures (Fig. 21.28).[199] However, if the fracture occurs at the level of or coronal to the crest of the alveolar bone, the prognosis is extremely poor.

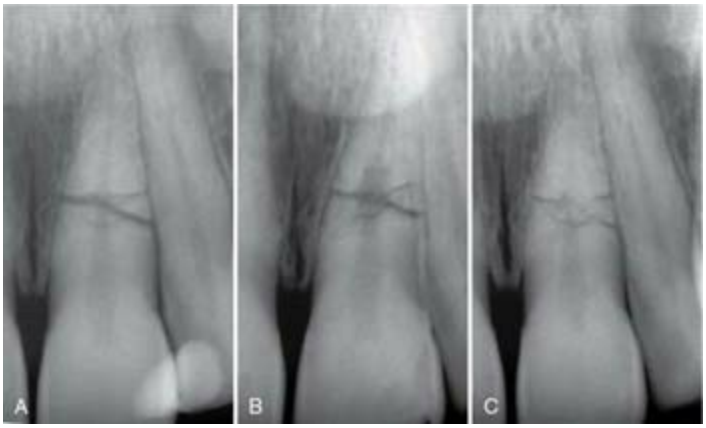

FIG. 21.28 Internal root resorption in the root fracture area; tooth was diagnosed with compound midroot fracture. **A,** Coronal portion was repositioned and then splinted to the lateral incisor with a composite splint. **B,** At 6-month recall, the pulp responded normally to cold, but an internal root-resorptive defect was noted on radiograph. No treatment was indicated, because the pulp did respond normally to vitality tests. **C,** At 42-month recall, the tooth was still asymptomatic, and the pulp still responded to vitality tests. The resorptive defect had healed, and signs of dystrophic calcifications were evident in the apical and coronal

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segments.

Set of three radiographs, marked A through C, compare the fracture in the mid-root of tooth after 6 and 42 month recall.

If reapproximation of the fractured segments is not possible, extraction of the coronal segment is indicated. The level of fracture and length of the remaining root are evaluated for restorability. If the apical root segment is long enough, gentle orthodontic eruption of this segment can be carried out to enable fabrication of a restoration.

Midroot and apical root fractures

Permanent pulpal necrosis occurs in 25% of root fractures.[56,108] Initially it is likely that in many cases the pulp in the coronal segment will become necrotic after the injury; however, because of a very large apical opening in the coronal segment, revascularization is possible if the segments are well reapproximated. In most cases, permanent necrosis occurs in the coronal segment, with only the apical segment remaining vital. Therefore endodontic treatment is indicated in the coronal root segment unless periapical pathology is seen in the apical segment. In most cases the pulpal lumen is wide at the apical extent of the coronal segment, and long-term calcium hydroxide treatment or an MTA apical plug is indicated (see the section Apexification). The coronal segment is filled after a hard-tissue barrier has formed apically in the coronal segment and periradicular healing has taken place.

In rare cases in which both the coronal and apical pulp are necrotic, treatment is more complicated. Endodontic treatment through the fracture is extremely difficult. Endodontic manipulations, medicaments, and filling materials all have a detrimental effect on healing of the fracture site. If healing of the fracture has been completed and is followed by necrosis of the apical segment, the prognosis is much improved.

In more apical root fractures, necrotic apical segments can be surgically removed. This is a viable treatment if the remaining coronal root segment is long enough to provide adequate periodontal support. Removal of the apical segment in midroot fractures leaves the coronal segment with a compromised attachment.

Follow-up

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After the splinting period is completed, follow-up is as for all dental traumatic injuries: at 3, 6, and 12 months and yearly thereafter for at least 5 years.

Prognosis

The following factors influence repair:

1. The degree of dislocation and mobility of the coronal fragment are extremely important in determining outcome.[10,22,108,170,199] Increased dislocation and coronal fragment mobility result in a poorer prognosis.
2. Immature teeth are seldom involved in root fractures, but in the unlikely event they are, the prognosis is good.[22,106]
3. The quality of treatment is vital to successful repair. The prognosis improves with quick treatment, close reduction of the root segments, and semirigid splinting for 2 to 4 weeks.[23]

Complications include pulp necrosis and root canal obliteration. Pulp necrosis can be treated successfully[56,108] by treating the coronal segment with calcium hydroxide to stimulate hard-tissue barrier formation. Root canal obliteration is common if the root segment (coronal or apical) remains vital.

Luxation injuries

Definitions

The types of luxation injury can be defined as follows:

1. *Concussion* implies no displacement, normal mobility, and sensitivity to percussion.
2. *Subluxation* implies sensitivity to percussion, increased mobility, and no displacement.
3. *Lateral luxation* implies displacement labially, lingually, distally, or incisally.
4. *Extrusive luxation* implies displacement in a coronal direction.

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5. *Intrusive luxation* implies displacement in an apical direction into the alveolus.

Definitions 1 through 5 describe injuries of increasing magnitude in terms of intensity of the injury and subsequent sequelae.

Incidence

Luxation injuries as a group are the most common of all dental injuries, with a reported incidence ranging from 30% to 44%.[63]

Treatment

Teeth that are concussed or subluxated do not need any immediate treatment. Responses to vitality tests should be investigated and noted. Even after mild injury, such as subluxation, the pulp might be unresponsive to vitality tests for several weeks if not months.[166] When the pulp is unresponsive initially after the trauma, patients should be recalled on a regular basis and monitored for any additional signs of infection of the root canal (discussed later).

Teeth with lateral and extrusive luxation should be repositioned as soon as possible. In lateral luxation, the apex might be perforating the facial bone plate, and the tooth must be slightly and gently pulled down to loosen the hold before it is repositioned in its original position. Current IADT guidelines call for 2 weeks of physiologic splinting in cases of extrusion luxation and 4 weeks for lateral luxation. A decision on root canal therapy follows the guidelines for avulsion (discussed later). If the tooth has a fully formed apex and was diagnosed to have moved into (if not through) the cortical plate (apical translocation), there is a good likelihood of the pulp being devitalized; therefore endodontic treatment should be initiated as early as 2 weeks after the injury. If the apex is still not fully formed, waiting for signs of revascularization is strongly recommended.

Permanent teeth that are intruded are not likely to spontaneously reerupt, especially if the apex is fully formed.[119] Alternative treatment, such as orthodontic extrusion or immediate surgical repositioning, should be considered. If orthodontic extrusion is planned for an intruded tooth, it should be initiated as soon as possible and should not be delayed longer than 2 to 3 weeks after the trauma. Only a few studies have evaluated the true efficacy of

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this approach. One study[189] using a dog model indicated that severely intruded teeth showed signs of ankylosis as early as 11 to 13 days after the trauma, despite initiation of orthodontic movement 5 to 7 days after the injury.

For the surgical approach, one study[55] (using a dog model) concluded that "a careful immediate surgical repositioning of a severely intruded permanent tooth with complete root formation has many advantages with few disadvantages." Another investigation,[67] a retrospective study of 58 intruded human teeth, indicated that surgical repositioning resulted in a predictable outcome, with only five of the teeth lost over the observation period. However, it was also observed that less surgical manipulation positively influenced the healing. If an intruded tooth is immediately reimplanted, it should be splinted for at least 4 weeks, but in most cases the splint needs to be left on the tooth longer.

Biologic consequences

Luxation injuries result in damage to the attachment apparatus (periodontal ligament and cemental layer), the severity of which depends on the type of injury sustained (concussion least, intrusion most). The apical neurovascular supply to the pulp is also affected to varying degrees, resulting in an altered or nonvital tooth.

Healing can be favorable or unfavorable. Favorable healing after a luxation injury occurs if the initial physical damage to the root surface and the resultant inflammatory response to the damaged external root surface are again covered with cementum. An unfavorable response occurs when there is direct attachment of bone to the root, with the root ultimately being replaced by bone.

There are two resorption responses in which the pulp plays an essential role:

1. In *external* inflammatory root resorption, the *necrotic infected pulp* provides the stimulus for periodontal inflammation in the ligament space. If the cementum has been damaged, the inflammatory stimulators in the pulp space are able to diffuse through the dentinal tubules and stimulate an inflammatory response over large areas of

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the periodontal ligament. Because of the lack of cemental protection, the periodontal inflammation includes root resorption in addition to the expected bone resorption.

2. With *internal* inflammatory root resorption, the inflamed pulp is the tissue involved in resorbing the root structure. The pathogenesis of internal root resorption is not completely understood. Here it is thought that coronal necrotic infected pulp provides a stimulus for a pulpal inflammation in the more apical parts of the remaining vital pulp.[10] If the internal root surface has lost its precemental protection during an injury, internal root resorption occurs in the area adjacent to the inflamed pulp. Thus both the necrotic infected pulp and the inflamed pulp contribute to this type of root resorption.

External root resorption

Caused by an injury (alone) to the external root surface.

If an injury harms the attachment, the by-products of this mechanical damage stimulate an inflammatory response. The healing response depends on the extent of the initial damage.

Localized injury: Healing with cementum.

When the injury is localized (e.g., after concussion or subluxation injury), mechanical damage to the cementum occurs, resulting in a local inflammatory response and a localized area of root resorption. If no further inflammatory stimulus is present, periodontal healing and root surface repair occur within 14 days (Fig. 21.29).[86] The resorption is localized to the area of mechanical damage, and treatment is not required because it is free of symptoms and not even visualized radiographically in most cases. However, in a minority of cases, small radiolucencies can be seen on the root surface if the radiograph is taken at a specific angle. It is important to avoid misinterpreting these cases as progressive. The pulp is not involved. If the pulp responds to sensitivity tests, this is a clue that no treatment should be performed. A wait-and-see attitude can allow spontaneous healing to take place. It is important to realize that in the initial stages, radiolucency could be followed by spontaneous repair, and endodontic treatment should *not* be

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initiated.

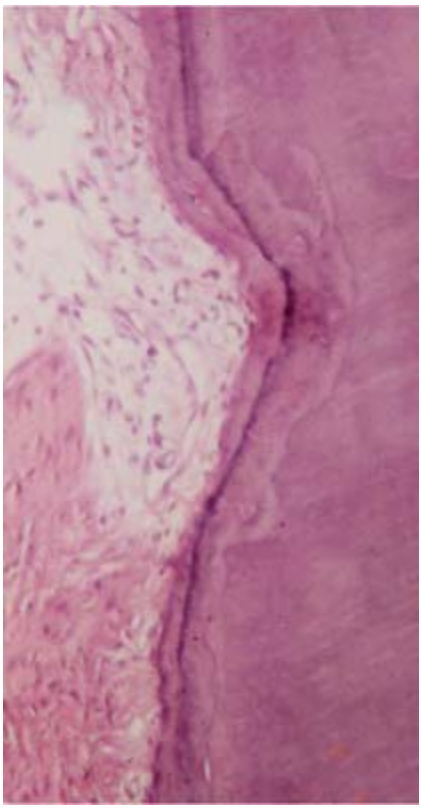

FIG. 21.29 Histologic section showing previous root resorptive defect healed with new cementum and periodontal ligament.

Stained micrograph shows delicate connective tissue with dark spots on the left of vertical line that extends toward the right at the center.

Diffuse injury: Healing by replacement resorption.

When the traumatic injury is severe (e.g., intrusive luxation or avulsion with extended dry time), involving diffuse damage on more than 20% of the root surface, an abnormal attachment can occur after healing takes place.[126] The initial reaction, as always, is inflammation in response to the severe mechanical damage to the root surface. After the initial inflammatory response, a diffuse area of root surface devoid of cementum results. Cells in the vicinity of the denuded root now compete to repopulate it. Often cells that are precursors of bone, rather than the slower-moving periodontal ligament cells, move across from the socket wall and populate the damaged root. Bone

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comes into contact with the root without an intermediate attachment apparatus. This phenomenon is termed *dentoalveolar ankylosis*.[126] Ankylosis results in failure of the tooth to erupt, leading to infraposition in the developing dentition and a high-pitch sound to percussion. However, the ankylosis and replacement resorption that follows cannot be reversed and can be considered a physiologic process because bone resorbs and reforms throughout life. Thus root is resorbed by osteoclasts, but in the reforming stage, bone is laid down instead of dentin, slowly replacing the root with bone. This process is termed *replacement resorption* (Fig. 21.30).[8,19] However, in these traumatic cases, the resorptive phase includes the root being "replaced" with bone (Fig. 21.31). An alternative term of "osseous replacement" has been used.[186]

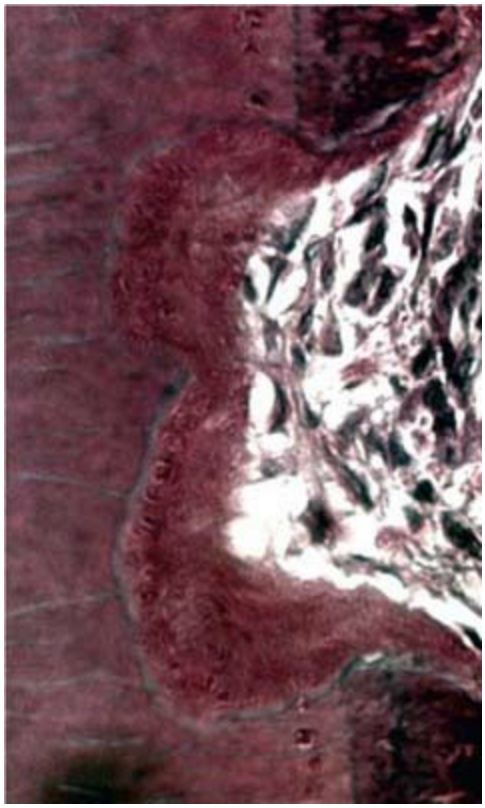

FIG. 21.30 Histologic appearance of replacement resorption. There is direct contact between bone and root structure. Resorptive defects are seen in the bone and root tissue, which is the normal physiologic process of bone turnover. The resorptive defects are filled by new bone and additional areas resorbed. In this way the entire root is replaced by bone at a rate dependent on the metabolic rate of the patient.

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Micrograph shows inflammatory superficial root resorption.

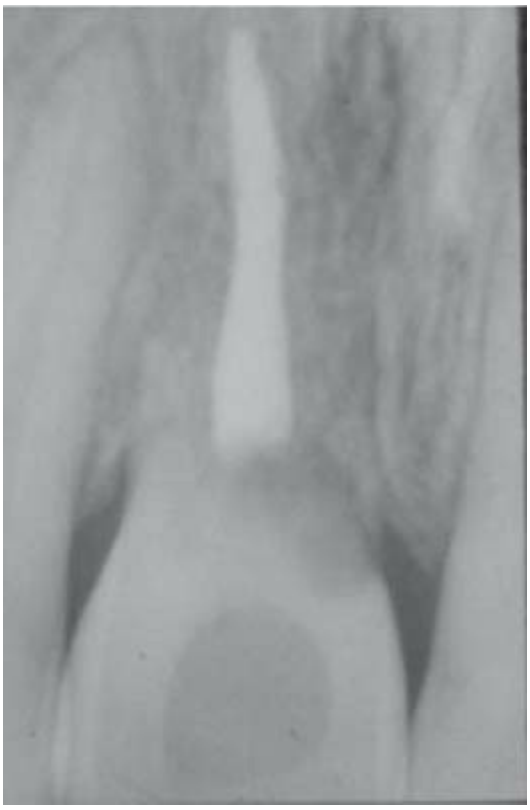

FIG. 21.31 Radiographic appearance of replacement resorption. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are not present.

Radiograph of tooth shows filled root canal and squarish dark patch on the crown.

Treatment.

Treatment strategies for limiting the effect of the traumatic injury to the periodontal structures are outside the scope of this chapter. However, in general they involve minimizing the initial inflammatory response to the injury. The initial inflammation is destructive in nature and increases the surface area of root to be covered in the healing phase.[25] The smaller the surface area to be covered by new cementum, the higher the chances of favorable repair.

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A study has indicated that if Ledermix, a drug combining corticosteroid and tetracycline, is placed in the root canal immediately after a severe trauma in which replacement resorption is expected, favorable healing occurs at a very high rate.[45] In a more recent study, it was shown that triamcinolone (the corticosteroid portion of the Ledermix paste) was as effective as Ledermix at inhibiting external root resorption.[49] Both of these studies were done on young dogs and need to be replicated in human studies.

Caused by an injury to the external root surface and inflammatory stimulus in the root canal.

Recognized inflammatory stimuli that cause root resorption are pressure, pulp space infection, and sulcular infection. This chapter focuses on pulp space infection.

Consequences of apical neurovascular supply damage

Pulp canal obliteration (calcification).

Pulp canal obliteration is common after luxation injuries (see Fig. 21.27). The frequency of pulp canal obliteration appears inversely proportional to pulp necrosis. The exact mechanism of pulp canal obliteration is unknown. It has been theorized that the sympathetic/parasympathetic control of blood flow to the odontoblasts is altered, resulting in uncontrolled reparative dentin.[10,20] Another theory is that hemorrhage and blood clot formation in the pulp after injury form a nidus for subsequent calcification if the pulp remains vital.[10,20] One study[16] found that pulp canal obliteration could usually be diagnosed within the first year after injury and was more frequent in teeth with open apices (>0.7 mm radiographically), those with extrusive and lateral luxation injuries, and those that had been rigidly splinted.[16]

Pulp necrosis.

The factors most important for the development of pulp necrosis are the type of injury (concussion least, intrusion most) and the stage of root development (mature apex more than an immature apex).[15] Pulp necrosis will most likely to lead to infection of the root canal system, with problematic consequences.

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Pulp space infection.

Pulp space infection in conjunction with damage to the external root surface results in periradicular root and bone resorption and continues in its active state as long as the pulpal stimulus (infection) remains. When the root loses its cemental protection, lateral periodontitis with root resorption can result (Fig. 21.32). CBCT imaging can show the extent of the resorption (Fig. 21.33).

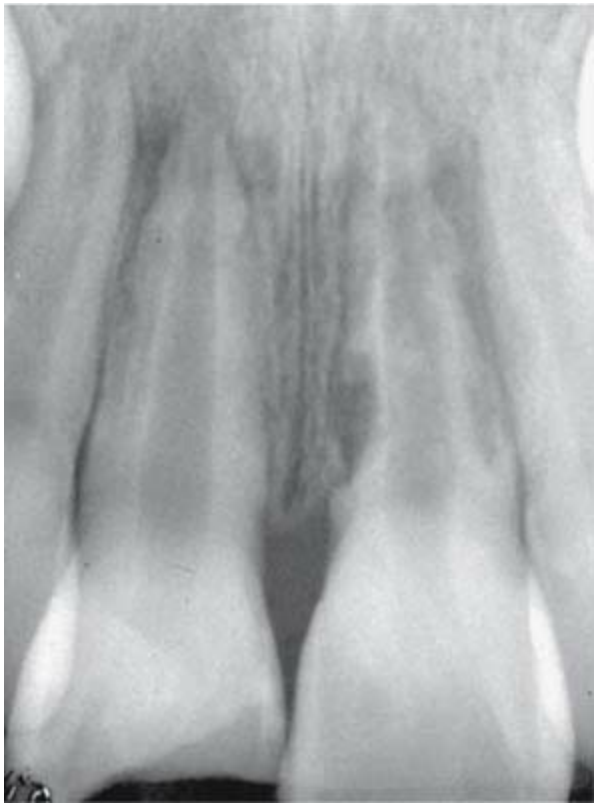

FIG. 21.32 Inflammatory root resorption caused by a pulp space infection. Note the radiolucencies in the root and surrounding bone.

Radiograph shows two teeth. The first tooth has chipped crown at the edge and wedge-shaped radiolucency in the root canal. The second tooth has radiolucent root canal with a dark patch on one side.

Source: (Courtesy Dr. Fred Barnett.)

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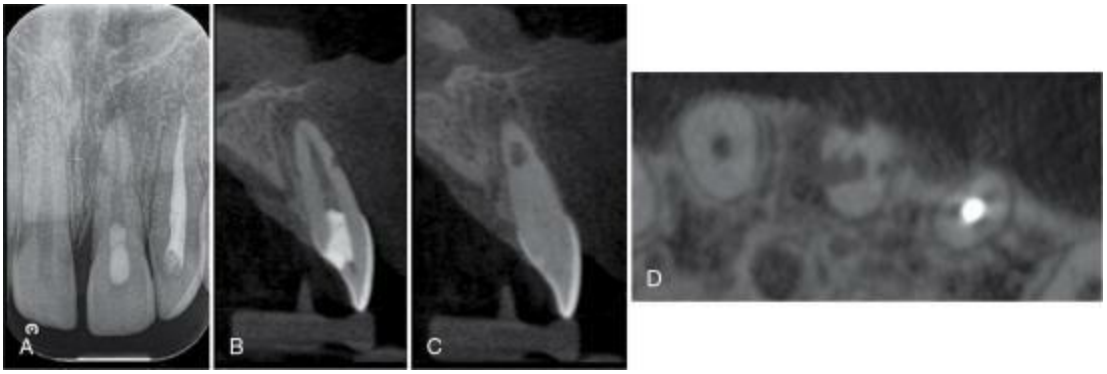

FIG. 21.33 **A,** Periapical radiograph of tooth #9 of a 16-year-old boy showing a mature tooth 6 months after avulsion and replantation within 5 minutes. Inflammatory root resorption is occurring as endodontic treatment was not commenced. **B** and **C,** CBCT sagittal images taken same day as the periapical radiograph. **D,** CBCT axial images.

Four radiographs are marked A through D.

A) It shows 9th tooth with two fused radiopaque patches in the root canal. 10th tooth has filled root canal

B) 9th tooth shows partially filled root canal extending from the crown.

C) Tooth shows a radiolucent oval near the root apex.

D) Teeth arch shows tooth on the left with radiopaque central cavity.

To have pulp space infection, the pulp must first become necrotic. Necrosis occurs after a fairly serious injury in which displacement of the tooth results in severing of the apical blood vessels. In mature teeth, pulp regeneration cannot occur, and usually by 3 weeks, the necrotic pulp becomes infected. (For details of the typical bacterial contents of a traumatized necrotic pulp, the reader is referred to Chapter 15 or to Bergenholtz.[36]) Because a serious injury is required for pulp necrosis, areas of cemental covering of the root usually are also affected, resulting in loss of its protective (insulating) quality. Now bacterial toxins can pass through the dentinal tubules and stimulate an inflammatory response in the periodontal ligament. The result is resorption of the root and bone. The periodontal infiltrate consists of granulation tissue with lymphocytes, plasma cells, and polymorphonuclear leukocytes. Multinucleated giant cells resorb the denuded root surface, and this continues until the stimulus (pulp space bacteria) is removed (Fig. 21.34).[181] Radiographically, the resorption is observed as progressive radiolucent areas

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of the root and adjacent bone (see Fig. 21.32).

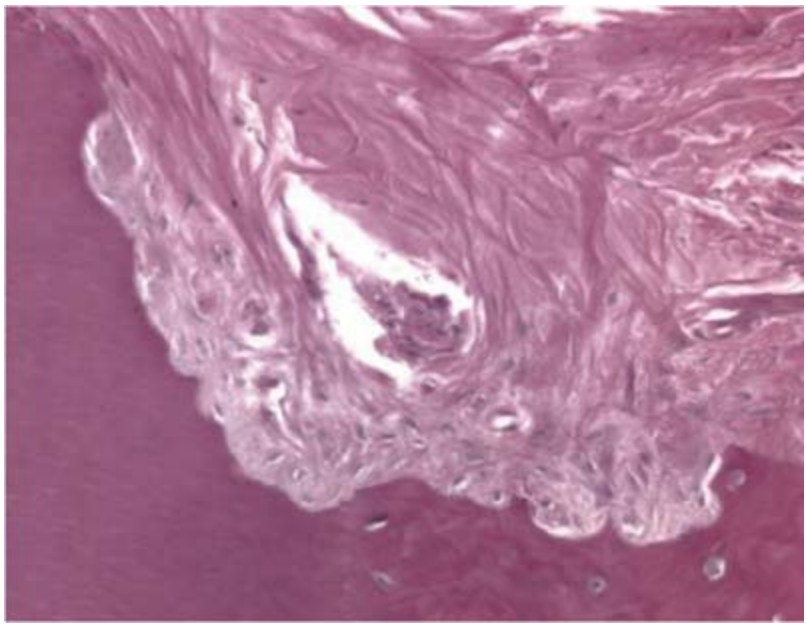

FIG. 21.34 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the dentin of the root.

Stained micrograph shows large multinucleated cells scattered in the bone cavity consisting of dense fibroblastic cells.

Treatment.

Assessing attachment damage caused by the traumatic injury and minimizing the subsequent inflammation should be the focus of the emergency visit. The clinician's attention to pulp space infection should ideally be 7 to 10 days after the injury.[187,188] Root canal disinfection removes the stimulus to the periradicular inflammation, thus the resorption will stop.[86,187,188] In most cases a new attachment will form, but if a large area of root is affected, replacement resorption can result by the mechanism already described. Again, treatment principles include prevention of pulp space infection or elimination of the bacteria if they are present in the pulp space.

1. Prevention of pulp space infection
    a. *Reestablish the vitality of the pulp*. If the pulp stays vital, the canal will be free of bacteria, and external inflammatory root

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resorption will not occur. In severe injuries in which vitality has been lost, it is possible under some circumstances to promote revascularization of the pulp. Revascularization is possible in young teeth with incompletely formed apices if the teeth are replaced in their original position within 60 minutes of the injury (Fig. 21.35).[59] If the tooth has been avulsed, soaking it in doxycycline for 5 minutes or covering the root with minocycline powder before replantation has been shown to double or triple the revascularization rate.[59,150] However, even under the best conditions, revascularization fails to occur on many occasions, and a diagnostic dilemma results. If the pulp revascularizes, external root resorption will not occur, and the root will continue to develop and strengthen. If the pulp becomes necrotic and infected, the subsequent external inflammatory root resorption that develops could result in loss of the tooth in a very short time. At present, the diagnostic tools available cannot detect a vital pulp in this situation before approximately 6 months after successful revascularization. This period of time is obviously unacceptable because by that time the teeth that have not revascularized could be lost to the resorption process. Recently the laser Doppler flowmeter or the pulse oximeter has been shown to have diagnostic potential for the detection of revascularization in immature teeth (see Fig. 21.2). These devices appear to detect the presence of vital tissue in the pulp space by 4 weeks after the traumatic injury.[197]

b. *Prevent root canal infection by initiating root canal treatment at 7 to 10 days*. In teeth with closed apices, revascularization cannot occur. These teeth should be endodontically treated within 7 to 10 days of the injury, before the ischemically necrosed pulp becomes infected.[187,188] Endodontic treatment is extremely difficult so soon after a serious traumatic injury. It is beneficial to start the endodontic treatment with chemomechanical preparation, after which an intracanal dressing with a creamy mix of calcium hydroxide is placed (see Fig. 21.15).[187] Then, the clinician can fill the canal at his or her

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convenience after periodontal healing of the injury is complete, approximately 1 month after the instrumentation visit. There appears to be no necessity for long-term calcium hydroxide treatment in cases in which the endodontic treatment is started within 10 days of the injury.[187]

2. *Eliminate pulp space infection*. When root canal treatment is initiated later than 10 days after the accident or if active external inflammatory resorption is observed, the preferred antibacterial protocol consists of microbial control followed by long-term dressing with calcium hydroxide.[188] Calcium hydroxide can effect an alkaline pH in the surrounding dentinal tubules (Fig. 21.36), kill bacteria, and neutralize endotoxin, a potent inflammatory stimulator.

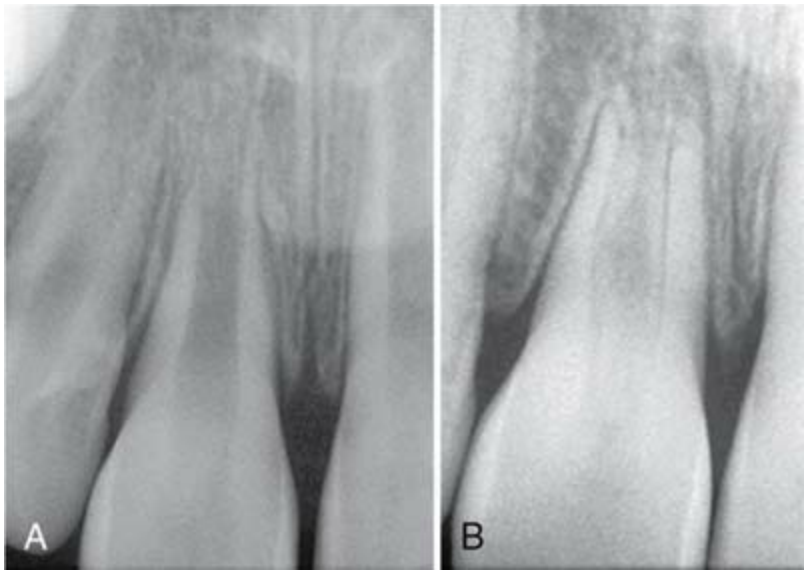

FIG. 21.35 Revascularization of immature root. A tooth with an open apex was replanted soon after the avulsion. The checkup radiograph 12 years later confirms that regrowth has taken place into the pulp chamber. It appears that a bone plug has grown in, and new periodontal ligament has formed, with a lamina dura within the pulp canal.

Two radiographs are marked A and B.

A) The central tooth has open root tip and radiolucency extended below the neck region.

B) The tooth has radiopaque root canal and a partial radiolucent patch (oval-

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shaped) in the root.

Source: (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

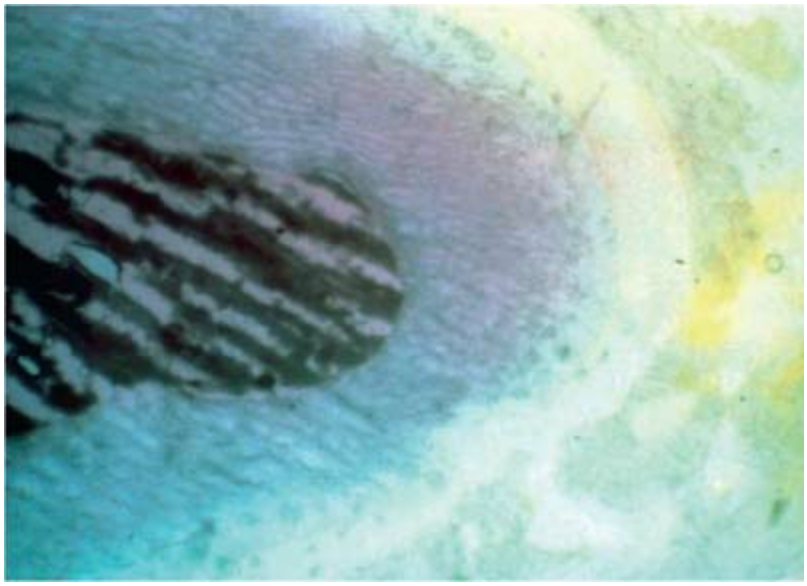

FIG. 21.36 High pH of calcium hydroxide. The root was filled with calcium hydroxide and then cut in cross section. A pH indication shows the high pH in the canal and surrounding root, whereas the surrounding tissue is a neutral pH.

Root apex shows oblique black lines surrounded by pinkish blue color, which is further followed by a white margin and then greenish-pink color with a tinge of yellow on one side.

The first visit consists of the microbial control phase, with cleaning and shaping of the canal and the placement of either a corticosteroid/antibiotic material or a creamy mix of calcium hydroxide using a Lentulo spiral. The patient is seen in approximately 1 month, at which time the canal is filled with a dense mix of calcium hydroxide. Once filled, the canal should appear radiographically to be calcified because the radiodensity of calcium hydroxide in the canal is usually similar to that of the surrounding dentin (see Fig. 21.16). A radiograph is then exposed at 3-month intervals. At each visit the tooth is tested for symptoms of periodontitis. In addition to stopping the resorptive process, calcium hydroxide washout is assessed. Because the root surface is so radiodense as to make assessment of healing difficult, the

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adjacent bone healing is assessed. If adjacent bone has healed, it is assumed that the resorptive process has stopped in the root as well; then the canal can be obturated with the permanent root filling material (Fig. 21.37).

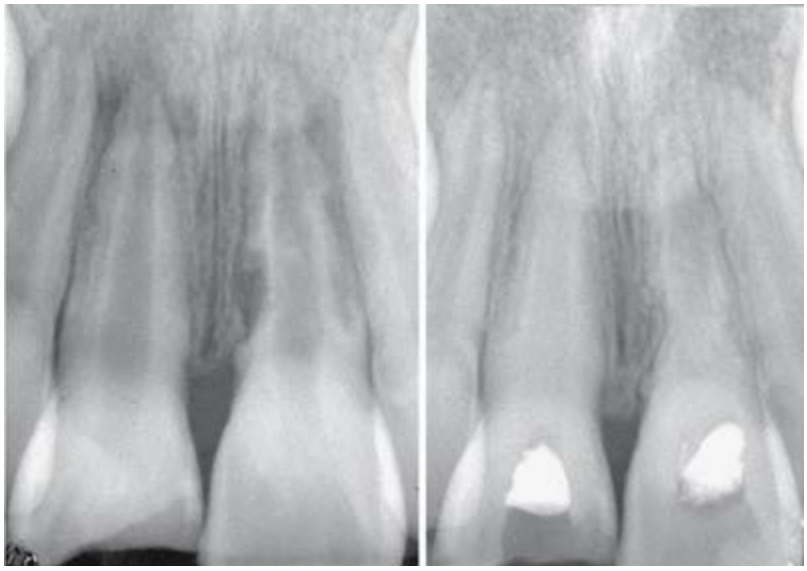

FIG. 21.37 Healing of external inflammatory root resorption after calcium hydroxide treatment. The radiolucencies seen before treatment have disappeared with the reestablishment of the lamina dura.

Radiograph on the left shows one tooth with chipped crown and wedge-shaped radiolucency in root canal. The adjacent tooth has radiolucent root canal with dark patches on both side and receding gum line.

Radiograph on the right shows crowns of two teeth with white depositions in dentin.

Source: (Courtesy Dr. Fred Barnett.)

Internal root resorption

Internal root resorption is rare in permanent teeth. Internal resorption is characterized by an oval-shaped enlargement of the root canal space.[21] External resorption, which is much more common, is often misdiagnosed as internal resorption.

Etiology.

Internal root resorption is characterized by resorption of the internal aspect of

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the root by multinucleated giant cells adjacent to the granulation tissue in the pulp (Fig. 21.38). Chronic inflammatory tissue is common in the pulp, but only rarely does it result in resorption. There are different theories on the origin of the pulpal granulation tissue involved in internal resorption. The most logical explanation is that it is inflamed pulp tissue caused by an infected coronal pulp space. Communication between the coronal necrotic tissue and the vital pulp is through appropriately oriented dentinal tubules (see Fig. 21.38).[191] One investigator[191] reported that resorption of the dentin is frequently associated with deposition of hard tissue resembling bone or cementum and not dentin. He postulates that the resorbing tissue is not of pulpal origin but is “metaplastic” tissue derived from the pulpal invasion of macrophage-like cells. Others[171] concluded that the pulp tissue was replaced by periodontium-like connective tissue when internal resorption was present. In addition to the requirement of the presence of granulation tissue, root resorption takes place only if the odontoblastic layer and predentin are lost or altered.[181]

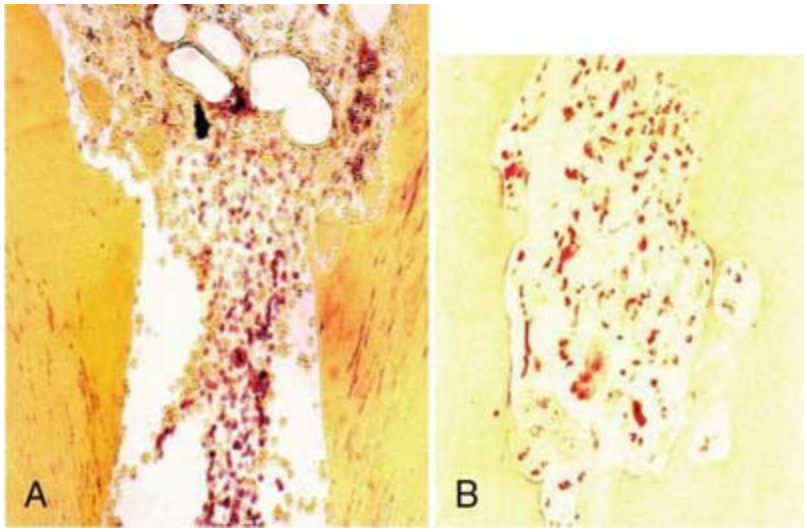

FIG. 21.38 Histologic appearance of internal root resorption. **A,** Section stained with Brown and Brenn. Bacteria are seen (in the dentinal tubules) communicating between the necrotic coronal segment and the apical granulomatous tissue and resorbing cells. **B,** An area of active internal root resorption.

A) Stained micrograph shows nearly oval and circular structures in the dentine with granular cells in the surrounding.

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B) Stained micrograph shows irregular light patch with scattered dark cells.

Source: (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

Reasons for the loss of predentin adjacent to the granulation tissue are not obvious. Trauma frequently has been suggested as a cause.[157,192] Another reason for the loss of predentin might be extreme heat produced when cutting on dentin without an adequate water spray. The heat presumably would destroy the predentin layer, and if later the coronal aspect of the pulp becomes infected, the bacterial products could initiate the typical inflammation in conjunction with resorbing giant cells in the vital pulp adjacent to the denuded root surface. Internal root resorption has been produced experimentally by the application of diathermy.[191]

Clinical manifestations.

Internal root resorption is usually asymptomatic and is first recognized clinically through routine radiographs. For internal resorption to be active, at least part of the pulp must be vital. The coronal portion of the pulp is often necrotic, whereas the apical pulp that includes the internal resorptive defect can remain vital. Therefore a negative sensitivity test result does not rule out active internal resorption. It is also possible that the pulp becomes nonvital after a period of active resorption, giving a negative sensitivity test, radiographic signs of internal resorption, and radiographic signs of apical inflammation. Traditionally, the pink tooth has been thought to be pathognomonic of internal root resorption. The pink color is due to the granulation tissue in the coronal dentin undermining the crown enamel. The pink tooth can also be a feature of invasive cervical resorption, which must be excluded before a diagnosis of internal root resorption is made. Invasive cervical resorption has been described and classified by Heithersay[92,93,94] by the extent of the resorptive lesion into the root, which initiates at the external cervical aspect of the tooth. Simple elimination of the resorptive tissue is ineffective in arresting its progress. Total removal or inactivation of the resorptive tissue is essential if recurrence (or concurrence) is to be avoided. Concurrence indicates the incomplete removal of the resorptive tissue at the time of treatment and recurrence is the reestablishment of the resorptive process. The reason for recurrence or concurrence is probably due to the

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invasive nature of the resorptive tissue whereby small infiltrative channels are created within the dentin, and these may interconnect with the periodontal ligament in positions more apical to the main resorptive defect. The use of trichloroacetic acid has been shown to be effective.[94]

Radiographic appearance.

The usual radiographic presentation of internal root resorption is a fairly uniform radiolucent enlargement of the pulp canal (Figs. 21.39 and 21.40). Because the resorption is initiated in the root canal, the resorptive defect includes some part of the root canal space, so the original outline of the root canal is distorted.

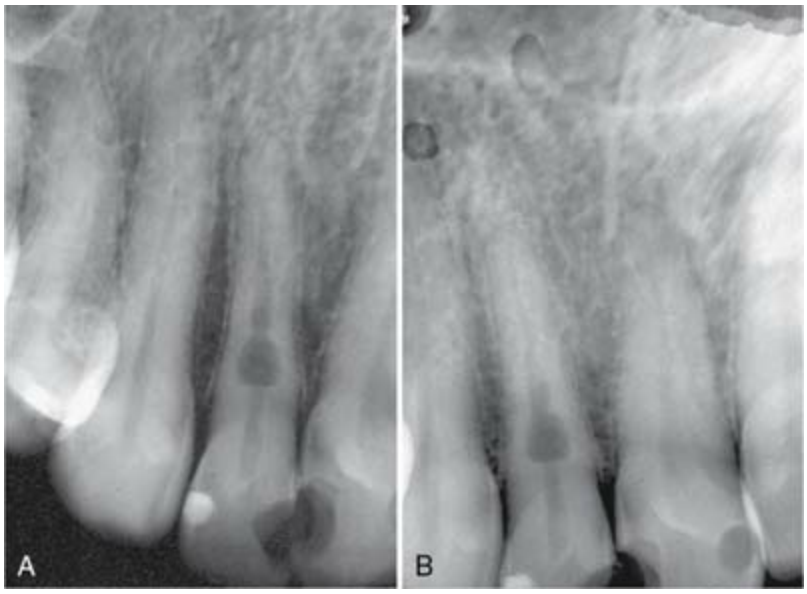

FIG. 21.39 Angled radiographs to show internal resorption. Radiographs from two different horizontal projections depict **(A)** the lesion within the confines of the canal on both views and **(B)** the adjacent bone intact on both views.

Radiograph A shows four teeth. The third tooth has a dark patch on the root canal. The crowns of both third and fourth teeth are broken at the connected site.

Radiograph B shows the third teeth with squarish dark patch below the neck region on the root canal. The fourth tooth has a round dark patch in the crown.

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Histologic appearance.

Like that of other inflammatory resorptive defects, the histologic picture of internal resorption is granulation tissue with multinucleated giant cells (see Fig. 21.37). An area of necrotic pulp is found coronal to the granulation tissue. Dentinal tubules containing microorganisms and communicating between the necrotic zone and the granulation tissue can sometimes be seen (see Fig. 21.38).[162,181,188,191] Unlike external root resorption, the adjacent bone is not affected with internal root resorption.

Treatment.

Treatment of internal root resorption is conceptually very easy. Because the resorptive defect is the result of the inflamed pulp and the blood supply to the tissue is through the apical foramina, endodontic treatment that effectively removes the blood supply to the resorbing cells is the treatment approach. After adequate anesthesia has been obtained, the canal apical to the internal defect is explored, and a working length short of the radiographic apex is used. The apical canal is thoroughly cleaned and shaped to ensure that the blood supply to the tissue resorbing the root is cut off.

By completion of the root canal instrumentation, it should be possible to obtain a blood-free and dry canal with paper points. Calcium hydroxide is spun into the canal to facilitate the removal of the tissue in the irregular defect at the next visit. At the second visit, the tooth and defect are filled with a warm gutta-percha technique (Fig. 21.41).

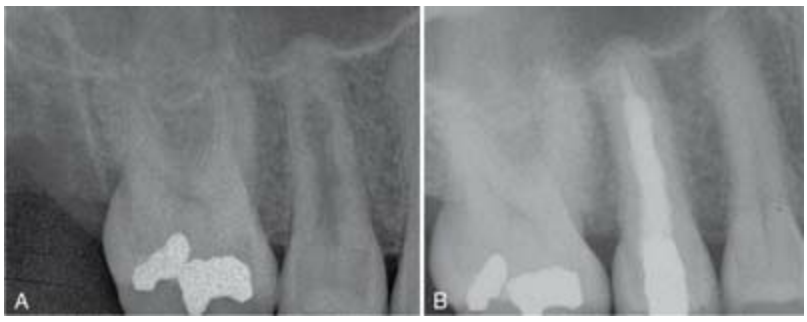

FIG. 21.41 **A,** Internal root resorption in a maxillary premolar with a history of trauma 7 years before the diagnosis (patient's head slammed against side window during an automobile accident). **B,** Three-year follow-up radiograph after endodontic treatment.

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Radiograph A shows upper premolar with radiolucent patch in-between the root and two radiopaque patches on the crown. The adjacent tooth has radiolucent root canal.

Radiograph B shows upper premolar with two radiopaque patches on the crown. The adjacent tooth has filled root canal.

Diagnostic features of external versus internal root resorption

It is often very difficult to distinguish external from internal root resorption, so misdiagnosis and incorrect treatment may result. The following sections present a list of typical diagnostic features of each resorptive type.

Radiographic features.

A change of angulation of x-rays should give a fairly good indication of whether a resorptive defect is internal or external. A lesion of internal origin appears close to the canal, whatever the angle of the x-ray (see Fig. 21.39). A defect on the external aspect of the root moves away from the canal as the angulation changes (Fig. 21.42). By using the buccal object rule, it is usually possible to distinguish whether the external root defect is buccal or lingual-palatal.

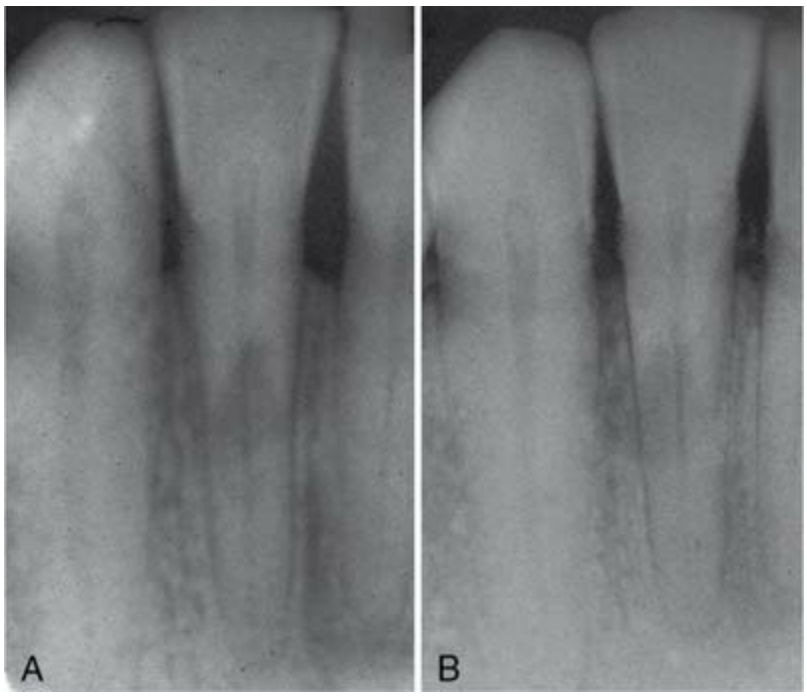

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FIG. 21.42 External root resorption. Radiographs from two different horizontal projections depict movement of the lesion to outside the confines of the root canal.

Radiograph A shows three teeth. The central tooth has a radiolucent patch below the neck region in the root. Radiograph B shows the central tooth with a radiolucent patch on one side of the canal.

With internal resorption, the outline of the root canal is usually distorted, and the root canal and radiolucent resorptive defect appear contiguous (see Fig. 21.40). When the defect is external, the root canal outline appears normal and can usually be seen "running through" the radiolucent defect (Fig. 21.43).

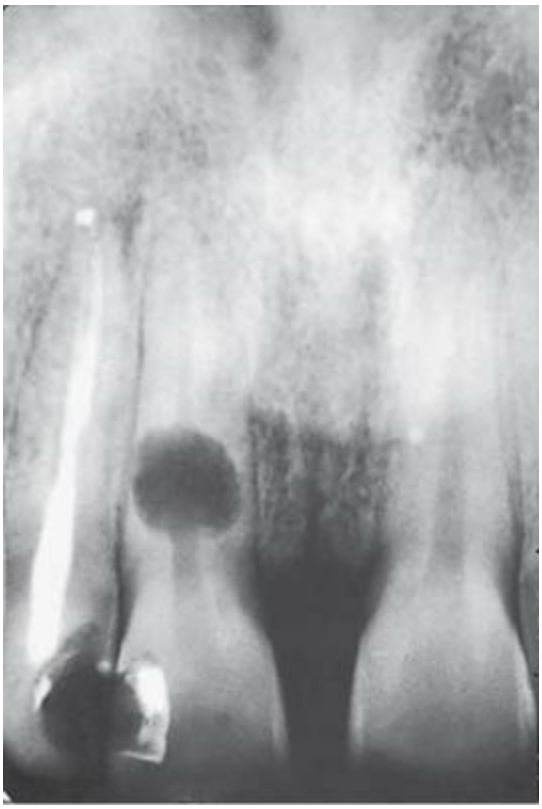

FIG. 21.40 A maxillary incisor with internal root resorption. Uniform enlargement of the pulp space is apparent. Outline of the canal cannot be seen in the resorptive defect.

Radiograph shows a round dark patch on the lateral side of crown in first tooth and another round patch in the root canal of second tooth.

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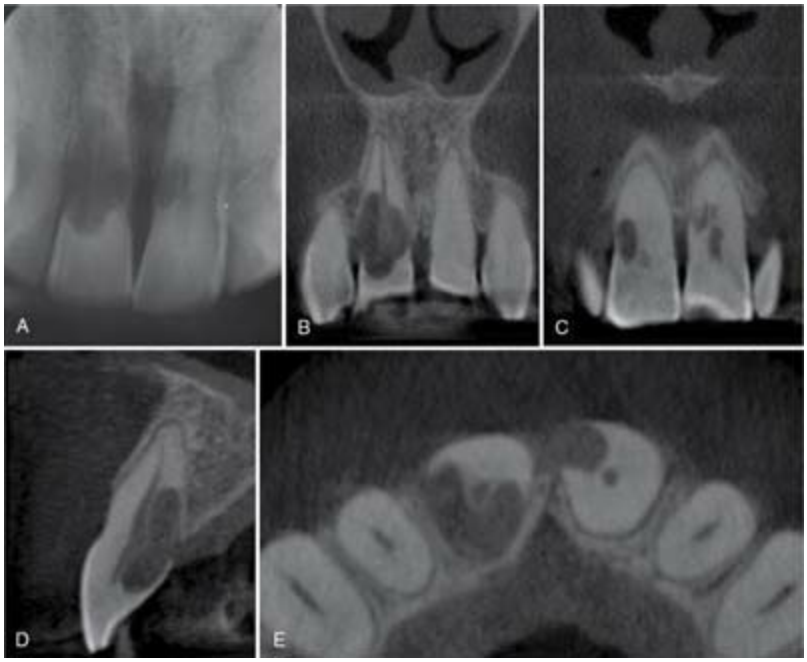

FIG. 21.43 **A,** Periapical radiograph of invasive cervical root resorption in tooth #8 and #9 in a 68-year-old female where the only known trauma was a fall onto bitumen aged 7 years when hit by a car. These teeth are diagnosed as having Heithersay Grade IV invasive cervical resorptive lesions which can be confirmed by CBCT imaging. **B** and **C,** CBCT coronal images. **D,** CBCT sagittal image of tooth #8. **E,** CBCT axial image. The outline of the canal is evident in Panels A, B, D, and E. The pulp does not appear exposed due to the anticlastic properties of predentin.

Five radiographs are marked A through E.

A) 8th tooth has a large oval-shaped dark patch. 9th tooth has a small dark patch on one side of the root canal.

B) 8th tooth has clear dark patch extended from dentin to the middle of root. 9th tooth is radiopaque.

C) 8th and 9th tooth show a dark patch on the right and at the center, respectively.

D) 8th tooth has a radiolucent root canal along with a large patch on the left.

E) 8th tooth has a U-shaped radiolucency. 9th tooth has a squarish radioluceny.

Invasive cervical resorption maybe accompanied by resorption of the bone in addition to the root (Fig. 21.44); radiolucencies can be apparent in the root and the adjacent bone. Internal root resorption does not involve the bone, and

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as a rule the radiolucency is confined to the root (see Fig. 21.39). On rare occasions, if the internal defect perforates the root, the bone adjacent to it is resorbed and appears radiolucent on the radiograph.

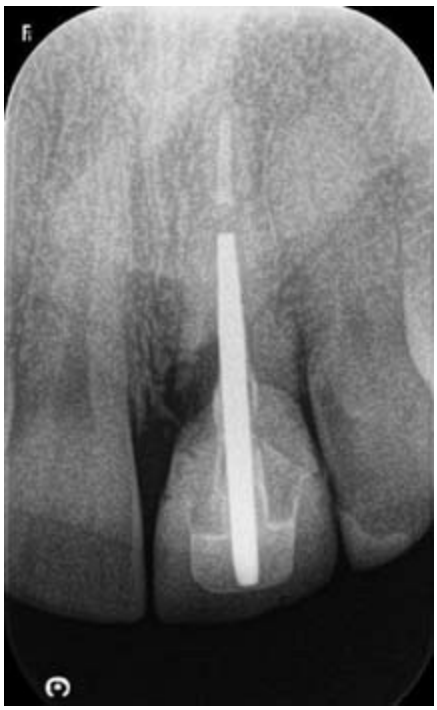

FIG. 21.44 Tooth #9 exhibiting invasive cervical resorption in a 31-year-old male. The tooth was injured 3 years prior after a fall from a scooter and was root filled and restored with a post crown. The resorption commences in the cervical external root and extends deeper into the root consistent with a Heithersay Grade III invasive cervical resorptive lesion. A resorptive defect is also present in the adjacent bone.

Radiograph shows central tooth with filled root canal. There is a radiolucent patch near the gum line.

Vitality testing.

External inflammatory resorption in the apical and lateral aspects of the root involves an infected pulp space, so no response to sensitivity tests supports the diagnosis. However, because invasive cervical resorption does not involve the pulp, a normal response to sensitivity testing is usually associated with this type of resorption. Internal root resorption usually occurs in teeth

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with vital pulps and responses to sensitivity testing. In teeth that exhibit internal root resorption, it is common to register a nonresponse to sensitivity testing because often the coronal pulp has been removed or is necrotic, and the active resorbing cells are more apical in the canal. In addition, the pulp might have become necrotic after active resorption took place.

Pink spot.

With apical and lateral external root resorption, the pulp is nonvital, so the granulation tissue that produces the pink spot is not present in these cases. For invasive cervical (Fig. 21.45) and internal root resorption, the pink spot due to the granulation tissue undermining the enamel is a possible sign.

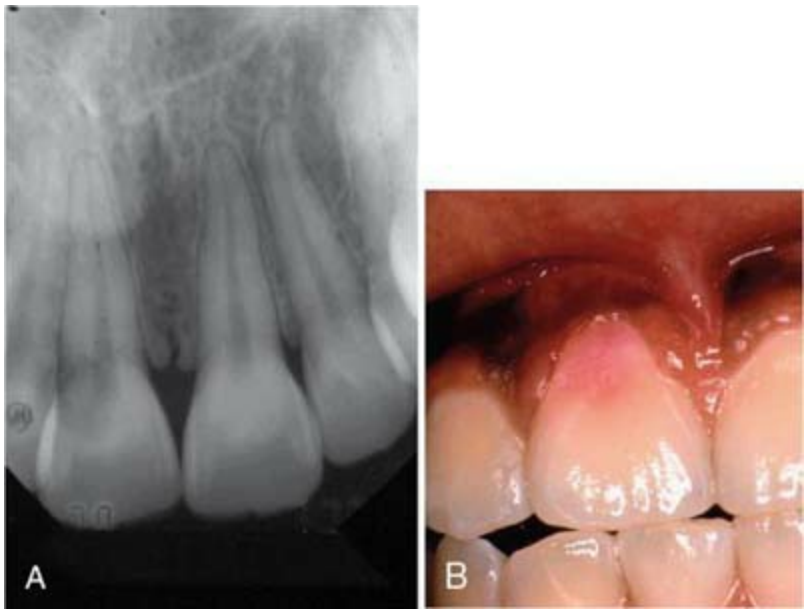

FIG. 21.45 Pink spot of invasive cervical root resorption. **A,** Radiographic appearance. **B,** Clinical appearance.

A) Radiograph shows a round radiolucent patch on the crown close to the gum line.

B) Close-up view shows a pink stain on the tooth.

Transient apical breakdown.

A variation on internal resorption, noted especially after luxation injuries to more mature teeth, is transient apical internal resorption. Disruption of the

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neurovascular bundle at the apex may result in inflammatory mediators that allow osteoclastic changes at the apex to accommodate vascular derived repair followed by remodeling of the apex when the resorptive processes resolve. Teeth considered to show a combination of color changes, altered or V-notched apical radiographic changes, and altered pulp sensibility over time may have “transient apical breakdown” (TAB) for which no treatment other than observation is necessary.[9]

Summary of possible diagnostic features

- External inflammatory root resorption due to pulp infection.
  - *Apical:* No response of the pulp to thermal or electric stimuli, with or without a history of trauma.
  - *Lateral:* History of trauma, no response of the pulp to thermal or electric stimuli, lesion moves on angled x-rays, root canal visualized radiographically overlying the defect, bony radiolucency also apparent.
- Invasive cervical resorption. Often a history of trauma (maybe forgotten, or its long-term risks not appreciated by the patient); positive pulp sensitivity test; lesion located at the attachment level of the tooth; lesion moves on angled x-rays; root canal outline is undistorted and can be visualized radiographically; crestal bony defect associated with the lesion; pink spot possible. CBCT imaging more reliably illustrates the extent of the resorption throughout the root (see Fig. 21.43).
- Internal root resorption. History of trauma, crown preparation, or pulpotomy; responsive pulp to thermal or electric stimuli likely, may occur at any location along the root canal (not only attachment level); lesion stays associated with the root canal on angled x-rays, radiolucency contained in the root without an adjacent bony defect; pink spot possible.

Most misdiagnoses of resorptive defects are made between invasive cervical and internal root resorptions. The diagnosis should always be confirmed as treatment proceeds. If root canal therapy is the treatment of choice for an apparent internal root resorption, the bleeding within the canal

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should cease quickly after pulp extirpation because the blood supply of the granulation tissue is the apical blood vessels. If bleeding continues during treatment, and particularly if it is still present at the second visit, the source of the blood supply is external, and treatment for perforating external resorption should be carried out. On obturation, it should be possible to fill the entire canal from within in internal resorption. Failure to achieve this should make the clinician suspicious of an external lesion that perforates the root. Finally, if the blood supply of an internal resorption defect is removed on pulp extirpation, any continuation of the resorptive process on recall radiographs should alert the clinician to the possibility that an external resorptive defect was misdiagnosed.

Clinical management of the avulsed tooth

Favorable healing after an avulsion injury requires quick emergency intervention followed by evaluation and possible treatment at decisive times during the healing phase. The urgency of the emergency visit and the multidisciplinary nature of follow-up evaluations require that both the public and clinicians from many dental disciplines be knowledgeable about the treatment strategies involved.

Consequences of tooth avulsion

Tooth avulsion results in attachment damage and pulp necrosis. The tooth is "separated" from the socket due mainly to tearing of the periodontal ligament that leaves viable periodontal ligament cells on most of the root surface. In addition, small and localized cemental damage occurs from the crushing of the tooth against the socket.

If the periodontal ligament left attached to the root surface does not dry out, the consequences of tooth avulsion are usually minimal.[19,168] The hydrated periodontal ligament cells will maintain their viability and repair after replantation, with minimal destructive inflammation as a by-product. Because the areas of the crushing injury are localized, inflammation stimulated by the damaged tissues will be correspondingly limited, and favorable healing with new replacement cementum is likely to occur after the initial inflammation subsides (see Fig. 21.29).

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If excessive drying occurs before replantation, the damaged periodontal ligament cells elicit a severe inflammatory response over a diffuse area on the root surface. Unlike the situation described earlier, in which the area to be repaired after the initial inflammatory response is small, here a large area of root surface is affected that must be repaired by new tissue. The slower-moving cementoblasts cannot cover the entire root surface in time, and it is likely that in certain areas, bone will attach directly onto the root surface. In time, through physiologic bone recontouring, the entire root will be replaced by bone. As earlier noted, this has been termed *replacement resorption* (see Figs. 21.30 and 21.31).[25,184]

Pulpal necrosis always occurs after an avulsion injury. Although a necrotic pulp itself is not of consequence, the necrotic tissue is extremely susceptible to bacterial contamination. If revascularization does not occur or effective endodontic therapy is not carried out, the pulp space inevitably becomes infected. The combination of bacteria in the root canal and cemental damage on the external surface of the root results in an external inflammatory resorption that can be very serious and can lead to rapid loss of the tooth root (see Figs. 21.32 and 21.33).[181]

The consequences after tooth avulsion appear to be directly related to the severity and surface area of the inflammation on the root surface and resultant damaged root surface that must be repaired. Treatment strategies should always be considered in the context of limiting the extent of the periradicular inflammation, thus tipping the balance toward favorable responses (cemental) rather than unfavorable ones (replacement resorption or inflammatory resorption).

Treatment objectives

Treatment is directed at avoiding or minimizing resultant inflammation due to the two main consequences of the avulsed tooth: attachment damage and pulpal infection.

Attachment damage as a direct result of the avulsion injury cannot be avoided. However, considerable additional damage can occur to the periodontal ligament in the time that the tooth is out of the mouth (primarily because of drying). Treatment is directed at minimizing this damage (and the resultant inflammation) so that the fewest possible complications result.

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When severe additional damage cannot be avoided and replacement resorption of the root is considered certain, steps are taken to slow the replacement of the root by bone to maintain the tooth in the mouth for as long as possible.

In the open apex tooth, all efforts are made to promote revascularization of the pulp, thus avoiding pulp space infection. When revascularization fails (in the open apex tooth) or is not possible (in the closed apex tooth), all treatment efforts are made to prevent or eliminate toxins from the root canal space.

Clinical management

Emergency treatment at the accident site

Replant if possible, or place in an appropriate storage medium. As mentioned, damage to the attachment apparatus that occurred during the initial injury is unavoidable but usually minimal. However, all efforts must be made to minimize necrosis of the remaining periodontal ligament while the tooth is out of the mouth. Pulpal sequelae are not a concern initially and are dealt with at a later stage of treatment.

The single most important factor to ensure a favorable outcome after replantation is the speed with which the tooth is replanted.[21,27] Of utmost importance is the prevention of drying, which causes loss of normal physiologic metabolism and morphology of the periodontal ligament cells.[27,168] Every effort should be made to replant the tooth within the first 15 to 20 minutes.[32] This usually requires emergency personnel at the site of the injury with some knowledge of treatment protocol. The clinician should communicate clearly with the person at the site of the accident. Ideally this information should have been given at an earlier time; for example, as an educational offering to school nurses or athletic trainers. Failing this, the information can be given over the phone. The aim is to replant a clean tooth with an undamaged root surface as gently as possible, after which the patient should be brought to the office immediately. If doubt exists that the tooth can be replanted adequately, the tooth should quickly be stored in an appropriate medium until the patient can get to the dental office for replantation. Suggested storage media, in order of preference, are milk, saliva (either in the vestibule of the mouth or in a container into which the patient expectorates),

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physiologic saline, and water.[100] Water is the least desirable storage medium because the hypotonic environment causes rapid cell lysis and increased inflammation on replantation.[42,43]

Cell culture media in specialized transport containers, such as Hanks Balanced Salt Solution (HBSS), have shown superior ability to maintain the viability of the periodontal ligament fibers for extended periods.[186] Presently they are considered impractical because they need to be present at the accident site before the injury occurs. However, if we consider that more than 60% of avulsion injuries occur close to home or school,[89] it seems reasonable to assume that it would be beneficial to have these media available in emergency kits at these sites. It would also be advantageous to have them in ambulances and in the kits of emergency response personnel who are likely to treat the more serious injuries in which teeth might otherwise be sacrificed to a more serious life-threatening situation.

Management in the dental office

Emergency visit.

Prepare socket, prepare root, replant, construct a functional splint, and administer local and systemic antibiotics.

Recognizing that a dental injury might be secondary to a more serious injury is essential. The attending dental clinician is likely to be the first health care provider the patient sees after a head injury, so ruling out any injuries to the brain (e.g., concussion) and/or central nervous system (CNS) in general is paramount. If on examination a CNS injury is suspected, immediate referral to the appropriate expert is the first priority, above and beyond the dental injury. Once a CNS injury has been ruled out, the focus of the emergency visit is the attachment apparatus. The aim is to replant the tooth with a minimum of irreversibly damaged cells (that will cause inflammation) and the maximal number of periodontal ligament cells that have the potential to regenerate and repair the damaged root surface.

Diagnosis and treatment planning

If the tooth was replanted at the site of injury, a complete history is taken to assess the likelihood of a favorable outcome. The position of the replanted

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tooth is assessed and adjusted if necessary. On rare occasions, the tooth may be gently removed to prepare the root to increase the chances of a favorable outcome (discussed later).

If the patient presents with the tooth out of the mouth, the storage medium should be evaluated and the tooth placed in a more appropriate medium if required. HBSS is presently considered the best medium for this purpose. Milk or physiologic saline is also appropriate for storage purposes.

The medical and accident histories are taken, and a clinical exam is carried out, with emphasis on questions about when, how, and where the injury occurred.

The clinical examination should include an examination of the socket to ascertain whether it is intact and suitable for replantation. The socket is gently rinsed with saline, and when it has been cleared of the clot and debris, its walls are examined directly for the presence, absence, or collapse of the socket wall. The socket and surrounding areas, including the soft tissues, should be radiographed. Three vertical angulations are required for diagnosis of the presence of a horizontal root fracture in adjacent teeth.[21] The remaining teeth in both the upper and lower jaws should be examined for injuries, such as crown fractures. Any soft-tissue lacerations should be noted and, if tooth fragments are missing, explored. A photograph prior to any treatment is beneficial especially if legal concerns or insurance claims are involved.[13]

Preparation of the root

Preparation of the root depends on the maturity of the tooth (open versus closed apex) and on the dry time of the tooth before it was placed in a storage medium. A dry time of 60 minutes is considered the point where survival of root periodontal ligament cells is unlikely.

Extraoral dry time less than 60 minutes

Closed apex

The root should be rinsed of debris with water or saline and replanted in as gentle a fashion as possible.

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If the tooth has a closed apex, revascularization is not possible,[59] but because the tooth was dry for less than 60 minutes (replanted or placed in appropriate medium), the chance for periodontal healing exists. Most importantly, the chance of a severe inflammatory response at the time of replantation is lessened. A dry time of less than 15 to 20 minutes is considered optimal, and periodontal healing would be expected.[19,32,168]

A continuing challenge is the treatment of the tooth that has been dry for more than 20 minutes (periodontal cell survival is assured) but less than 60 minutes (periodontal cell survival unlikely). In these cases, logic suggests that the root surface consists of some cells with the potential to regenerate and some that will act as inflammatory stimulators.

Open apex

Gently rinse off debris, if available, soak in doxycycline for 5 minutes or cover with minocycline, and replant.

In an open apex tooth, revascularization of the pulp and continued root development are possible (see Fig. 21.35). Investigators[59] found in monkeys that soaking the tooth in doxycycline (1 mg in approximately 20 mL of physiologic saline) for 5 minutes before replantation significantly enhanced complete revascularization (Fig. 21.46, *A*). This result was confirmed later in dogs by other investigators.[150,197] A study found that covering the root with minocycline (Arestin, OraPharma, Warminster, PA), which attaches to the root for approximately 15 days, further increased the revascularization rate in dogs (see Fig. 21.46, *B*).[150] Although animal studies do not provide us with a prediction of the rate of revascularization in humans, it is reasonable to expect that the same enhancement of revascularization that occurred in two animal species also will occur in humans.[31] As for a closed apex tooth, the open apex tooth is gently rinsed and replanted.

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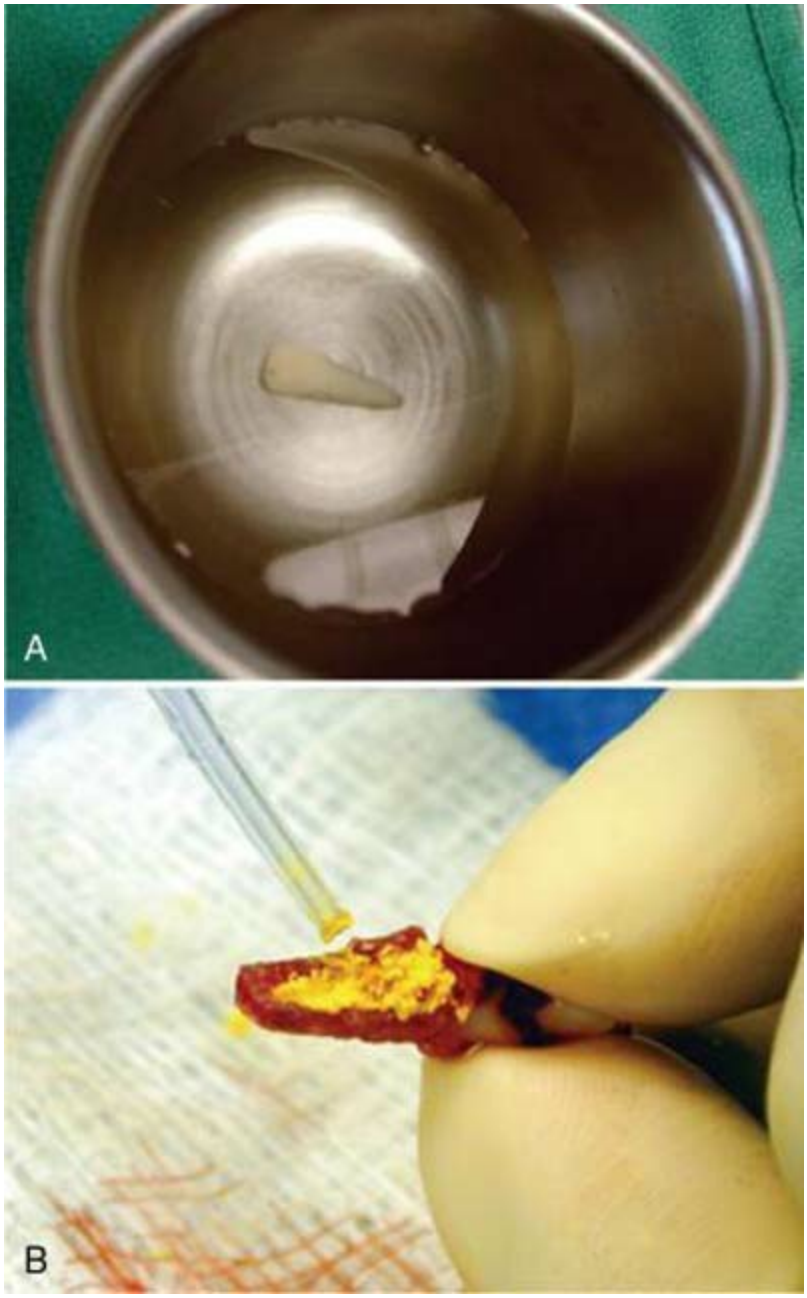

FIG. 21.46 **A,** Avulsed tooth soaking in doxycycline. **B,** Minocycline powder placed on the root surface before replantation.

A) Tooth is dipped in a steel bowl one-fifth filled with water.

B) Close-up view shows a tooth model held in gloved hands being filled by yellow powder by using glass rod.

Extraoral dry time more than 60 minutes

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Closed apex

Remove the periodontal ligament by placing in acid for 5 minutes, soak in fluoride, and replant.

When the root has been dry for 60 minutes or more, survival of the periodontal ligament cells is not expected.[27,168] In such cases the root should be prepared to be as resistant to resorption as possible (attempting to slow the osseous replacement process). These teeth should be soaked in acid for 5 minutes to remove all remaining periodontal ligament and thus remove the tissue that would initiate the inflammatory response on replantation. The tooth can then be soaked in 2% stannous fluoride for 5 minutes and replanted.[41,159] A few years ago, studies were published that indicated that an enamel matrix protein, Emdogain (Straumann USA, Andover, MA), could be beneficial in teeth with extended extraoral dry times, not only to make the root more resistant to resorption but also, possibly, to stimulate the formation of new periodontal ligament from the socket.[74,104] Unfortunately, more recent studies have shown that the positive effect of Emdogain is only temporary, and most of these teeth start to resorb after a few years.[154]

If the tooth has been dry for more than 60 minutes and no consideration is given to preserving the periodontal ligament, the endodontics may be performed extraorally. In the case of a tooth with a closed apex, no advantage exists to this additional step at the emergency visit. However, in a tooth with an open apex, endodontic treatment performed after replantation involves a long-term apexification procedure. In these cases, completing the root canal treatment extraorally, in which a seal in the blunderbuss apex is easier to achieve, may be advantageous. When endodontic treatment is performed extraorally, it must be performed aseptically with the utmost care to achieve a root canal system that is thoroughly disinfected.

Open apex

Replant? If yes, treat as with closed apex tooth. Endodontic treatment may be performed out of the mouth.

Because these teeth are in young patients in whom facial development is usually incomplete, many pediatric clinicians consider the prognosis to be so poor and the potential complications of an ankylosed tooth so severe, they recommend that these teeth not be replanted. Considerable debate exists as to

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whether it would be beneficial to replant the root even though it will inevitably be lost due to osseous replacement. If the patients are followed carefully and the root submerged by decoronation procedure at the appropriate time,[8,73,130] the height, and, more important, the width of the alveolar bone will be maintained, allowing for easier permanent restoration at the appropriate time when the child's facial development is complete.

Preparation of the socket

The socket should be left undisturbed before replantation.[21] Emphasis is placed on removal of obstacles in the socket to facilitate replacement of the tooth into the socket.[87] It should be lightly aspirated if a blood clot is present. If the alveolar bone has collapsed or may interfere with replantation, a blunt instrument should be inserted carefully into the socket in an attempt to reposition the wall.

Splinting

A splint that allows for physiologic movement of the tooth during healing and that is in place for a minimal period results in a decreased incidence of ankylosis.[4,17,87] Flexible (physiologic) fixation for 1 to 2 weeks is recommended.[4,6,7] Composite and wire splints where the wire diameter is no greater than 0.4 mm is considered to be a flexible splint.[124] The splint should allow movement of the tooth, should have no memory (so the tooth is not moved during healing), and should not impinge on the gingiva and/or prevent maintenance of oral hygiene in the area. Many splints satisfy the requirements of an acceptable device.[111] Fishing line has recently been recommended as a splinting material and, when bonded with resin modified glass ionomer cement, reduces the amount of iatrogenic damage to enamel when the splint is removed.[75a,111] A new titanium trauma splint (TTS) has recently been shown to be particularly effective and easy to use (Fig. 21.47).[190] After the splint is in place, a radiograph should be exposed to verify the positioning of the tooth and as a preoperative reference for further treatment and follow-up. When the tooth is in the best possible position, adjusting the bite to ensure that it has not been splinted in a position causing

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traumatic occlusion is important. One week is sufficient to create periodontal support to maintain the avulsed tooth in position.[185] Therefore the splint should be removed after 1 to 2 weeks. The only exception is with avulsion in conjunction with alveolar fractures, for which 4 to 8 weeks is the suggested time of splinting.[185]

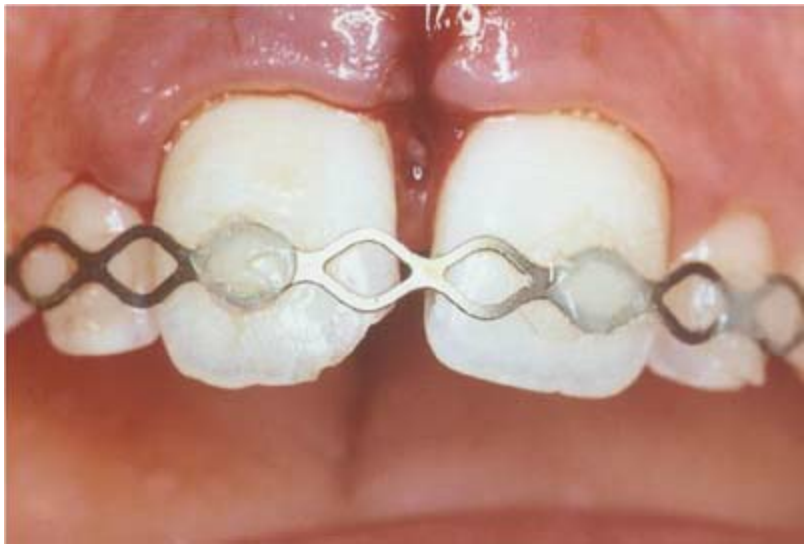

FIG. 21.47 Titanium trauma splint (TTS) in place.

Close-up view of upper jaw shows TTS holding the teeth. The central incisors have a cavity filled with fluid in the gum line.

Management of the soft tissues

Soft-tissue lacerations of the socket gingiva should be tightly sutured. Lacerations of the lip are fairly common with these types of injuries. The clinician should approach lip lacerations with some caution; a plastic surgery consult might be prudent. If these lacerations are sutured, care must be taken to clean the wound thoroughly beforehand because dirt or even minute tooth fragments left in the wound affect healing and the esthetic result.

Adjunctive therapy

Systemic antibiotics given at the time of replantation and before endodontic

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treatment are effective in preventing bacterial invasion of the necrotic pulp and therefore subsequent inflammatory resorption.[87] Tetracycline has the additional benefit of decreasing root resorption by affecting the motility of the osteoclasts and reducing the effectiveness of collagenase.[151] The administration of systemic antibiotics is recommended, beginning at the emergency visit and continuing until the splint is removed.[87] For patients not susceptible to tetracycline staining, the antibiotic of choice is doxycycline twice daily for 7 days at the appropriate dosage for patient age and weight.[151,152] Penicillin V 1000 mg as a loading dose, followed by 500 mg 4 times daily for 7 days, has also been shown to be beneficial. The bacterial content of the sulcus also should be controlled during the healing phase. In addition to stressing to the patient the need for adequate oral hygiene, the use of chlorhexidine rinses for 7 to 10 days is helpful.

As stated previously, there is a benefit in removal of the pulp contents at the emergency visit and placing Ledermix or corticosteroid into the root canal.[45,49] The medicament was able to shut down the inflammatory response after replantation and inhibit the spread of dentinoclasts to allow for more favorable healing compared with those teeth that did not have the medicament.[144,145]

The need for analgesics should be assessed on an individual case basis. The use of pain medication stronger than nonprescription nonsteroidal antiinflammatory drugs (NSAIDs) is unusual. The patient should be sent to a physician for consultation regarding a tetanus booster within 48 hours of the initial visit.

Second visit

The second visit should take place 1 to 2 weeks after the trauma. At the emergency visit, emphasis was placed on the preservation and healing of the attachment apparatus. The focus of the second visit is the prevention or elimination of potential irritants from the root canal space. These irritants, if present, provide the stimulus for the progression of the inflammatory response, bone and root resorption. Also at this visit, the course of systemic antibiotics is completed; the chlorhexidine rinses can be stopped. At this appointment the splint is removed; the tooth might still have class I or class II mobility after splint removal, but all indications are that it will continue to

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heal better without the splint.[4]

Endodontic treatment

Extraoral time less than 60 minutes

Closed apex

Initiate endodontic treatment after 1 to 2 weeks. When endodontic treatment is delayed or signs of resorption are present, provide long-term calcium hydroxide treatment before obturation.

No chance exists for revascularization of teeth with closed apices; therefore endodontic treatment should be initiated at the second visit 7 to 10 days later.[18,59] If therapy is initiated at this optimum time, the pulp should be necrotic without (or with minimal) infection.[129,187] Endodontic therapy with an effective interappointment antibacterial agent[187] over a relatively short period (1 to 2 weeks) is sufficient to ensure effective disinfection of the canal.[163] Long-term calcium hydroxide treatment should always be considered if the injury occurred more than 2 weeks before initiation of the endodontic treatment or especially if radiographic evidence of inflammatory resorption is present.[187]

The root canal is thoroughly cleaned and shaped, irrigated, and then filled with calcium hydroxide. The canal is obturated when a radiographically intact periodontal membrane can be demonstrated around the root (see Fig. 21.37). Calcium hydroxide is an effective antibacterial agent[46,163] and favorably influences the local environment at the resorption site, theoretically promoting healing.[182] It also changes the environment in the dentin to a more alkaline pH, which may slow the action of the resorptive cells and promote hard-tissue formation.[182] However, changing of the calcium hydroxide should be kept to a minimum (not more than every 3 months) because it has a necrotizing effect on the cells attempting to repopulate the damaged root surface.[125]

Calcium hydroxide is considered the material of choice in the prevention and treatment of inflammatory root resorption, but it is not the only medicament recommended in these cases. Some attempts have been made not

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only to remove the stimulus for the resorbing cells but also to affect them directly. The antibiotic-corticosteroid paste Ledermix is effective in treating inflammatory root resorption by inhibiting the spread of dentinoclasts[144,145] without damaging the periodontal ligament; however, its ability to diffuse through the human tooth root has been demonstrated,[2] and its release and diffusion are enhanced when it is used in combination with calcium hydroxide paste.[3]

Open apex

Avoid endodontic treatment and look for signs of revascularization. At the first sign of an infected pulp, initiate apexification procedure.

Teeth with open apices have the potential to revascularize and continue root development; initial treatment is directed toward reestablishing the blood supply.[59,150,196] The initiation of endodontic treatment is avoided if at all possible, unless definite signs of pulp necrosis are present (e.g., periradicular inflammation). An accurate diagnosis of pulp vitality is extremely challenging in these cases. After trauma, a diagnosis of necrotic pulp is particularly undesirable because infection in these teeth is potentially more harmful due to cemental damage accompanying the traumatic injury. External inflammatory root resorption can be extremely rapid in these young teeth because the tubules are wide and allow irritants to move freely to the external surface of the root.[59,196]

Patients should be recalled every 3 to 4 weeks for pulp vitality testing. Studies indicate that thermal tests with carbon dioxide snow (−78°C) or dichlorodifluoromethane (−40°C) placed at the incisal edge or pulp horn are the best methods of sensitivity testing, particularly in young permanent teeth.[79,80,140] One of these two tests must be included in the pulp vitality testing. Reports confirm the superiority of the laser Doppler flowmeter in the diagnosis of revascularization of traumatized immature teeth[197]; however, the cost of such an instrument precludes its use in the average dental office. Radiographic signs (apical breakdown and/or signs of lateral root resorption) and clinical signs (pain on percussion and palpation) of pulp pathosis are carefully assessed. At the first sign of pathosis, endodontic treatment should be initiated, and after disinfection of the root canal space, an apexification procedure should be carried out.

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Extraoral time more than 60 minutes

Closed apex

Teeth with closed apices are treated endodontically in the same way as teeth that had an extraoral time of less than 60 minutes.

Open apex (if replanted)

If endodontic treatment was not performed out of the mouth, initiate apexification procedure.

The chance of revascularization in these teeth is extremely poor,[184,188] so no attempt is made to revitalize them. An apexification procedure is initiated at the second visit if root canal treatment was not performed at the emergency visit. If endodontic treatment was performed at the emergency visit, the second visit is a recall visit to assess initial healing only.

Temporary restoration

Effectively sealing the coronal access is essential to prevent infection of the canal between visits. Recommended temporary restorations are reinforced zinc oxide eugenol cement, acid etch composite resin, or glass ionomer cement. The depth of the temporary restoration is critical to its sealability. A depth of at least 4 mm is recommended, so a cotton pellet should not be placed; the temporary restoration is placed directly onto the calcium hydroxide in the access cavity. Calcium hydroxide should be removed from the walls of the access cavity before the temporary restoration is placed because calcium hydroxide is soluble and washes out when it comes into contact with saliva, leaving a defective temporary restoration.

After initiation of the root canal treatment, the splint is removed. If time does not permit complete removal of the splint at this visit, the resin tacks are smoothed so as not to irritate the soft tissues; the remaining resin is removed at a later appointment.

At this appointment, healing is usually sufficient to allow a detailed clinical examination of the teeth surrounding the avulsed tooth. Pulp vitality tests, reaction to percussion and palpation, and periodontal probing measurements should be carefully recorded for reference at follow-up visits.

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Root filling visit

This visit should take place at the clinician's convenience or after long-term calcium hydroxide therapy, when an intact lamina dura is traced.

If the endodontic treatment was initiated 1 to 2 weeks after the avulsion and a thorough examination confirms normality, filling of the root canal at this visit is acceptable. Long-term use of calcium hydroxide is also a proven option for these cases. If endodontic treatment was initiated more than 2 weeks after the avulsion or active resorption is visible, the pulp space must first be disinfected before root filling. Traditionally, the reestablishment of a lamina dura (see Fig. 21.37) is a radiographic sign that the canal bacteria have been controlled. When an intact lamina dura can be traced throughout, root filling can take place.

The canal is cleaned, shaped, and irrigated under strict asepsis (i.e., a rubber dam). After completion of cleaning and shaping, the canal can be filled.

Permanent restoration

Much evidence exists that coronal leakage caused by defective temporary and permanent restorations results in a clinically relevant amount of bacterial contamination of the root canal after root filling.[149] Therefore the tooth should receive a permanent restoration as soon as possible. The depth of restoration is important for a tight seal, so the deepest restoration possible should be made. A post should be avoided if possible. Because most avulsions occur in the anterior region of the mouth where esthetics is important, composite resins combined with dentin bonding agents are recommended in these cases.

Follow-up care

Follow-up evaluations should take place at 3 months, 6 months, and yearly for at least 5 years. If replacement resorption is identified (see Fig. 21.31), a more closely monitored follow-up schedule is indicated. In the case of inflammatory root resorption (see Figs. 21.32 and 21.33), a new attempt at disinfection of the root canal space by standard retreatment might reverse the process. Teeth adjacent to and surrounding the avulsed tooth or teeth may

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show pathologic changes long after the initial accident, so these teeth, too, should be tested at follow-up visits and the results compared with those collected soon after the accident.

Late complications

The IADT and AAE guidelines as stated previously recommend 5-year follow-up after the initial trauma. Heithersay[95,96] wrote a series on the "life cycles of traumatized teeth" discussing follow-up of cases of up to 51 years post injury. Resorption is a common posttraumatic sequelae presenting many years after this initial trauma. Changes in the color of the crown also need to be monitored as either a need for bleaching of the tooth or a change in the condition of the tooth such as intracanal calcification. This chapter has presented many concepts and treatment approaches at the time of trauma. However, it is important to consider that there can be either unfortunate events over time that need secondary management or failure in diagnosis that is apparent years later when signs and symptoms of infection or resorption and inappropriate or poor treatment were performed at the time of injury (Figs. 21.48 and 21.49; see also Fig. 21.43).

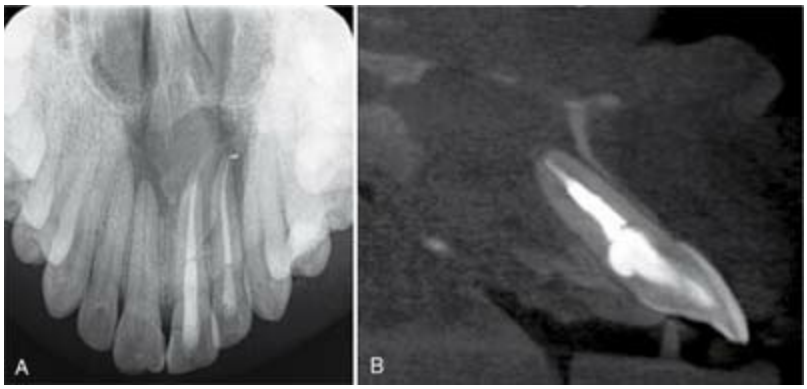

FIG. 21.48 **A,** An occlusograph showing root fillings in teeth #9 and #10 in a 44-year-old male 25 years after the teeth had been traumatized. Symptoms and infection returned after endodontic treatment and surgical correction was required. **B,** CBCT imaging revealed internal resorption within the canal.

A) Radiograph shows upper jaw with filled root canals with a radiolucent line at the center in left incisors. There is a large radiolucent patch at the root apices of incisors.

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B) Radiograph shows fading filling in the root apices and crown.

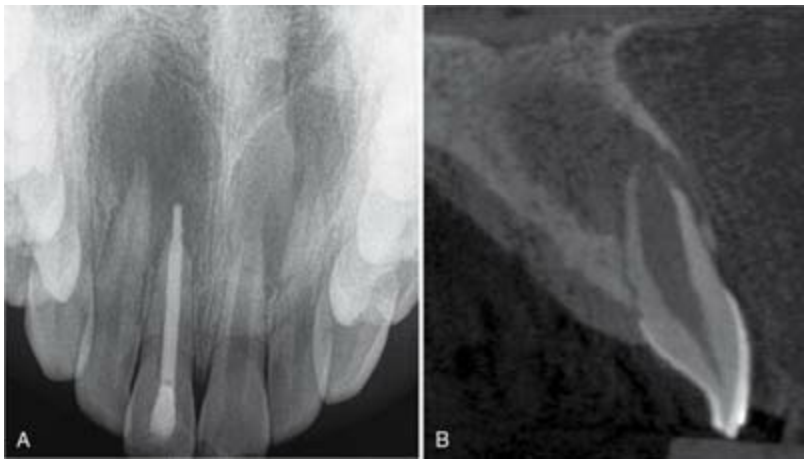

FIG. 21.49 **A,** An occlusograph showing extensive periapical radiolucencies associated with the maxillary incisor teeth in a 23-year-old male. The patient sustained a traumatic injury when he was 8 years of age. Tooth #8 has extruded gutta-percha through the open apex indicating either a failed apexification procedure or poor obturation of the canal due to the open apex. Tooth #9 has an open apex indication pulp necrosis likely occurred at or soon after the traumatic injury but no treatment was undertaken. **B,** CBCT imaging revealing the immature root formation of tooth #9 and periapical lesion. Surface resorption on the palatal cervical root is suggestive of a prior palatal luxation injury.

A) Radiograph shows upper jaw where eighth root has filled root canal and ninth tooth has open and wide radiolucent canal. Seventh and eighth teeth have a radiolucent patch.

B) Radiograph of eighth tooth shows radiolucent root canal, which narrows down toward the crown and widens toward the root tip.

Dentoalveolar trauma to the primary dentition

The classification of injuries to the primary dentition is the same as the permanent dentition, although management may be different. Consideration must be given to the risk of damage to the developing permanent teeth. Furthermore, there may also be behavioral issues of the child that require different approaches in patient care. Instances where care is required to minimize risk to the permanent dentition include intrusive injuries when the intruded root is close to the developing permanent tooth. It is generally not

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advisable to replant an avulsed primary tooth. However, most luxation injuries heal spontaneously, so conservative treatment for preschool children is recommended. Immediate treatment is advised for complicated crown fractures, crown-root fractures, root fractures when the coronal fragment is displaced, extrusive luxations, and alveolar fractures.[75,130]

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References

1. AAE clinical considerations for a regenerative procedure. https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/currentregenerativeendodo
2. Abbott PV, Heithersay GS, Hume WR. *Release and diffusion through human tooth roots in vitro of corticosteroid and tetracycline trace molecules from Ledermix paste*, Endod Dent Traumatol 1988;4: 55.
3. Abbott PV, Hume WR, Heithersay GS. *Effects of combining Ledermix and calcium hydroxide pastes on the diffusion of corticosteroid and tetracycline through human tooth roots in vitro*, Endod Dent Traumatol 1989;5: 188.
4. About I, Murray PE, Franquin JC, et al: *The effect of cavity restoration variables on odontoblast cell numbers and dental repair*, J Dent 2001;29: 109.
5. Accorinte L, Holland R, Reis A, et al: *Evaluation of mineral trioxide aggregate and calcium hydroxide cement as pulp-capping agents in human teeth*, J Endod 2008;34: 1.
6. Deleted in page review.
7. Andersson L, Friskopp J, Blomlof L. *Fiber-glass splinting of traumatized teeth*, ASDC J Dent Child 1983;50: 21.
8. Andersson L, Malmgren B. *The problem of dentoalveolar ankylosis and subsequent replacement resorption in the growing patient*, Aust Endod J 1999;25: 57.
9. Andreasen FM. *Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth*, Endod Dent Traumatol 1986;2: 9.
10. Andreasen FM. *Pulpal healing after luxation injuries and*

Telegram: @dental_k

www.konkur.in

root fracture in the permanent dentition, Endod Dent Traumatol 1989;5: 111.

11. Andreasen FM, Andreasen JO. *Resorption and mineralization processes following root fracture of permanent incisors*, Endod Dent Traumatol 1988;4: 202.
12. Andreasen FM, Andreasen JO, Bayer T. *Prognosis of root-fractured permanent incisors: prediction of healing modalities*, Endod Dent Traumatol 1989;5: 11.
13. Andreasen FM, Kahler B. *Diagnosis of acute dental trauma: the importance of standardized documentation: a review*, Dent Traumatol 2015;31: 340.
14. Andreasen FM, Kahler B. *Pulpal response after acute dental injury in the permanent dentition: clinical implications-a review*, J Endod 2015;41: 299.
15. Andreasen FM, Pedersen BV. *Prognosis of luxated permanent teeth: the development of pulp necrosis*, Endod Dent Traumatol 1985;1: 207.
16. Andreasen FM, Zhijie Y, Thomsen BL, et al: *Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition*, Endod Dent Traumatol 1987;3: 103.
17. Andreasen JO. *Etiology and pathogenesis of traumatic dental injuries: a clinical study of 1,298 cases*, Scand J Dent Res 1970;78: 329.
18. Andreasen JO. *Luxation of permanent teeth due to trauma: a clinical and radiographic follow-up study of 189 injured teeth*, Scand J Dent Res 1970;78: 273.
19. Andreasen JO. *Effect of extra-alveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys*, Int J Oral Surg 1981;10: 43.
20. Andreasen JO. *Review of root resorption systems and models: etiology of root resorption and the homeostatic*

Telegram: @dental_k

www.konkur.in

mechanisms of the periodontal ligament, Davidovitch Z The biological mechanisms of tooth eruption and resorption 1989; EB-SCOP Media Birmingham, AL.

21. Andreasen JO, Andreasen FM, Andersson L. Textbook and color atlas of traumatic injuries to the teeth ed 4 2007; Wiley-Blackwell Hoboken, NJ.
22. Andreasen JO, Andreasen FM, Mejare I, et al: *Healing of 400 intra-alveolar root fractures. Part 1. Effect of pre-injury and injury factors such as sex, age, stage of root development, fracture type, location of fracture and severity of dislocation*, Dent Traumatol 2004;20: 192.
23. Andreasen JO, Andreasen FM, Mejare I, et al: *Healing of 400 intra-alveolar root fractures. Part 2. Effect of treatment factors such as treatment delay, repositioning, splinting type and period and antibiotics*, Dent Traumatol 2004;20: 203.
24. Andreasen JO, Farik B, Munksgaard EC. *Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture*, Dent Traumatol 2002;18: 134.
25. Andreasen JO, Hjorting-Hansen E. *Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss*, Acta Odontol Scand 1966;24: 263.
26. Andreasen JO, Hjorting-Hansen E. *Intraalveolar root fractures: radiographic and histologic study of 50 cases*, J Oral Surg 1967;25: 414.
27. Andreasen JO, Kristerson L. *The effect of limited drying or removal of the periodontal ligament: periodontal healing after replantation of mature permanent incisors in monkeys*, Acta Odontol Scand 1981;39: 1.
28. Andreasen JO, Ravn JJ. *Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish*

Telegram: @dental_k

www.konkur.in

population sample, Int J Oral Surg 1972;1: 235.
29. Arakawa M, Kitasako Y, Otsuki M, et al: *Direct pulp capping with an auto-cured sealant resin and a self-etching primer*, Am J Dent 2003;16: 61.
30. Banchs F, Trope M. *Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol?*, J Endod 2004;30: 196.
31. Barrett AP, Reade PC. *Revascularization of mouse tooth isografts and allografts using autoradiography and carbon-perfusion*, Arch Oral Biol 1981;26: 541.
32. Barrett EJ, Kenny DJ. *Avulsed permanent teeth: a review of the literature and treatment guidelines*, Endod Dent Traumatol 1997;13: 153.
33. Bastone EB, Freer TJ, McNamara JR. *Epidemiology of dental trauma: a review of the literature*, Aust Dent J 2000;45: 2.
34. Becerra P, Ricucci D, Loghin S, et al: *Histological study of a human immature permanent premolar with chronic apical abscess after revascularization/revitalization*, J Endod 2014;40: 133.
35. Belobrov I, Parashos P. *Treatment of tooth discoloration after the use of white mineral trioxide aggregate*, J Endod 2011;37: 1017.
36. Bergenholtz G. *Micro-organisms from necrotic pulp of traumatized teeth*, Odontol Revy 1974;25: 347.
37. Bernardes RA, de Moraes IG, Duarte MAH, et al: *Use of cone-beam volumetric tomography in the diagnosis of root fractures*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108: 270.
38. Bernardes RA, de Paulo RS, Pereira LO, et al: *Comparative study of cone beam computed tomography and intraoral periapical radiographs in diagnosis of*

Telegram: @dental_k

www.konkur.in

lingual-simulated external root resorptions, Dent Traumatol 2012;28: 268.

39. Bhaskar SN, Rappaport HM. *Dental vitality tests and pulp status*, J Am Dent Assoc 1973;86: 409.
40. Binnie WH, Rowe AH. *A histological study of the periapical tissues of incompletely formed pulpless teeth filled with calcium hydroxide*, J Dent Res 1973;52: 1110.
41. Bjorvatn K, Selvig KA, Klinge B. *Effect of tetracycline and SnF2 on root resorption in replanted incisors in dogs*, Scand J Dent Res 1989;97: 477.
42. Blomlof L. *Milk and saliva as possible storage media for traumatically exarticulated teeth prior to replantation*, Swed Dent J Suppl 1981;8: 1.
43. Blomlof L, Lindskog S, Andersson L, et al: *Storage of experimentally avulsed teeth in milk prior to replantation*, J Dent Res 1983;62: 912.
44. Bose R, Nummikoski P, Hargreaves K. *A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures*, J Endod 2009;35: 1343.

44a Bourguignon C, Cohenca N, Lauridsen E, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations*, Dent Traumatol 2020; doi:10.1111/edt.12578.

45. Bryson EC, Levin L, Banchs F, et al: *Effect of immediate intracanal placement of Ledermix Paste on healing of replanted dog teeth after extended dry times*, Dent Traumatol 2002;18: 316.
46. Bystrom A, Claesson R, Sundqvist G. *The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the*

Telegram: @dental_k

www.konkur.in

treatment of infected root canals, Endod Dent Traumatol 1985;1: 170.

47. Canakci V, Akgul HM, Akgul N, et al: *Prevalence and handedness correlates of traumatic injuries to the permanent incisors in 13 17-year-old adolescents in Erzurum, Turkey*, Dent Traumatol 2003;19: 248.
48. Chala S, Abouqal R, Rida S. *Apexification of immature teeth with calcium hydroxide or mineral trioxide aggregate: systematic review and meta-analysis*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: e36.
49. Chen H, Teixeira FB, Ritter AL, et al: *The effect of intracanal anti-inflammatory medicaments on external root resorption of replanted dog teeth after extended extra-oral dry time*, Dental Traumatol 2008;24: 74.
50. Cohenca N, Simon JH, Marhtur A, et al: *Clinical indications for digital imaging in dento-alveolar trauma. Part 2. Root resorption*, Dent Traumatol 2007;23: 105.
51. Cohenca N, Simon JH, Roges R, et al: *Clinical indications for digital imaging in dento-alveolar trauma. Part 1. Traumatic injuries*, Dent Traumatol 2007;23: 95.
52. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone beam volumetric tomography*, J Endod 2007;33: 1121.
53. Cox CF, Keall CL, Keall HJ, et al: *Biocompatibility of surface-sealed dental materials against exposed pulps*, J Prosthet Dent 1987;57: 1.
54. Cox CF, Tarim B, Kopel H, et al: *Technique sensitivity: biological factors contributing to clinical success with various restorative materials*, Adv Dent Res 2001;15: 85.
55. Cunha RF, Pavarini A, Percinoto C, et al: *Influence of surgical repositioning of mature permanent dog teeth following experimental intrusion: a histologic assessment*,

Telegram: @dental_k

www.konkur.in

Dent Traumatol 2002;18: 304.

56. Cvek M. *Treatment of non-vital permanent incisors with calcium hydroxide. IV. Periodontal healing and closure of the root canal in the coronal fragment of teeth with intra-alveolar fracture and vital apical fragment: a follow-up*, Odontol Revy 1974;25: 239.
57. Cvek M. *A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture*, J Endod 1978;4: 232.
58. Cvek M. *Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha: a retrospective clinical study*, Endod Dent Traumatol 1992;8: 45.
59. Cvek M, Cleaton-Jones P, Austin J, et al: *Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors*, Endod Dent Traumatol 1990;6: 170.
60. Cvek M, Cleaton-Jones PE, Austin JC, et al: *Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys*, J Endod 1982;8: 391.
61. Cvek M, Hollender L, Nord CE. *Treatment of non-vital permanent incisors with calcium hydroxide. VI. A clinical, microbiological and radiological evaluation of treatment in one sitting of teeth with mature or immature root*, Odontol Revy 1976;27: 93.
62. Cvek M, Nord CE, Hollender L. *Antimicrobial effect of root canal debridement in teeth with immature root: a clinical and microbiologic study*, Odontol Revy 1976;27: 1.
63. Da Silva AC, Passeri LA, Mazzonetto R, et al: *Incidence of dental trauma associated with facial trauma in Brazil: a 1-year evaluation*, Dent Traumatol 2004;20: 6.

Telegram: @dental_k

www.konkur.in

64. Day P, Flores MT, O'Connell A, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 3. Injuries in the primary dentition*, Dent Traumatol 2020; doi:10.1111/edt.12576.
65. Deutsch AS, Musikant BL, Cavallari J, et al: *Root fracture during insertion of prefabricated posts related to root size*, J Prosthet Dent 1985;53: 786.
66. Durack C, Patel S, Davies J, et al: *Diagnostic accuracy of small volume cone beam computed tomography and intraoral periapical radiography for the detection of simulated external inflammatory root resorption*, Int Endod J 2011;44: 136.
67. Ebeleseder KA, Santler G, Glockner K, et al: *An analysis of 58 traumatically intruded and surgically extruded permanent teeth*, Endod Dent Traumatol 2000;16: 34.
68. Eklund G, Stalhane I, Hedegard B. *A study of traumatized permanent teeth in children aged 7-15 years. III. A multivariate analysis of post-traumatic complications of subluxated and luxated teeth*, Sven Tandlak Tidskr 1976;69: 179.
69. Endodontics Colleagues for Excellence. Summer 2014. The treatment of traumatic dental injuries. https://www.aae.org/specialty/wp-content/uploads/sites/2/2017/06/ecfe_summer2014-final.pdf.
70. Ersin NK, Eronat N. *The comparison of a dentin adhesive with calcium hydroxide as a pulp-capping agent on the exposed pulps of human and sheep teeth*, Quintessence Int 2005;36: 271.
71. Estrela C, Reis Bueno M, Alencar AHG. *Method to evaluate inflammatory root resorption by using cone beam*

Telegram: @dental_k

www.konkur.in

computed tomography, J Endod 2009;35: 1491.

72. Faraco IM Jr, Holland R. *Response of the pulp of dogs to capping with mineral trioxide aggregate or a calcium hydroxide cement*, Dent Traumatol 2001;17: 163.
73. Filippi A, Pohl Y, von Arx T. *Decoronation of an ankylosed tooth for preservation of alveolar bone prior to implant placement*, Dent Traumatol 2001;17: 93.
74. Filippi A, Pohl Y, von Arx T. *Treatment of replacement resorption with Emdogain: preliminary results after 10 months*, Dent Traumatol 2001;17: 134.
75. Flores MT. *Traumatic injuries in the primary dentition*, Dent Traumatol 2002;18: 287.

75a Fouad AF, Abbott PV, Tsilingaridis G, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth*, Dent Traumatol 2020; doi:10.1111/edt.12573.

76. Frank AL. *Therapy for the divergent pulpless tooth by continued apical formation*, J Am Dent Assoc 1966;72: 87.
77. Fuks AB, Bielak S, Chosak A. *Clinical and radiographic assessment of direct pulp capping and pulpotomy in young permanent teeth*, Pediatr Dent 1982;4: 240.
78. Fuks AB, Cosack A, Klein H, et al: *Partial pulpotomy as a treatment alternative for exposed pulps in crown-fractured permanent incisors*, Endod Dent Traumatol 1987;3: 100.
79. Fulling HJ, Andreasen JO. *Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing*, Scand J Dent Res 1976;84: 286.
80. Fuss Z, Trowbridge H, Bender IB, et al: *Assessment of reliability of electrical and thermal pulp testing agents*, J

Telegram: @dental_k

www.konkur.in

Endod 1986;12: 301.
81. Gazelius B, Olgart L, Edwall B. *Restored vitality in luxated teeth assessed by laser Doppler flowmeter*, Endod Dent Traumatol 1988;4: 265.
82. Gelbier MJ, Winter GB. *Traumatised incisors treated by vital pulpotomy: a retrospective study*, Br Dent J 1988;164: 319.
83. Giuliani V, Baccetti T, Pace R, et al: *The use of MTA in teeth with necrotic pulps and open apices*, Dent Traumatol 2002;18: 217.
84. Goldberg F, Kaplan A, Roitman M, et al: *Reinforcing effect of a resin glass ionomer in the restoration of immature roots in vitro*, Dent Traumatol 2002;18: 70.
85. Granath LE, Hagman G. *Experimental pulpotomy in human bicuspids with reference cutting technique*, Acta Odontol Scand 1971;29: 155.
86. Hammarstrom L, Lindskog S. *General morphological aspects of resorption of teeth and alveolar bone*, Int Endod J 1985;18: 93.
87. Hammarstrom L, Pierce A, Blomlof L, et al: *Tooth avulsion and replantation: a review*, Endod Dent Traumatol 1986;2: 1.
88. Hebling J, Giro EM, Costa CA. *Biocompatibility of an adhesive system applied to exposed human dental pulp*, J Endodontol 1999;25: 676.
89. Hedegard B, Stalhane I. *A study of traumatized permanent teeth in children 7-15 years. I*, Sven Tandlak Tidskr 1973;66: 431.
90. Heide S, Mjor IA. *Pulp reactions to experimental exposures in young permanent monkey teeth*, Int Endod J 1983;16: 11.
91. Heithersay GS. *Calcium hydroxide in the treatment of*

Telegram: @dental_k

www.konkur.in

pulpless teeth with associated pathology, J Br Endod Soc 1975;8: 74.

92. Heithersay GS. *Clinical, radiologic, and histopathologic features of invasive cervical resorption*, Quintessence Int 1999;30: 27.
93. Heithersay GS. *Invasive cervical resorption: an analysis of potential predisposing factors*, Quintessence Int 1999;30: 83.
94. Heithersay GS. *Treatment of invasive cervical resorption: an analysis of results using topical application of trichloracetic acid, curettage, and restoration*, Quintessence Int 1999;30: 96.
95. Heithersay GS. *Life cycles of traumatized teeth: long-term observations from a cohort of dental trauma victims*, Aust Dent J Suppl 1, 2016;61: 120.
96. Heithersay GS. *Life cycles of traumatized teeth: long-term observations from a cohort of dental trauma victims-series 2*, Aust Dent J Suppl 1, 2016;61: 317.
97. Heithersay GS, Kahler B. *Healing responses following transverse root fracture: a historical review and case reports showing healing with (a) calcified tissue and (b) dense fibrous connective tissue*, Dent Traumatol 2013;29: 253.
98. Heller AL, Koenigs JF, Brilliant JD, et al: *Direct pulp capping of permanent teeth in primates using a resorbable form of tricalcium phosphate ceramic*, J Endod 1975;1: 95.
99. Herforth A, Strassburg M. *Therapy of chronic apical periodontitis in traumatically injuring front teeth with ongoing root growth*, Dtsch Zahnarztl Z 1977;32: 453.
100. Hiltz J, Trope M. *Vitality of human lip fibroblasts in milk, Hanks Balanced Salt Solution and Viaspan storage media*, Endod Dent Traumatol 1991;7: 69.

Telegram: @dental_k

www.konkur.in

101. Holland R, Bisco Ferreira L, de Souza V, et al: *Reaction of the lateral periodontium of dogs' teeth to contaminated and noncontaminated perforations filled with mineral trioxide aggregate*, J Endod 2007;33: 1192.
102. Holloway GA Jr, Watkins DW. *Laser Doppler measurement of cutaneous blood flow*, J Invest Dermatol 1977;69: 306.
103. Hoshino E, Kurihara-Ando N, Sato I, et al: *In-vitro antibacterial susceptibility of bacteria taken from infected root dentine to a mixture of ciprofloxacin, metronidazole and minocycline*, Int Endod J 1996;29: 125.
104. Iqbal MK, Bamaas N. *Effect of enamel matrix derivative (Emdogain) upon periodontal healing after replantation of permanent incisors in beagle dogs*, Dent Traumatol 2001;17: 36.
105. Iwaya S, Ikawa M, Kubota M. *Revascularization of an immature permanent tooth with apical periodontitis and sinus tract*, Dental Traumatol 2001;17: 185.
106. Jacobsen I. *Root fractures in permanent anterior teeth with incomplete root formation*, Scand J Dent Res 1976;84: 210.
107. Jacobsen I, Kerekes K. *Diagnosis and treatment of pulp necrosis in permanent anterior teeth with root fracture*, Scand J Dent Res 1980;88: 370.
108. Jacobsen I, Zachrisson BU. *Repair characteristics of root fractures in permanent anterior teeth*, Scand J Dent Res 1975;83: 355.
109. Jarvinen S. *Fractured and avulsed permanent incisors in Finnish children: a retrospective study*, Acta Odontol Scand 1979;37: 47.
110. Kahler B, Heithersay GS. *An evidence-based appraisal of splinting luxated, avulsed and root-fractured teeth*, Dent

Telegram: @dental_k

www.konkur.in

Traumatol 2008;24: 2.
111. Kahler B, Hu JY, Marriot-Smith CS, et al: *Splinting of teeth following trauma: a review and a new splinting recommendation*, Aust Dent J Suppl 1, 2016;61: 59.
112. Kahler B, Mistry S, Moule A, et al: *Revascularization outcomes: a prospective analysis of 16 consecutive cases*, J Endod 2014;40: 333.
113. Kahler B, Rossi-Fedele G. *A review of tooth discoloration after regenerative endodontic therapy*, J Endod 2016;42: 563.
114. Kahler B, Rossi-Fedele G, Chugal N, et al: *An evidence-based review of the efficacy of treatment approaches for immature permanent teeth with pulp necrosis*, J Endod 2017;43: 1052.
115. Kahler SL, Shetty S, Andreasen FM, et al: *The effect of long-term dressing with calcium hydroxide on the fracture susceptibility of teeth*, J Endod 2018;44: 464.
116. Kakehashi S, Stanley HR, Fitzgerald RJ. *The effect of surgical exposures on dental pulps in germ-free and conventional laboratory rats*, Oral Surg 1965;20: 340.
117. Kamburoglu K, Ilker Cebeci AR, Göran Gröndahl H. *Effectiveness of limited cone-beam computed tomography in the detection of horizontal root fracture*, Dent Traumatol 2009;25: 256.
118. Katebzadeh N, Dalton BC, Trope M. *Strengthening immature teeth during and after apexification*, J Endod 1998;24: 256.
119. Kenny DJ, Barrett EJ, Casas MJ. *Avulsions and intrusions: the controversial displacement injuries*, J Can Dent Assoc 2003;69: 308.
120. Kerekes K, Heide S, Jacobsen I. *Follow-up examination of endodontic treatment in traumatized juvenile incisors*, J

Telegram: @dental_k

www.konkur.in

Endod 1980;6: 744.
121. Kim SG, Malek M, Sigurdsson A, et al: *Regenerative endodontics: a comprehensive review*, Int Endod J 2018;51: 1367.
122. Koliniotou-Koumpia E, Tziafas D. *Pulpal responses following direct pulp capping of healthy dog teeth with dentine adhesive systems*, J Dent 2005;33: 639.
123. Kositbowornchai S, Nuansakul R, Sikram S, et al: *Root fracture detection: a comparison of direct digital radiography with conventional radiography*, Dentomaxillofac Radiol 2001;30: 106.
124. Kwan SC, Johnson JD, Cohenca N. *The effect of splint material and thickness on tooth mobility after extraction and replantation using a human cadaveric model*, Dent Traumatol 2012;28: 277.
125. Lengheden A, Blomlof L, Lindskog S. *Effect of delayed calcium hydroxide treatment on periodontal healing in contaminated replanted teeth*, Scand J Dent Res 1991;99: 147.
126. Lindskog S, Pierce AM, Blomlof L, et al: *The role of the necrotic periodontal membrane in cementum resorption and ankylosis*, Endod Dent Traumatol 1985;1: 96.
127. Love RM. *Bacterial penetration of the root canal of intact incisor teeth after a simulated traumatic injury*, Endod Dent Traumatol 1996;12: 289.
128. Love RM, Ponnambalam Y. *Dental and maxillofacial skeletal injuries seen at the University of Otago School of Clinicianry, New Zealand, 2000-2004*, Dent Traumatol 2008;24: 170.
129. Lundin SA, Noren JG, Warfvinge J. *Marginal bacterial leakage and pulp reactions in Class II composite resin restorations in vivo*, Swed Dent J 1990;14: 185.

Telegram: @dental_k

www.konkur.in

130. Malmgren B, Andreasen JO, Flores MT, et al: *International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 3. Injuries in the primary dentition*, Dent Traumatol 2012;28: 174.
131. Maroto M, Barberia E, Planells P, et al: *Treatment of a non-vital immature incisor with mineral trioxide aggregate (MTA)*, Dent Traumatol 2003;19: 165.
132. Martin G, Ricucci D, Gibbs JL, et al: *Histological findings of revascularized/revitalized immature permanent molar with apical periodontitis using platelet-rich plasma*, J Endod 2013;39: 138.
133. Masterton JB. *The healing of wounds of the dental pulp: an investigation of the nature of the scar tissue and of the phenomena leading to its formation*, Dent Pract Dent Rec 1966;16: 325.
134. May JJ, Cohenca N, Peters OA. *Contemporary management of horizontal root fractures to the permanent dentition: diagnosis, radiologic assessment to include cone-beam computed tomography*, Pediatr Dent 2013;35: 120.
135. Mejare I, Hasselgren G, Hammarstrom LE. *Effect of formaldehyde-containing drugs on human dental pulp evaluated by enzyme histochemical technique*, Scand J Dent Res 1976;84: 29.
136. Mesaros S, Trope M, Maixner W, et al: *Comparison of two laser Doppler systems on the measurement of blood flow of premolar teeth under different pulpal conditions*, Int Endod J 1997;30: 167.
137. Murray PE, Smith AJ, Windsor LJ, et al: *Remaining dentine thickness and human pulp responses*, Int Endod J 2003;33: 36.

Telegram: @dental_k

www.konkur.in

138. Myers WC, Fountain SB. *Dental pulp regeneration aided by blood and blood substitutes after experimentally induced periapical infection*, Oral Surg Oral Med Oral Pathol 1974;37: 441.
139. Nair PN, Duncan HF, Pitt Ford TR, et al: *Histological, ultrastructural and quantitative investigations on the response of healthy human pulps to experimental capping with mineral trioxide aggregate: a randomized controlled trial*, Int Endod J 2008;41: 128.
140. Ohman A. *Healing and sensitivity to pain in young replanted human teeth: an experimental, clinical and histological study*, Odontol Tidskr 1965;73: 166.
141. Parirokh M, Kakoei S. *Vital pulp therapy of mandibular incisors: a case report with 11-year follow up*, Aust Endod J 2006;32: 75.
142. Patel S, Dawood A, Pitt Ford T, et al: *The potential applications of cone beam computed tomography in the management of endodontic problems*, Int Endod J 2007;40: 818.
143. Patel S, Durack C, Abella F, et al: *Cone beam computed tomography in endodontics: a review*, Int Endod J 2015;48: 3.
144. Pierce A, Heithersay G, Lindskog S. *Evidence for direct inhibition of dentinoclasts by a corticosteroid/antibiotic endodontic paste*, Endod Dent Traumatol 1988;4: 44.
145. Pierce A, Lindskog S. *The effect of an antibiotic/corticosteroid paste on inflammatory root resorption in vivo*, Oral Surg Oral Med Oral Pathol 1987;64: 216.
146. Pileggi R, Dumsha TC, Myslinksi NR. *The reliability of electric pulp test after concussion injury*, Endod Dent Traumatol 1996;12: 16.

Telegram: @dental_k

www.konkur.in

147. Rabie G, Barnett F, Tronstad L. *Long-term splinting of maxillary incisor with intra alveolar root fracture*, Endod Dent Traumatol 1988;4: 99.
148. Ravn JJ. *Follow-up study of permanent incisors with complicated crown fractures after acute trauma*, Scand J Dent Res 1982;90: 363.
149. Ray HA, Trope M. *Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration*, Int Endod J 1995;28: 12.
150. Ritter AL, Ritter AV, Murrah V, et al: *Pulp revascularization of replanted immature dog teeth after treatment with minocycline and doxycycline assessed by laser Doppler flowmetry, radiography, and histology*, Dent Traumatol 2004;20: 75.
151. Sae-Lim V, Wang CY, Choi GW, et al: *The effect of systemic tetracycline on resorption of dried replanted dogs' teeth*, Endod Dent Traumatol 1998;14: 127.
152. Sae-Lim V, Wang CY, Trope M. *Effect of systemic tetracycline and amoxicillin on inflammatory root resorption of replanted dogs' teeth*, Endod Dent Traumatol 1998;14: 216.
153. Saroglu I, Sonmez H. *The prevalence of traumatic injuries treated in the pedodontic clinic of Ankara University, Turkey, during 18 months*, Dent Traumatol 2002;18: 299.
154. Schjott M, Andreasen JO. *Emdogain does not prevent progressive root resorption after replantation of avulsed teeth: a clinical study*, Dental Traumatol 2005;21: 46.
155. Schroder U. *Reaction of human dental pulp to experimental pulpotomy and capping with calcium hydroxide (thesis)*, Odont Revy Suppl 25, 1973;24: 97.

Telegram: @dental_k

www.konkur.in

156. Schroder U, Granath LE. *Early reaction of intact human teeth to calcium hydroxide following experimental pulpotomy and its significance to the development of hard tissue barrier*, Odontol Revy 1971;22: 379.
157. Seltzer S. Endodontology 1988; Lea & Febiger Philadelphia.
158. Seltzer S, Bender IB, Turkenkopf S. *Factors affecting successful repair after root canal therapy*, J Am Dent Assoc 1963;52: 651.
159. Selvig KA, Zander HA. *Chemical analysis and microradiography of cementum and dentin from periodontically diseased human teeth*, J Periodontol 1962;33: 103.
160. Shuping GB, Orstavik D, Sigurdsson A, et al: *Reduction of intracanal bacteria using nickel-titanium rotary instrumentation and various medications*, J Endod 2000;26: 751.
161. Silva GA, Lanza LD, Lopes-Junior N, et al: *Direct pulp capping with a dentin bonding system in human teeth: a clinical and histological evaluation*, Oper Dent 2006;31: 297.
162. Silverman S. *The dental structures in primary hyperparathyroidism*, Oral Surg 1962;15: 426.
163. Sjogren U, Figdor D, Spangberg L, et al: *The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing*, Int Endod J 1991;24: 119.
164. Sjogren U, Hagglund B, Sundqvist G, et al: *Factors affecting the long-term results of endodontic treatment*, J Endod 1990;16: 498.
165. Skaare AB, Jacobsen I. *Dental injuries in Norwegians aged 7-18 years*, Dent Traumatol 2003;19: 67.
166. Skieller V. *The prognosis for young teeth loosened after*

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mechanical injuries, Acta Odontol Scand 1960;18: 171.
167. Skoglund A, Tronstad L. *Pulpal changes in replanted and autotransplanted immature teeth of dogs*, J Endod 1981;7: 309.
168. Soder PO, Otteskog P, Andreasen JO, et al: *Effect of drying on viability of periodontal membrane*, Scand J Dent Res 1977;85: 164.
169. Spangberg L, Rutberg M, Rydinge E. *Biologic effects of endodontic antimicrobial agents*, J Endod 1979;5: 166.
170. Stalhane I, Hedegard B. *Traumatized permanent teeth in children aged 7-15 years*, Sven Tandlak Tidskr 1975;68: 157.
171. Stanley HR. *Diseases of the dental pulp*, Tieck RW Oral pathology 1965; McGraw-Hill New York.
172. Stanley HR, Lundy T. *Dycal therapy for pulp exposures*, Oral Surg Oral Med Oral Pathol 1972;34: 818.
173. Subay RK, Demirci M. *Pulp tissue reactions to a dentin bonding agent as a direct capping agent*, J Endod 2005;31: 201.
174. Swift EJ Jr, Trope M. *Treatment options for the exposed vital pulp*, Pract Periodontics Aesthet Dent 1999;11: 735.
175. Tapias MA, Jimenez-Garcia R, Lamas F, et al: *Prevalence of traumatic crown fractures to permanent incisors in a childhood population: Mostoles, Spain*, Dent Traumatol 2003;19: 119.
176. Teixeira FB, Teixeira EC, Thompson JY, et al: *Fracture resistance of roots endodontically treated with a new resin filling material*, J Am Dent Assoc 2004;135: 646.
177. Torabinejad M, Hong CU, McDonald F, et al: *Physical and chemical properties of a new root-end filling material*, J Endod 1995;21: 349.
178. Torabinejad M, Rastegar AF, Kettering JD, et al:

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Bacterial leakage of mineral trioxide aggregate as a root-end filling material, J Endod 1995;21: 109.

179. Trabert KC, Caput AA, Abou-Rass M. *Tooth fracture: a comparison of endodontic and restorative treatments*, J Endod 1978;4: 341.
180. Tronstad L. *Reaction of the exposed pulp to Dycal treatment*, Oral Surg Oral Med Oral Pathol 1974;38: 945.
181. Tronstad L. *Root resorption: etiology, terminology and clinical manifestations* Endod Dent Traumatol 1988;4: 241.
182. Tronstad L, Andreasen JO, Hasselgren G, et al: *pH changes in dental tissues after root canal filling with calcium hydroxide*, J Endod 1980;7: 17.
183. Tronstad L, Mjor IA. *Capping of the inflamed pulp*, Oral Surg Oral Med Oral Pathol 1972;34: 477.
184. Trope M. *Root resorption of dental and traumatic origin: classification based on etiology*, Pract Periodontics Aesthet Dent 1998;10: 515.
185. Trope M. *Clinical management of the avulsed tooth: present strategies and future directions*, Dent Traumatol 2002;18: 1.
186. Trope M, Friedman S. *Periodontal healing of replanted dog teeth stored in Viaspan, milk and Hank's balanced salt solution*, Endod Dent Traumatol 1992;8: 183.
187. Trope M, Moshonov J, Nissan R, et al: *Short vs long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth*, Endod Dent Traumatol 1995;11: 124.
188. Trope M, Yesilsoy C, Koren L, et al: *Effect of different endodontic treatment protocols on periodontal repair and root resorption of replanted dog teeth*, J Endod 1992;18: 492.

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www.konkur.in

189. Turley PK, Joiner MW, Hellstrom S. *The effect of orthodontic extrusion on traumatically intruded teeth*, Am J Orthod 1984;85: 47.
190. von Arx T, Filippi A, Buser D. *Splinting of traumatized teeth with a new device: TTS (titanium trauma splint)*, Dent Traumatol 2001;17: 180.
191. Wedenberg C, Lindskog S. *Experimental internal resorption in monkey teeth*, Endod Dent Traumatol 1985;1: 221.
192. Wedenberg C, Zetterqvist L. *Internal resorption in human teeth: a histological, scanning electron microscopic, and enzyme histochemical study*, J Endod 1987;13: 255.
193. Weiss M. *Pulp capping in older patients*, N Y State Dent J 1966;32: 451.
194. Windley W, Teixeira F, Levin L, et al: *Disinfection of immature teeth with a triple antibiotic paste*, J Endod 2005;31: 439.
195. Witherspoon DE, Small JC, Harris GZ. *Mineral trioxide aggregate pulpotomies: a case series outcomes assessment*, J Am Dent Assoc 2006;137: 610.
196. Yanpiset K, Trope M. *Pulp revascularization of replanted immature dog teeth after different treatment methods*, Endod Dent Traumatol 2000;16: 211.
197. Yanpiset K, Vongsavan N, Sigurdsson A, et al: *Efficacy of laser Doppler flowmetry for the diagnosis of revascularization of reimplanted immature dog teeth*, Dent Traumatol 2001;17: 63.
198. Yoldaş SE, Bani M, Atabek D, et al: *Comparison of the potential discoloration effect of Bioaggregate, Biodentine, and white mineral trioxide aggregate on bovine teeth: In vitro research*, J Endod 2016;42: 1815.

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199. Zachrisson BU, Jacobsen I. *Long-term prognosis of 66 permanent anterior teeth with root fracture*, Scand J Dent Res 1975;83: 345.

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*The author acknowledges the outstanding work of Drs. Trope, Barnett, Sigurdsson, and Chivian in previous editions of this text. The present chapter is built on their foundational work.

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22: Chronic cracks and fractures

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CHAPTER OUTLINE

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The presence of cracks or fractures in teeth can be difficult to diagnose and clinically manage. They may present with no symptoms or the symptoms may be vague or specific. Symptoms alone are often insufficient for making a definitive diagnosis and the radiographic interpretation can be inconclusive. Although the clinical management of the crack or fracture depends on its extent, often the extent of the crack or fracture cannot be clinically determined.

According to the 2016 version of the American Association of Endodontists (AAE) *Glossary of Endodontic Terms*, a *crack* is defined as "a thin surface disruption of enamel and dentin, and possibly cementum, of unknown depth or extension." A *fracture* is defined as "a split or break in bone, cartilage or tooth structure. When involving tooth structure, it is a disruption of the enamel and/or dentin and/or cementum that is assumed to be of greater depth than a crack, of unknown depth or extension, visible or not visible clinically or radiographically, and with separated or unseparated segments."[3a]

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Impact trauma

Traumatic injury to the tooth can be accompanied with a fracture in the crown or root system. These types of traumatic fractures mainly occur in the anterior segment of the mouth and are elaborated on in Chapter 21). In contrast, the cracks and fractures described in this chapter are often not associated with a traumatic event. These cracks and fractures are frequently the result of an accumulating, unobserved trauma resulting from either normal or excessive occlusal forces that are repetitively applied[22,35,46,48,71,72,91] without the patient's awareness. Specifically discussed in this chapter will be crown originating fractures and longitudinal fractures in the roots.

Diagnostic challenge

Three categories of cracks and fractures are discussed in this chapter: crown originating fractures, split teeth, and vertical root fractures (VRFs). Each is often undiagnosed or misdiagnosed for a relatively long time.[16,19,81] The astute clinician can usually diagnose a pulpitis, apical periodontitis, or an abscess after appropriate diagnostic tests and clinical assessments are performed. However, because of the wide variety of clinical presentations from cracks and fractures, the diagnosis is less straightforward.[16,18,79] To complicate the diagnosis, most cracks and fractures exhibit no radiographic manifestation in the early stages, depriving the dentist of one of the most objective diagnostic tools.

Symptoms from a crack or fracture may be slight and present for *several months* before an accurate diagnosis is made,[5,16,19] which may be frustrating for both the patient and the dentist, and often causes the patient to develop a subsequent loss of trust and confidence in the dentist. The final diagnosis is typically reached at a relatively late stage of these conditions, often after complications have already occurred. Complications may include a subsequent fracture of the tooth or cusp or significant periradicular bone loss associated with a VRF as seen by radiographic examination (see also Chapter 2). For this reason, this chapter emphasizes the *early diagnosis* of these conditions. The collection of signs and symptoms associated with cracks and fractures may be confusing; the clinician should consider these as indicators

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during the examination and diagnostic process.

Fracture mechanics

Fracture mechanics is the field of biomechanics concerned with the propagation of *cracks* in a given material until the formation of the final catastrophic *fracture*.[7,53] Many terms have been loosely used in the dental literature to describe the clinical entities that are the subject of this chapter.[5,9,18,36] Here, the term *crack* will be used in the biomechanical sense: a *partial discontinuity* in a material that may propagate and eventually lead to a complete discontinuity, also known as a *fracture*.[7,53] In this context, a cusp may be described as *cracked*, a condition that may continue over time into a *fractured cusp*. A *cracked tooth* generally presents in a longitudinal direction, along the long axis of the root. It may be so called until a final fracture occurs that separates the tooth into two parts, a condition termed a *split tooth*. Similarly, microcracks may appear in the radicular dentin of an endodontically treated tooth, and these cracks may propagate with time until a *vertical root fracture* occurs, at which point the full thickness of the dentinal wall shows discontinuity: a through-and-through fracture. Such a fracture may be *incomplete*, involving one wall of the root, or *complete*, separating the root into two parts (Fig. 22.1).[81]

FIG. 22.1 Complete vertical root fracture. A cross section of a mesial root of a mandibular molar with a complete vertical root fracture. The fracture is in the buccolingual plane and extends from the buccal convexity of the root to its lingual convexity.

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The mesial root of tooth shows two pink residues.

Cracked and fractured cusps

Definition

A *cracked cusp* is characterized by a crack between a cusp and the rest of the tooth structure, to the extent of allowing microscopic flexure upon mastication. This crack typically does not involve the pulp. With time, the crack may propagate, eventually resulting in a *fractured cusp*.[5]

Diagnosis

Patient history

In the case of a cracked cusp, the patient history is the most important tool for making a diagnosis. The patient will likely complain about pain when chewing, to the extent of not being able to chew on the side on which the crack occurred.[18,36,70] The patient will also often state that the condition existed for a relatively long period of time and that his or her dentist could not find the source or visualize any radiographic findings to suggest a cusp fracture.[3,16,48] When asked whether the pain is sharp or dull, patients will typically report a sharp pain that makes them immediately stop chewing on that side. The diagnostic challenge is attempting to determine which cusp of which tooth is involved, as patients often have difficulties determining the specific location of the discomfort.[16,18,36,70] Because the pain has a pulpal origin, the patient's proprioception may not be accurate, as no periodontal ligament is involved. Occasionally, the masticatory pain may radiate to nondental locations on the same side of the face,[16,48,72] as also described in Chapter 4.

Clinical manifestation

Early manifestation.

The typical characteristic of a cracked cusp is generally a sharp pain upon

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chewing, although the affected tooth may not be sensitive, or only selectively sensitive, to percussion.[71] The tooth is vital, and its response to a cold stimulus may be normal; but with time, this response may resemble a reversible pulpitis, which may be either localized or referred to other odontogenic or nonodontogenic locations.[16,48,72]

Cracked cusps are often associated with extensive occlusal restorations,[3,29,72,79] which may undermine and weaken the cusp and predispose it to initiating or perpetuating a crack from occlusal forces. Nevertheless, a cracked cusp may also be present in intact nonrestored teeth or teeth that are minimally restored.[13,71]

Late manifestation.

With time, a crack under a cusp may propagate and result in a fractured cusp. If the fracture line occurs supragingivally, the fractured cusp will simply separate from the tooth. However, if the fracture line extends subgingivally, gingival fibers or the periodontal ligament will often retain the fractured cusp. Initially, it may be possible to move the cusp by wedging a sharp explorer into the fracture line, making the fractured cusp more visible. Often, from continued mastication, a localized and more acute pain may develop secondary to the movement of the fractured fragment on the underlying tooth structure. The pulpal pain that is typical at an earlier stage (the cracked cusp) will typically resolve once a complete fracture occurs. However, with the resulting tooth structure having exposed dentin (or even a pulp exposure), a resulting reversible or irreversible pulpitis may ensue.

Diagnosis

A cracked cusp may be diagnosed, to a large extent, on the basis of the patient history. To locate the affected tooth, a *biting test* should be performed using a Tooth Slooth (Professional Results, Laguna Niguel, CA) or a similar device (Fig. 22.2).[9,12] The device is composed of a small pyramid with a flattened top that is placed on a selective cusp, while the wider part of the device is applied to opposing teeth while the patient occludes. The application of these forces to a cracked cusp will generate a sharp pain, which may occur upon pressure or released.[3,35,44] The patient will typically state that this sensation has reproduced the sensation of their chief complaint.

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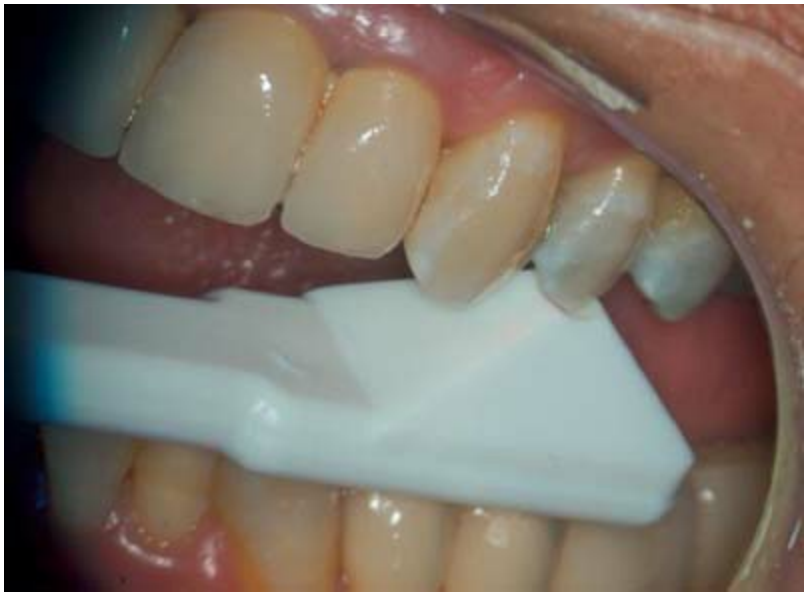

FIG. 22.2 The Tooth Slooth device. Application for a bite test: the tip of the pyramid is touching the tested cusp while the wide base is supported by multiple contacts.

Close-up view shows tooth slooth examining the grayish premolar in lower jaw.

Magnification with such devices as loupes or an operating microscope can be helpful when looking for a crack. If the tooth does not have an extensive intracoronal restoration, *transillumination* may also assist in revealing the crack line. If the tooth has a large restoration, the removal of the restoration may facilitate the effective use of this diagnostic tool (Fig. 22.3). The light source should be intense but with small dimensions; it is applied to the tooth at the area of the suspected cusp fracture, with the lights of the dental unit, microscope, and room extinguished. The light penetrates the tooth structure up to the crack, leaving the part beyond the crack relatively dark (see Fig. 22.3).

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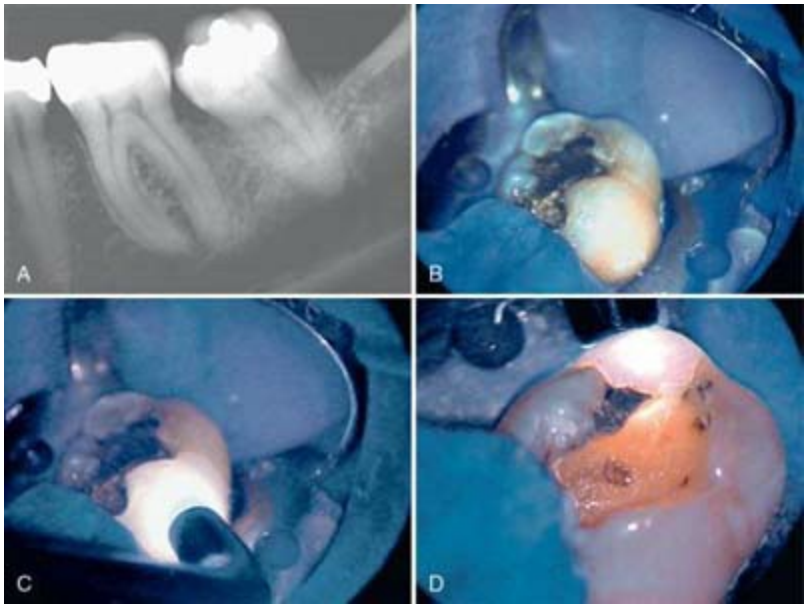

FIG. 22.3 Transillumination for the detection of a cracked cusp/tooth. An intense but small light source is applied to the suspect tooth, preferably in relative darkness. The light is transmitted through the tooth structure but is reflected from the crack plane, leaving the area behind the crack in darkness.

A) Radiograph shows right tooth with radiolucent root canals. Large part of the crown is radiopaque. Adjacent tooth has irregular radiopaque patch on the crown.

B) Tooth is isolated using rubber dam clamp and the cavity of tooth is filled with grey material.

C) Tooth is examined using light source.

D) Tooth shows the removal of upper enamel layer on one side of the cusp. The soft and tissue with a dark patch are visible.

Once the crack propagates, resulting in a fractured cusp, the diagnosis becomes more objective, as the fractured cusp will either be missing or can be moved by wedging an explorer into the fracture line (Fig. 22.4).

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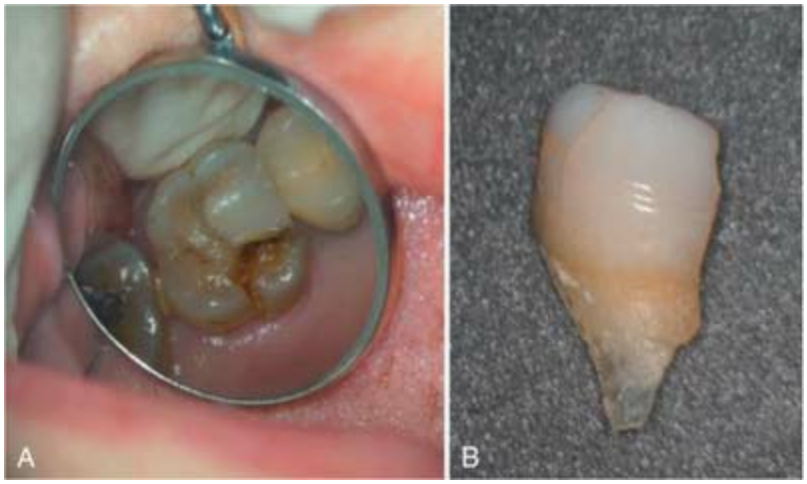

FIG. 22.4 A fractured cusp. **A,** The mesiopalatal cusp of a maxillary first right molar was fractured. The fractured cusp was movable and was retained by the periodontal ligament. **B,** The cusp was removed, and the tooth was considered restorable. It was treated with root canal treatment and a crown.

A) Close-up view shows the fractured tooth with brown stains using tooth mirror.

B) Extracted tooth having orange surface and transparent root.

Etiology

Extensive intracoronal restorations may be a predisposing factor for cracked and fractured cusps.[3,29,72,79] Otherwise, the etiology of these conditions is similar to that of cracked teeth (see Cracked and Split Teeth, Etiology).

Treatment planning

Cracked cusp

Treatment should consist of protecting the affected cusp from occlusal forces, both to prevent pain while chewing and to prevent the propagation of the crack into a full fracture. This can be facilitated with restorative treatment and an occlusal evaluation. Full-coverage crown or onlay is recommended,[5,16,40] although bonded composite restorations have also been proposed.[28,65] One should keep in mind that if the cracked cusp is not protected, the tooth may eventually fracture, complicating the treatment or

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even compromising the salvageability of the tooth. If the fracture plane extends apically into the root, the tooth may potentially become nonrestorable.[5,9,12] Endodontic treatment is indicated only if signs and symptoms of pulpal pathosis are observed. In addition, if the removal of the cracked cusp and associated restoration will result in little or no remaining coronal tooth structure, then elective root canal treatment may be necessary for prosthetic reasons. When such a treatment plan is selected, one should also perform an occlusal reduction of the tooth as soon as possible to remove the tooth from active occlusion. The patient should be instructed to be careful when chewing until the tooth is restored with a crown.

Fractured cusp

The treatment of a fractured cusp depends on the amount of tooth structure remaining. If the missing cusp is limited in size, then the conservative restoration of a bonded composite resin may be indicated to cover the exposed dentin. In contrast, when a larger fragment has fractured and is either removed or missing, a full crown or an onlay may be necessary.

In certain cases, when cracked cusps are found in intact teeth or in teeth with no extensive restoration, it is difficult to predict the direction in which the crack is propagating. Therefore, in these cases, when considering endodontic and restorative treatment, the patient should be advised as to the potential decrease in prognosis, as described later.

Cracked and split teeth

Definition

As previously described, a *cracked tooth* has “a thin surface disruption of enamel and dentin, and possibly cementum, of unknown depth or extension.” It exhibits a crack that incompletely separates the tooth crown into two parts. If the crack is allowed to propagate longitudinally, the tooth will eventually fracture into two fragments, resulting in a *split tooth*. The American Association of Endodontists describes a split tooth as “a continuation of a crack or vertical root fracture whereby the fractured segments are completely separated longitudinally; it may occur buccal-lingually or mesial-distally; it may cause an isolated periodontal defect(s) or sinus tract; it may be

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radiographically evident."

Diagnosis

Patient history

In cases of a cracked tooth, the patient history may be similar to that of a cracked cusp; there may be a sharp pain upon mastication and prolonged inability of the dentist to diagnose the source of the pain.[16,19] Similar to a cracked cusp, the diagnosis of a cracked tooth is often made on the basis of the patient history alone. Often it is challenging for the practitioner to determine the location of the offending tooth. With time, the patient may report that he or she *used to have* a sharp pain and now experiences great sensitivity to cold stimuli; the patient may even report, at a later stage, that the pain has subsided. These observations are consistent with pulpitis or pulp necrosis, which may develop in the affected tooth over time.[13]

Clinical manifestation

Early manifestation.

Cracked teeth may have extensive restorations with a weakened crown, or they may have minimal or no restorations. A cracked tooth begins with a crack in the clinical crown, which may gradually propagate in an apical direction.[3,5,28,48] Such cracks typically run in the mesiodistal direction, often splitting the crown into the buccal and lingual fragments. In the early stages, the tooth may be vital and painful to mastication. The pain may be sharp, to the extent that the patient is unable to chew on the affected side. This condition may persist for an extended period of time.[16,18,19] The pain may be localized or referred to any tooth, maxillary or mandibular, on the same side of the mouth.[16,48,72] No radiographic manifestations are present at these early stages, as the crack is microscopic and runs perpendicular to the x-ray beam. The affected tooth may or may not be sensitive to percussion at this point, and pulp testing may be normal or indicative of increased sensitivity to cold stimuli.

Late manifestation.

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The late manifestation of a cracked tooth may include pulp involvement and potentially the loss of pulp vitality[13] or apical propagation of the fracture resulting in a split tooth. In one investigation,[13] 27 nonvital molar and premolar teeth that had minimal or no restorations or caries were studied. Upon extraction, these teeth were examined under a surgical operating microscope or using micro–computed tomography (CT) analysis. In all of these teeth, a longitudinal crack was observed, which extended to the pulp and often to an external surface of the root. Although the study had a limited sample size, the clinician should understand the potentially poor prognosis from cracked teeth, especially when the crack is suspected to be the *cause* of pulp necrosis ("fracture necrosis") (Fig. 22.5).[13]

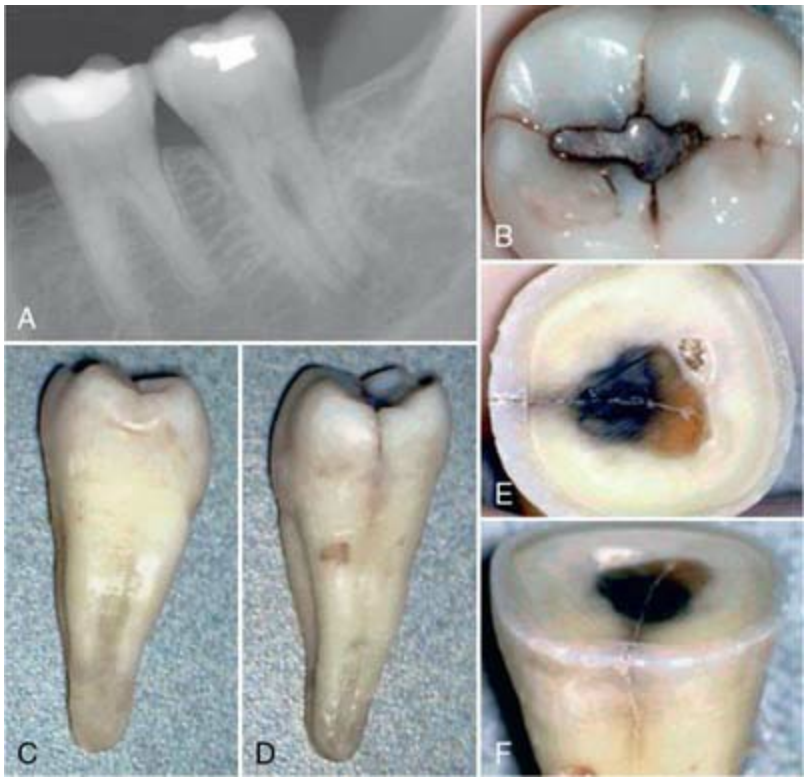

FIG. 22.5 A case of fracture-induced necrosis. A tooth with minimal or no restoration or caries is unlikely to become nonvital. **A,** This radiograph of a mandibular second molar shows a restoration that is distant from the pulp chamber, yet the tooth is nonvital and symptomatic. **B,** On occlusal examination, a slight crack is observed on the distal marginal ridge. **C,** After extraction, the mesial aspect of the crown and root shows no indication of a fracture. **D,** However, the distal aspect of the crown and coronal root shows the fracture. **E** and **F,**

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When the crown is sectioned, the crack can be observed to extend well into the pulp chamber.

A) Radiograph shows two teeth with radiopaque patches on the crown. The pulp chamber is depressed.

B) Occlusal view of tooth shows silver filling in the cavity with black lines in pits.

C) Tooth has smooth surface. The root tip is less dense.

D) Tooth with a dark spot below the neck region.

E) Tooth shows a cavity and a brown spot. The cavity has blackish residue and brown tissue.

F) Tooth with a dark hole. The lateral side of tooth has a vertical line.

Pulp involvement occurs more often in cases of centrally located cracks than in cracks with a more buccal or lingual location (i.e., extending from marginal ridge to marginal ridge through the central fossa).[18,91] These centrally located cracks commonly affect the roof of the pulp chamber at a later stage. Consequently, pulp vitality may be compromised and later lost due to bacterial penetration through the crack. The sharp pain upon mastication that is typical of the early stage may disappear once pulp vitality is lost. Consequently, apical periodontitis in an apparently intact molar may be a late manifestation of an untreated case of a cracked tooth.[13] When pulp necrosis occurs, the radiographic manifestation may be an apical radiolucency, which is undistinguishable from that of apical periodontitis (see Fig. 22.5).

A crack may propagate over time through the pulp chamber and into the root, resulting in a complete fracture that separates the tooth into two parts, a condition termed *split tooth*. This is defined in the AAE *Glossary of Endodontic Terms* as a longitudinal fracture ("a root fracture extending in the axial plane within the tooth") that becomes "a continuation of a crack or vertical root fracture whereby the fractured segments are completely separated longitudinally."[3a] When this split occurs, the resulting parts of the tooth may be movable by wedging a sharp explorer into the fissure.[5,9] Given time and masticatory forces, more evident movement of the parts may be observed. The radiographic presentation at such a late stage may eventually

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develop into a diffuse radiolucency surrounding the root. At this late stage, narrow isolated deep periodontal pockets may be present.[9] However, such pockets are typically located mesially or distally and, if adjacent teeth are present, they will be difficult if not impossible to detect.

Cracked and split teeth may present with a large and variable collection of signs and symptoms that are potentially confusing.[16,18,79] Only by being aware of the *process* leading from early to late manifestations can clinicians interpret these signs and symptoms and identify the specific point they are encountering on the potential timeline of this process.

A *definitive* combination of factors, signs, and symptoms that when collectively observed allows the clinician to conclude the existence of a specific disease state is termed a *syndrome*. However, given the multitude of signs and symptoms that cracked roots can present with, it is often difficult to achieve an objective definitive diagnosis. For this reason, the terminology of *cracked tooth syndrome*[18] should be avoided.

Diagnosis

Early detection of a cracked or split tooth is imperative to resolve the patient's symptoms as well as increasing the prognosis. However, the use of the Tooth Slooth device (see Fig. 22.2) may or may not provide as clear a result for a symmetrically cracked tooth, as each of the parts of the tooth may be rather stable. Asking the patient to chew on a cotton roll[9] or on the tip of a cotton-tip applicator placed at a particular site may reproduce the symptoms. Nevertheless, this method may not indicate whether the source is a maxillary or mandibular tooth and may need further measures to pinpoint the involved tooth. *Magnification* using either loupes or an operating microscope can be helpful for detecting a fracture line. In addition, dyes, such as methylene blue or tincture of iodine, which are applied either to the outer surface of the crown or to the dentin after the removal of an existing intracoronal restoration, can be helpful for visualizing the crack (see Fig. 22.3). Transillumination can also be applied to the suspected tooth and, if the tooth has no restorations, this method may yield an impressively straightforward diagnosis (see Fig. 22.3). Anesthetizing the suspected tooth, followed by asking the patient to chew again on the cotton roll, may further confirm the diagnosis and finally differentiate the origin as a mandibular or maxillary

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tooth. At a later stage, when splitting of the tooth has occurred, wedging of a sharp explorer into the fracture line will provide a clear diagnosis of a split tooth.

In general, the diagnosis of a crack in a tooth can be difficult. When there are signs and symptoms of a pulpitis or necrosis, it is incumbent upon the clinician to determine the source that initiated the signs and symptoms. In the case of a problematic tooth with no apparent reason for a pulpitis or necrosis, like a tooth with minimal or no caries, restoration, or trauma, a crack or fracture must be considered.[13] In some cases, an objective diagnosis may not be possible; however, with the possibility of a crack or fracture, the patient should be advised of a potential decrease in the endodontic or restorative prognosis.[5,12]

Etiology

Masticatory forces are the cause of cracked teeth.[22,35,46,48,71,72,91] Thus, the dietary habit of chewing on coarse food has been proposed as a contributing factor.[22,91] Bruxism or clenching of the teeth as well as occlusal prematurities are also frequent causes of cracked teeth.[18,22,35,46,48,71,72,91] For this reason, certain teeth may be more prone to developing cracks, such as mandibular second molars and maxillary premolars.[18] Masticatory habits such as chewing ice may also predispose teeth to cracks. The term *fatigue root fracture*, proposed by Yeh,[91] encompasses all of these causes.

In certain cases, traumatic injuries, such as a severe upward blow to the mandible (e.g., during a car or sports accident) can also cause a tooth crack or fracture. Another potential cause is the unexpected chewing of a hard object (e.g., a cherry pit or an un-popped corn kernel in popcorn). The occlusal forces applied by the first molars are as high as 90 kg,[46] which, when fully applied to a small surface area, may damage the tooth structure. However, in most cases, a tooth crack can be attributed to no specific cause other than normal or excessive masticatory forces.[22,35,46,48,71,72,91]

Treatment planning

Cracked tooth

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When a cracked tooth is either suspected or determined, the patient should be informed that the prognosis is reduced and is sometimes questionable.[5,9,12] Protecting a tooth from the propagation of a crack and improving comfort while chewing are the principal goals in the treatment of cracked teeth. Both goals can often be immediately achieved by placing an orthodontic band around the tooth[3,9,16] or by placing a provisional crown. These procedures allow the clinician to evaluate the extent of pulp involvement by checking whether the pulpal symptoms subside in response to the intervention.[3,9,16]

Protecting the tooth from further splitting forces by the placement of a permanent crown is essential in these cases.[3,5,9,16,40] Unfortunately, a crown alone is often not sufficient to resolve symptoms, and endodontic treatment may be considered prior to the placement of the permanent crown, depending on the pulpal symptoms.[9]

A root canal treatment followed by a permanent crown has the benefit of immediately eliminating the long-lasting, painful symptoms as well as early protection from occlusal forces that may cause propagation of a cracked tooth to become a split tooth. Nevertheless, when 127 cracked teeth with reversible pulpitis were treated with a crown alone, 20% of these teeth converted to irreversible pulpitis within 6 months and required root canal treatment.[52] In contrast, none of the other teeth needed a root canal over a 6-year evaluation period.[52] However, it should also be understood that varying percentages of crowned teeth, cracked or noncracked, may also require endodontic treatment, merely from the trauma of the crown preparation. By comparison, it has been shown that when suspected cracked teeth were restored with resin bonded restorations, only 7% required subsequent endodontic treatment or extraction.[65]

After removal of an intracoronal restoration or when penetrating the dentin to prepare for endodontic access, one may observe discoloration along the crack in the dentin. Once the access cavity has been completed, the pulp chamber floor and the distal and mesial walls of the pulp chamber should be carefully inspected to check for the presence and extent of any cracks. Upon evaluating 245 restored teeth, cracks were observed preoperatively in 23.3% of the teeth; however, when the restorations were removed, 60% of these teeth were found to have cracks.[1] Methylene blue dye may be helpful in this type of examination. If a crack is found that reaches from the mesial wall,

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through the floor of the pulp chamber and into the distal wall, then the prognosis for the tooth is poor, and extraction should be considered (see Fig. 22.5).[5,9,12] If the crack does not reach the pulp chamber or is limited to the coronal parts of the mesial or distal wall, the subsequent protection of the tooth with a crown may save the tooth. Nevertheless, as mentioned previously, the patient should be advised that the treatment success may be compromised and that long-term follow-up will be required.[5,9,12]

Given that such cracks occasionally occur in teeth with minimal or no restorations and that all of the pain may cease once the pulp is removed, it may be tempting to limit the final restoration to an intracoronal amalgam or composite restoration. This temptation should be resisted by all means, as the forces that caused the crack are still present, and the apical propagation of the crack and loss of the tooth is still likely.

Split tooth

When the tooth is split either through its entire length or obliquely (Fig. 22.6), extraction is typically the only treatment option.[5,9] However, if the fracture line is such that the split results in large and small segments, and if the removal of the small fragment preserves enough tooth structure that is restorable, then retention and restoration of the tooth may be considered.[5] If this is a consideration, the patient should be aware that periodontal crown lengthening may be necessary. Proper interdisciplinary management of cases like this is imperative so as to ensure that the prognosis is evaluated and maximized.

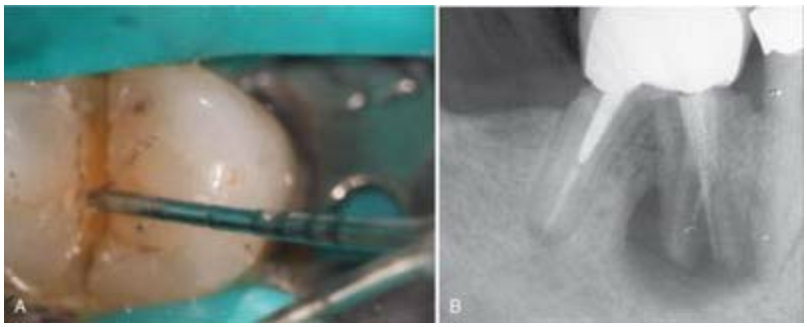

FIG. 22.6 **A,** Observing split tooth by probing or pushing cusp. **B,** Radiographic evidence of split tooth.

A) A probe is placed on the deep vertical cut of crown in tooth.

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B) Radiograph shows a large dark patch at the tip of split root canal.

Source: (**A,** Courtesy Dr. M. Malek.)

Vertical root fracture

Definition

A *vertical root fracture* is defined in the AAE *Glossary of Endodontic Terms* as a longitudinal fracture "in the root whereby the fractured segments are incompletely separated; it may occur buccal-lingually or mesial-distally; it may cause an isolated periodontal defect(s) or sinus tract; it may be radiographically evident"[3a] (Figs. 22.7–22.9; see also Fig. 22.1). Previous definitions described VRFs as initiating from the apical root and traveling coronally, or only as a result of endodontic treatment. In the latest edition of the AAE *Glossary of Endodontic Terms*, these stipulations have been removed. It is differentiated from a split root in that segments associated with the fracture are not completely separated.

Diagnosis

Patient history

In the case of a VRF, a patient may complain of pain or sensitivity related to or adjacent to a specific tooth. Sensitivity and discomfort while chewing are also common complaints.[81] Swelling may occasionally occur in the area. There is often a long history of failing to diagnose the cause of the pain and discomfort. A history of repeated clinical and radiographic examinations that revealed no cause for the pain is also common in VRF cases. After recent endodontic retreatment, if the symptoms remain and the dentist is unable to determine the cause of the symptoms, the patient may lose confidence in the dentist. Often retreatment or surgical retreatment may have been attempted to reveal the accurate diagnosis.[1] Unfortunately, such ineffective treatment attempts may only worsen the dentist-patient relationship.

Clinical manifestations

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Susceptible teeth and vertical root fracture location.

VRFs are commonly associated with endodontically treated teeth with or without a post.[5] Nevertheless, VRFs can also occur in teeth with no previous root canal treatment.[22] The most susceptible sites and tooth groups are the maxillary and mandibular premolars, mesial roots of the mandibular molars, mesio-buccal roots of the maxillary molars, and mandibular incisors.[82] However, VRFs may occasionally occur in other teeth and roots as well. VRFs may progress in the bucco-lingual direction in these teeth and roots, which are typically narrow mesiodistally and wide buccolingually.[41] However, VRFs may also propagate diagonally, thus affecting the mesial or distal aspect of the root (see Fig. 22.7). VRFs may be initiated at any root level.[5] They may also be initiated at the apical part of the root and propagate coronally (see Fig. 22.8, *C*). Some VRFs originate at the coronal-cervical part of the root and extend apically (see Fig. 22.8, *A*), and in other cases a VRF may be initiated as a midroot fracture (see Figs. 22.8, *B* and 22.9, *C* and *D*).

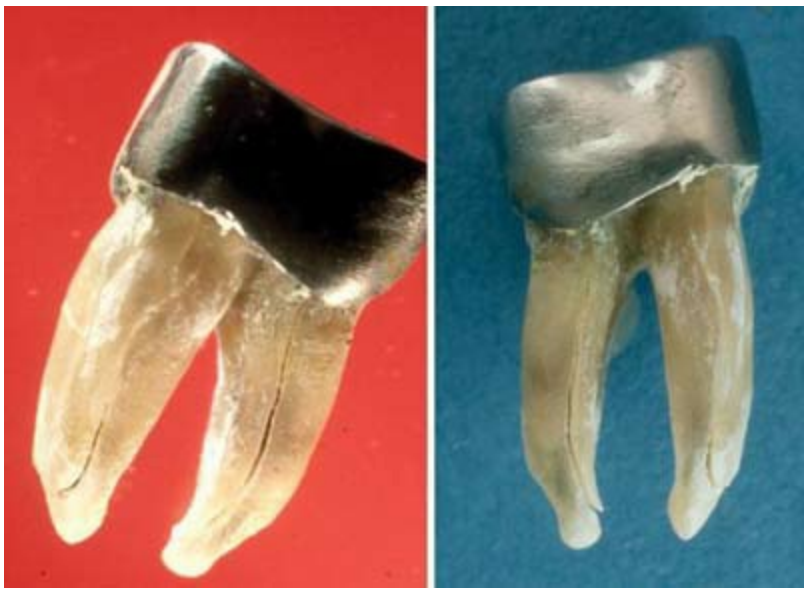

FIG. 22.7 Diagonal vertical root fracture (VRF) in a molar. A lingual view of a right first mandibular molar revealing a diagonal VRF in the mesial root.

The root fractures in the two root canals of molar are shown from two different angles. The lateral sides of crown in both views have smooth silver surface.

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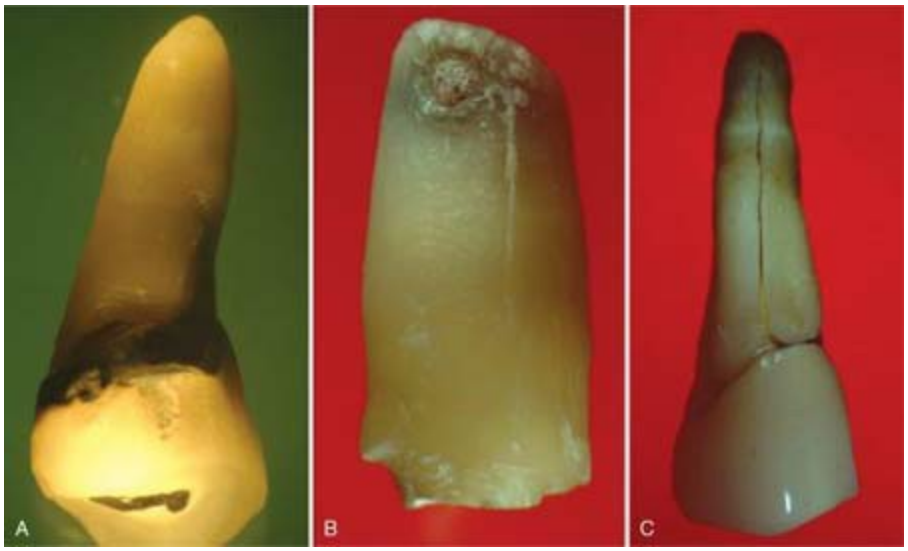

FIG. 22.8 Three types of vertical root fractures (VRFs). **A,** A coronally located VRF extending apically as far as one third of the root. **B,** A midroot VRF extending along the middle third of the root. **C,** An apically located VRF extending coronally as far as the apical two thirds of the root.

A) Tooth shows a wide crack extending from left side below the crown to middle of the root on the right, forming an almost V shape.

B) Root shows a circular rough surface at the tip with a vertical fracture extended up to middle of the root.

C) Tooth shows wedge-shaped root with deep vertical fracture dividing the root into two equal halves.

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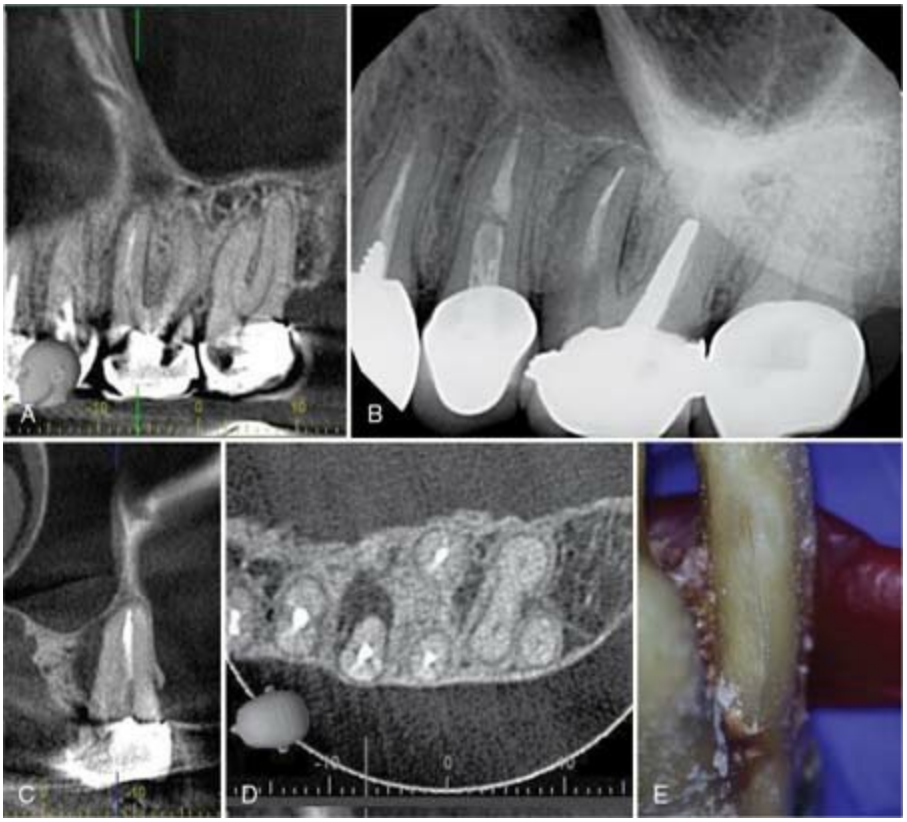

FIG. 22.9 **A** to **E,** Vertical root fractures observed with periapical radiograph, cone-beam computed tomography and clinically after extraction.

Set of four radiographs, A through D, shows different views of tooth with vertical root fracture. E) At the lower right corner, the root has a vertical cut in extracted tooth.

Source: (Courtesy Dr. R. Ganik.)

It is commonly believed that VRFs begin as microcracks in the area of the root canal surface of the radicular dentin and gradually propagate outward until the full thickness of the radicular dentin is fractured.[5,10,21,57,88] Other studies[14,17,51,77,92] have shown that microcracks can also be initiated at the outer surface of the root and propagate inward. Therefore, the correlation between microcracks in the radicular dentin and the formation of VRFs should be further investigated.

Early manifestation.

In the early stages of a VRF, there may be pain or discomfort on the affected side of the tooth. In particular, the tooth may feel uncomfortable and sensitive

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upon chewing, although this pain is often of a dull nature, as opposed to the sharp pain typical of a cracked cusp or tooth with a vital pulp. As the fracture and subsequent infection progresses, swelling often occurs, and a sinus tract may be present. Noteworthy is that the location of a sinus tract associated with a VRF is more coronal than a sinus tract that is typically associated with a case of a chronic apical abscess (Fig. 22.10).[80,82] These signs and symptoms are frequently similar to those encountered from nonhealing root canal treatment.[58,87] In the early stages, radiographic findings are unlikely because (1) the root canal filling may obstruct the detection of the fracture (Fig. 22.11, *A*), and (2) the bone destruction (which still has limited mesiodistal dimensions) may be obstructed by the superimposed root structure (Fig. 22.12).

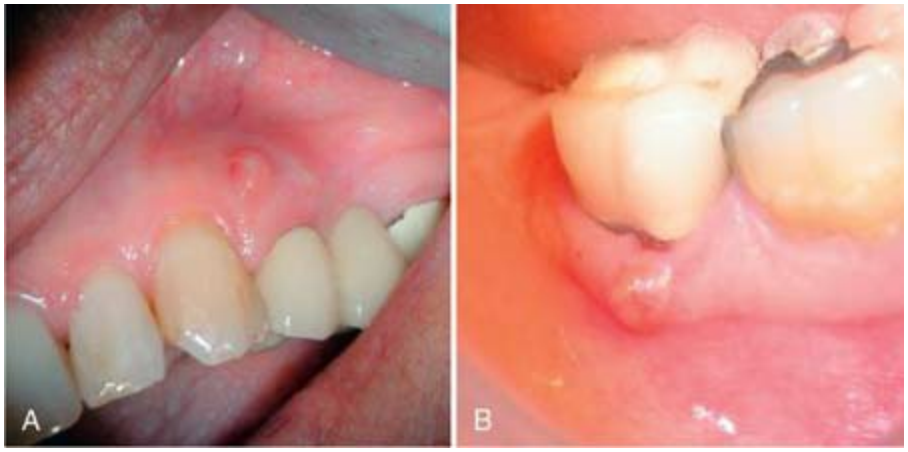

FIG. 22.10 Coronally located sinus tracts. **A,** A draining sinus tract at a coronal location originating from a buccal vertical root fracture (VRF) in the first left maxillary premolar. **B,** A draining sinus tract at the gingival margin of a right first mandibular molar with a buccal VRF in the mesial root.

A) Close-up view of upper jaw shows a lesion below the gum line of first premolar.

B) Close-up view shows tooth with black gum lining. The fluid from lesion has been drained out.

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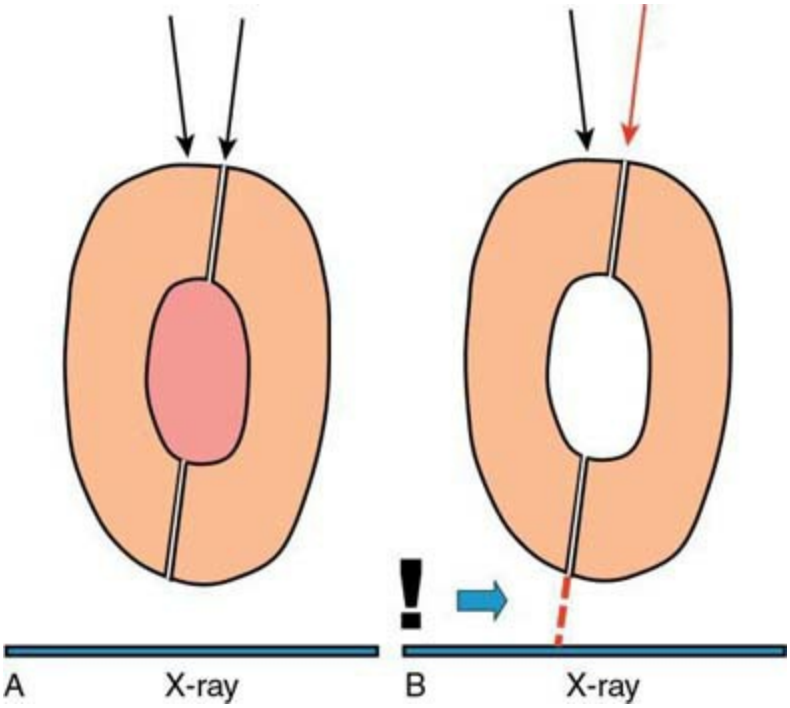

FIG. 22.11 Radiographic examination of filled versus empty canals. **A,** The buccolingual projection of a filled root will fail to detect a vertical root fracture (VRF) at an early stage. **B,** The removal of the root filling and use of radiography at different mesiodistal angulations may reveal the VRF.

Two diagrams, A and B, at the top depict filled and empty root canal, respectively. In diagram B, the X-ray passes in the root canal.

Next, Set of five images, A through E, depicts VRF.

A) Early stage shows outgrowth from root passing the cortical bone margin.

B) Radiograph shows filled root canal with porous tissue in surrounding.

C) Close-up view shows extended root canal forming a cavity in the cortical bone.

D) Late stage shows root canal penetrates properly in deep cortical bone.

E) Radiograph shows radiolucent patches around the root. The root canal has a radiolucent patch below the gum line.

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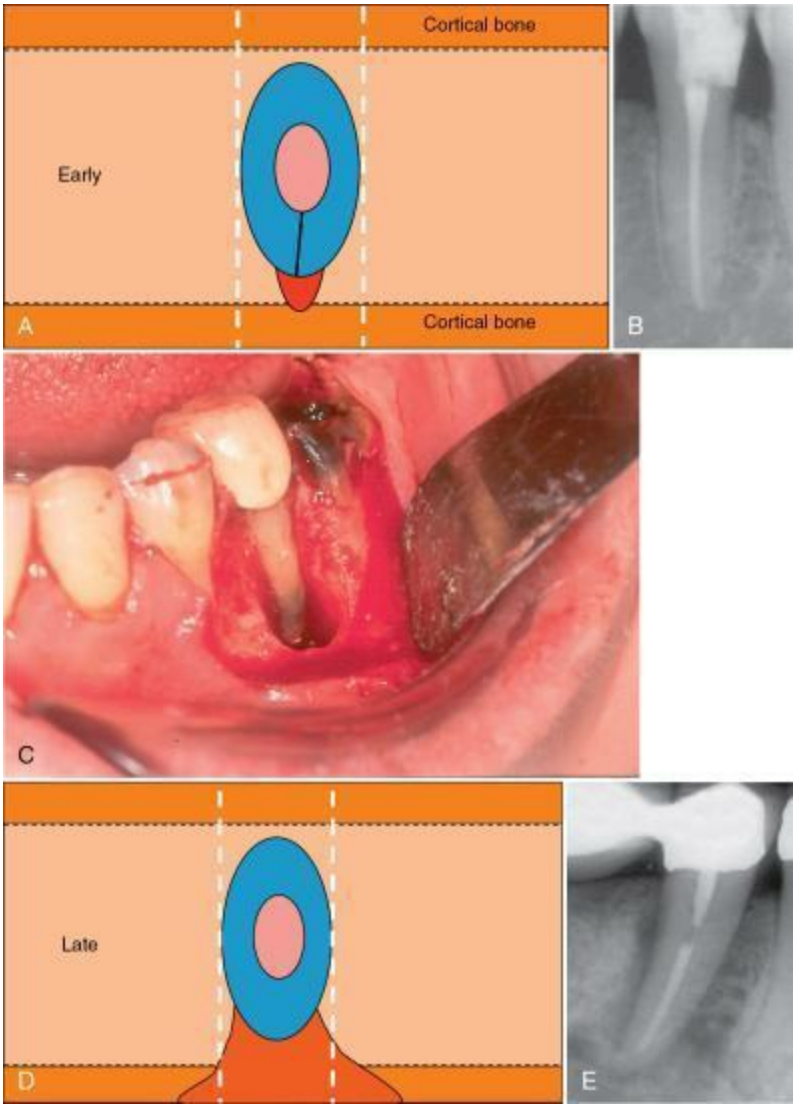

FIG. 22.12 Early versus late radiographic presentation of a vertical root fracture–associated bone defect. At an early stage, a bone defect *(red)* is not likely to be detected in a periapical radiograph, as the root will overlap with the defect **(A** and **B).** At later stages, when major damage has occurred to the cortical plate **(C),** the bone defect may be large enough to extend beyond the silhouette of the root **(C** and **D)** and appear as a radiolucent defect along the root **(E).**

Four radiographs are marked A through D.

A) Second mandibular premolar shows filled root canal and radiolucency adjacent to the root extending from gum line to the root tip.

B) The tissue around the root has very less density.

C) Small radiolucent patch near the root tip of left premolar.

D) Left molar has two radiolucent patches along the one root canal extending

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from the gum line to mid-root.

Source: (Surgical image courtesy Dr. Devora Schwartz-Arad, Ramat-Hasharon, Israel.)

Often in a case of a VRF, there is the presence of a deep, narrow, and isolated periodontal pocket. This presentation may be inconsistent with the surrounding periodontal status.[80,82,86] This specific type of periodontal defect occurs secondary to the bony dehiscence caused by the VRF and is substantially different in its presentation from the pockets caused by advanced periodontitis (discussed later).

Late manifestation.

A longstanding VRF is easier to detect. The major destruction of the alveolar bone adjacent to the root has already occurred, allowing the VRF to be more likely revealed in a periapical radiograph (see Fig. 22.6, *B*). One of the most typical radiographic signs is a *J-shaped* or *halo radiolucency*, which is a confluence of periapical and periradicular bone loss (i.e., bone loss apically combining with the bone loss on the side of the root, extending coronally) (Fig. 22.13).[83,84] In addition, the pocket now approximating the fracture, which was initially tight and narrow, may become wider and easier to detect. In longstanding cases in which the bone destruction is extensive, the VRF may result in a split root whereby the segments of the root separate, resulting in radiographic evidence clearly revealing an objective split root (Fig. 22.14).

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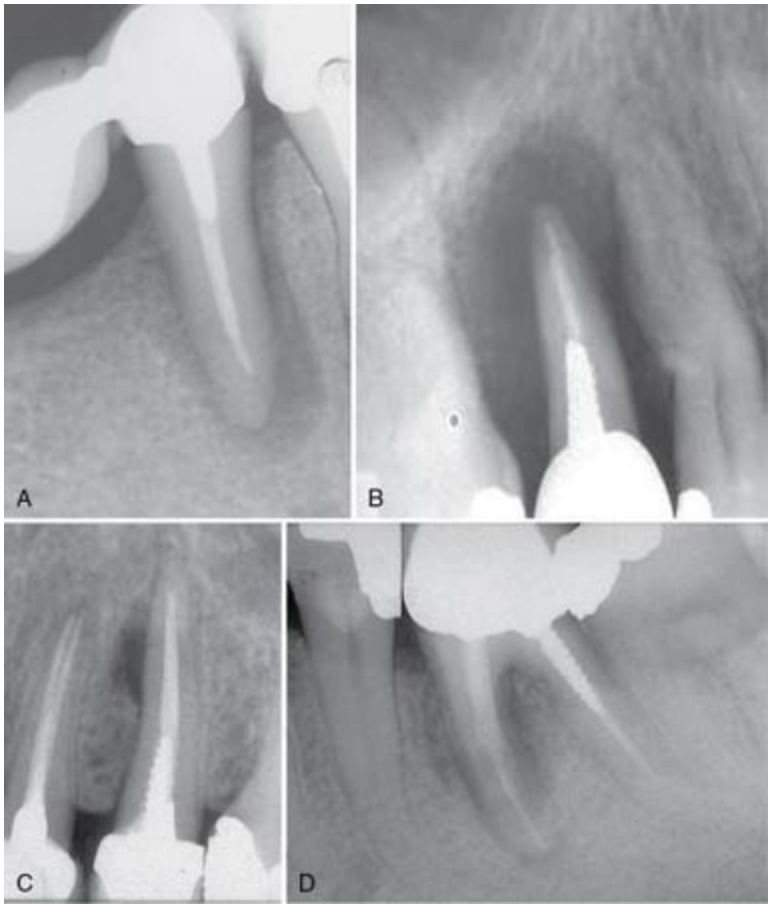

FIG. 22.13 Radiographic presentations of longstanding vertical root fractures (VRFs). **A,** J-shaped "halo" associated with a VRF in a second right mandibular premolar. **B,** Extensive bone damage associated with a complete VRF in a second right maxillary premolar. **C,** Limited bone damage associated with a midroot VRF in a second left maxillary premolar. **D,** Bone damage associated with a VRF in the mesial root of a first left mandibular molar. All such presentations are typical of longstanding VRFs.

Four radiographs are marked A through D.

A) Second mandibular premolar shows filled root canal and radiolucency adjacent to the root extending from gum line to the root tip.

B) The tissue around the root has very less density.

C) Small radiolucent patch near the root tip of left premolar.

D) Left molar has two radiolucent patches along the one root canal extending from the gum line to mid-root.

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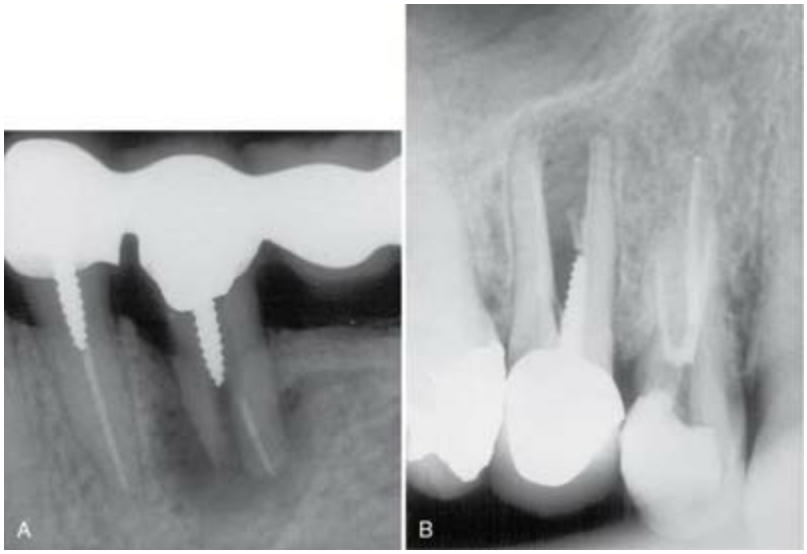

FIG. 22.14 Radiographs of longstanding vertical root fractures. No special diagnostic skills are needed to diagnose such cases.

A) Radiograph shows left tooth with a large radiolucent patch at the root tip and half-filled root canal.

B) Radiograph shows V-shaped radiolucent region in-between the root canal. The adjacent tooth has filled root canals and fractures.

Diagnosis

Importance of early diagnosis.

Accurate and timely diagnosis is crucial in VRF cases, allowing the extraction of the tooth or root before extensive damage to the alveolar bone occurs. Early diagnosis is particularly important when implants are a potential part of the future restorative process; when an extraction is performed at an early stage, the uncomplicated placement of an implant is more likely. When the tooth is extracted after extensive damage has already occurred, bone regeneration procedures may be required,[43] adding additional cost and time to the restoration process.

The American Association of Endodontists stated in 2008[5] that a sinus tract and a narrow, isolated periodontal probing defect associated with a tooth that has undergone a root canal treatment, with or without post placement, can be considered pathognomonic for the presence of a VRF.

However, the combination of the following two factors makes the early

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diagnosis difficult: (1) many of the clinical symptoms associated with VRFs mimic apical periodontitis or periodontal disease, and (2) the narrow and tight pocket associated with early stages of VRF is difficult to detect using rigid probes (see the Vertical Root Fracture Pockets section), especially when the pocket is interproximal. Consequently, a delay in the accurate diagnosis —or a misdiagnosis of a VRF—may often occur.

Misdiagnosis of vertical root fractures.

Certain cases of longstanding VRFs are so discernible that no dentist should miss the diagnosis (see Fig. 22.14). Nevertheless, two retrospective case series, one by Fuss et al.[33] and another by Chan et al.,[22] reported that general practitioners often misdiagnose VRFs. The teeth that were extracted in these studies had often been diagnosed as endodontic failures or refractive periodontal pockets, only to realize after extraction that in some of them the actual cause was a VRF.

Vertical root fracture pockets.

The pockets that are typical of the early stages of VRFs differ substantially from the deep pockets associated with advanced periodontal disease. The deep pockets associated with periodontal disease develop as a result of the bacterial biofilm that initially accumulates at the cervical areas of the tooth and the destructive host response to these bacteria.[39] Therefore, deep periodontal pockets are typically wider coronally and easier to probe. This pocket anatomy allows an easy insertion of a rigid periodontal probe (Fig. 22.15). These types of periodontal pockets typically present with the deeper part of the pocket at the mesial or distal aspects of the tooth. In addition, periodontal disease often affects groups of teeth rather than an isolated location of a single tooth.

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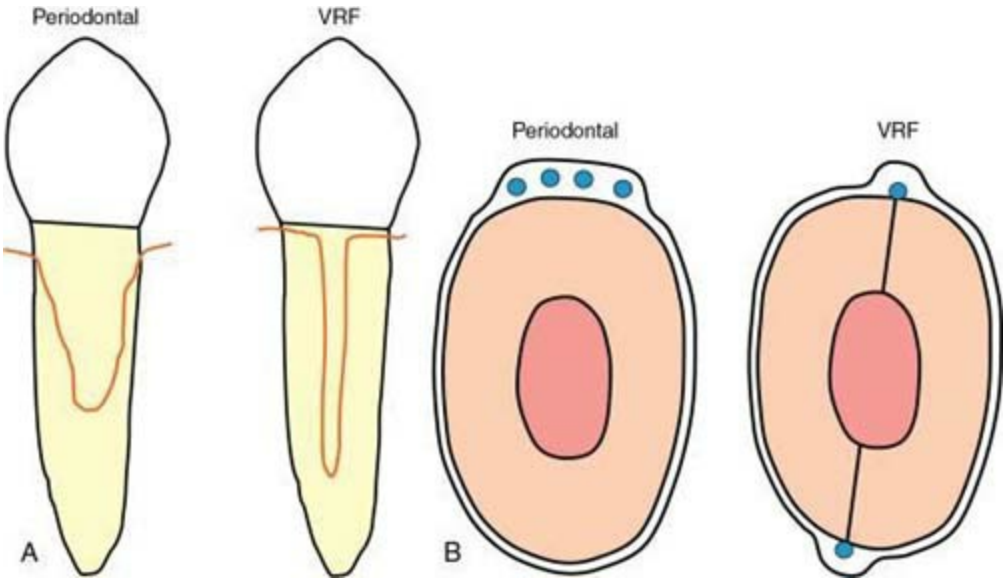

FIG. 22.15 Vertical root fracture *(VRF)* pocket. **A,** Periodontal pockets *(left)* are wide coronally, whereas VRF pockets *(right)* are narrow and deep. **B,** Periodontal pockets *(left)* are loose and allow probing at various sites, whereas VRF pockets *(right)* are narrow and tight. If not checked carefully at every millimeter of the sulcus, an early VRF pocket can easily be missed. Note that periodontal pockets appear more commonly in the proximal sides of the root, whereas VRF pockets are more common on the buccal or lingual sides.

A) Tooth has a nearly bell-shaped periodontal pocket extending below the neck region to the midway of root. Another tooth has a narrow U-shaped VRF pocket extending at the base of crown to three-fourth length of root.

B) Tooth with periodontal pocket has four spheres at the top. Another tooth with VRF pocket has two spheres, one at the top and the other at the base.

The pockets associated with VRFs develop due to bacterial penetration into the fracture, triggering a destructive host response that occurs in the periodontal ligament along the entire length of the fracture. These bacteria may leak from an infected root canal[88]; however, when the VRF extends to the cervically exposed root, the microbes in the fracture may also originate from the oral cavity. In the early stages, the periodontal ligament is affected and destroyed along the longitudinal opening of the fracture, initially with a limited resorption to the adjacent bone. This permits the penetration of a periodontal probe. The pocket associated with a VRF is typically isolated and present only in a limited area adjacent to the affected tooth. This pocket is often located at the buccal or lingual convexity of the tooth. In the early

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stages, the pocket is deep but has a narrow coronal opening (see Fig. 22.15). The insertion of a probe first requires the detection of the coronal opening; often, light pressure is necessary for the insertion of the probe. Because the pocket is narrow, probe insertion may result in the blanching of the surrounding tissue (Fig. 22.16). This is specially the case when a more flexible plastic probe is used, as its coronal part is thicker than an equivalent metal probe (Fig. 22.17; also see Fig. 22.16). Therefore, the pocket associated with the early stages of a VRF is quite different from a typical periodontal pocket. This difference has been widely recognized, and terms such as *osseous defect*[28] and *probing defect*[5] have been used to emphasize the point. Nevertheless, these pockets do possess enough unique features to justify them being specifically termed a *VRF pocket*.

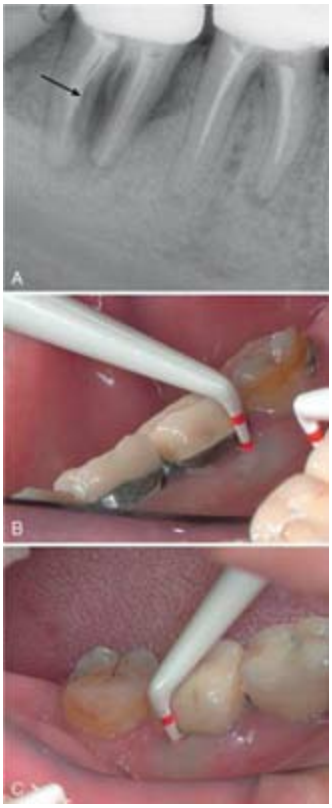

FIG. 22.16 Tight vertical root fracture (VRF) pockets. **A,** The distal root of a second right mandibular molar with a VRF. VRF pockets were found on both the lingual **(B)** and the buccal **(C)** sides. VRF pockets are tight, so inserting a probe into these pockets causes pressure blanching of the surrounding tissues.

A) Radiograph shows two teeth. Second right molar has filled radiopaque root

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canals with a wavy line extended from below the neck region to above the root tip. The tissue in-between both root canals has less-density.

B) Close-up view of teeth shows probe pressing the white gum line of tooth. The bases of crown are covered with silver strips.

C) Close-up view shows the probe inserting into the gum close to the molar.

Rigid metal periodontal probes may be ineffective in probing VRF pockets in the early stages of a VRF. Given that the pocket is deep, narrow, and tight, the bulge and obstruction of the tooth's crown may prevent the insertion of a metal probe into the pocket (see Fig. 22.17). A flexible probe should be used instead, such as a probe available from Premier Dental Products (Plymouth Meeting, PA) (see Fig. 22.17) or a similar device. This type of flexible probe should be included on every endodontic examination tray and is an essential tool when checking for potential VRF pockets.

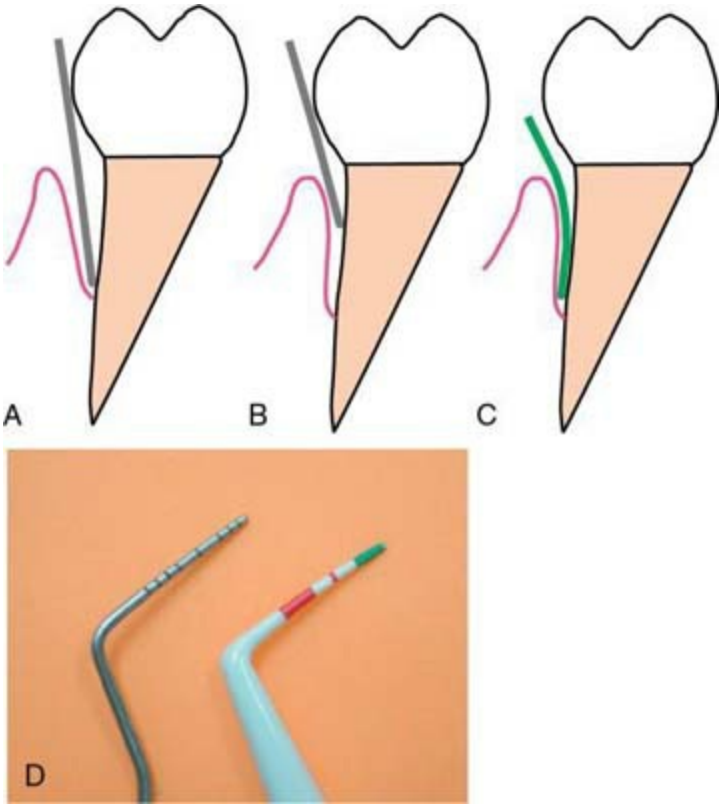

FIG. 22.17 Rigid versus flexible probes. **A,** In a loose periodontal pocket, a rigid metal probe can easily reach the depth of the pocket. **B,** In a tight, early-stage vertical root fracture (VRF) pocket, a rigid probe may be of limited value, as the bulge of the crown often prevents the

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insertion of the probe into the tight, deep pocket. **C,** A flexible probe **(D)** is more likely to detect VRF pockets at an early stage.

A) Probe reaches the midway of the root into the pocket.

B) Probe reaches the one-fourth part of the root at the top of pocket.

C) Probe is curved and reaches deep into the pocket.

D) Two L-shaped probes are shown.

Tamse et al.[82] reported the presence of a typical VRF pocket in 67% of the VRF cases reported. However, because the early detection of such pockets is technique sensitive and because traditional metal probes were used in the aforementioned study, the incidence of these pockets may in fact be higher than reported. When a typical VRF pocket is present on the convex surfaces of the root, on the buccal or lingual aspects, it is likely that the root has a VRF. In contrast, when such a pocket is located at the furcation of a molar, the pocket may indicate either a VRF or a sinus tract from an apical abscess that drained at a point of least resistance at the furcation area. In cases with a furcation pocket, when a VRF diagnosis cannot be conclusively determined, a positive healing response to the elimination of infection (by periodontal therapy or initiating root canal treatment if necessary) may differentiate between these two types of pathoses.

Coronally located sinus tract.

Sinus tracts that originate from a chronic apical abscess are typically detected at the site of least bone resistance, against the apical part of the root or in the area of the junction of the attached gingiva and the oral mucosa. Sinus tracts that are associated with a VRF pocket are often found in a more coronal position, as the source is not from a periapical lesion (see Fig. 22.10).[81]

In four clinical retrospective case series, coronally located sinus tracts were found in 13% to 35% of these cases.[59,80,82,86] As in the case of a VRF pocket, if the sinus tract is located at the furcation of a molar, this observation does not necessarily indicate a VRF, as periapical abscesses from re-infected root canal treatment can also drain in this coronal location.

Radiographic features.

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A definitive VRF diagnosis can sometimes be made based on the radiographic appearance of a thin radiolucent line extending longitudinally down the root.[73] Such lines, however, are difficult to detect and are commonly not seen in routine periapical radiographs because either the root canal filling has “masked” the fracture line or the angulation of the x-ray beam is not optimal for discerning the fracture (Fig. 22.18; also see Fig. 22.11). Rud and Omnell[73] claimed that it was possible to observe fracture lines in 35.7% of cases, but many of these cases were not true VRF instances. In clinical practice, it is still rare to observe a VRF on a radiograph, especially when only a single periapical radiograph is exposed. Such an observation requires the x-ray beam to align with the plane of the fracture as well as the fracture line not being superimposed over the radiopaque root filling. Therefore, two or three periapical radiographs should be exposed from different horizontal angulations when a fracture is suspected (Fig. 22.19; also see Fig. 22.18).[81] In most VRF cases, the clinician must make subjective *interpretations* or *predictions* based on the various patterns of periradicular bone destruction which, unfortunately, are also shared by other periodontal and endodontic-like lesions.[59,76,89]

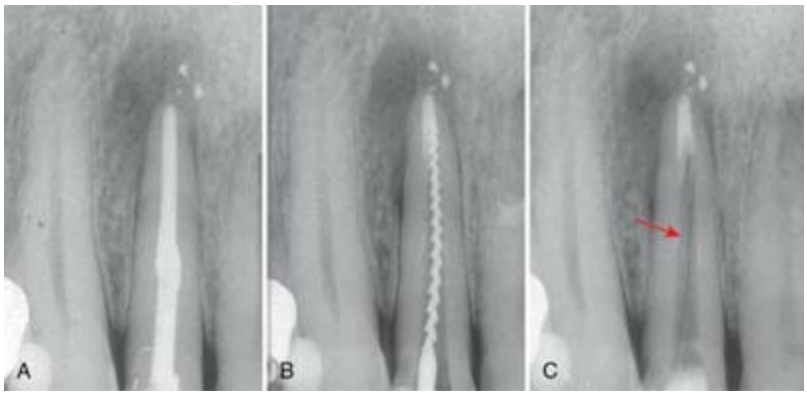

FIG. 22.18 Radiography of an empty canal: a clinical case. The right maxillary lateral incisor underwent a root canal treatment a few years ago. The patient complained about occasional pain on the palatal side. The tooth was sensitive to percussion and palpation on the palatal side. Radiography revealed a periapical radiolucency **(A).** An isolated, deep, and narrow pocket was found on the palatal side of the root. Both the patient and the referring dentist were reluctant to extract the tooth, assuming that the pocket could potentially be a sinus tract, and decided on retreatment. A radiograph taken with a file during the process **(B)** could have missed essential information that was obscured by the file,

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which was an evident vertical root fracture **(C).**

Three radiographs are marked A through C.

A) Lateral incisor has a filled root canal with a bulge at the center. The tissue near root tip is less dense.

B) Lateral incisor has zigzag pattern of filling in radiolucent root canal. The tissue near root tip is less dense.

C) Lateral incisor has a slant vertical line in radiolucent root canal extended from below the neck to three-fourth of the root.

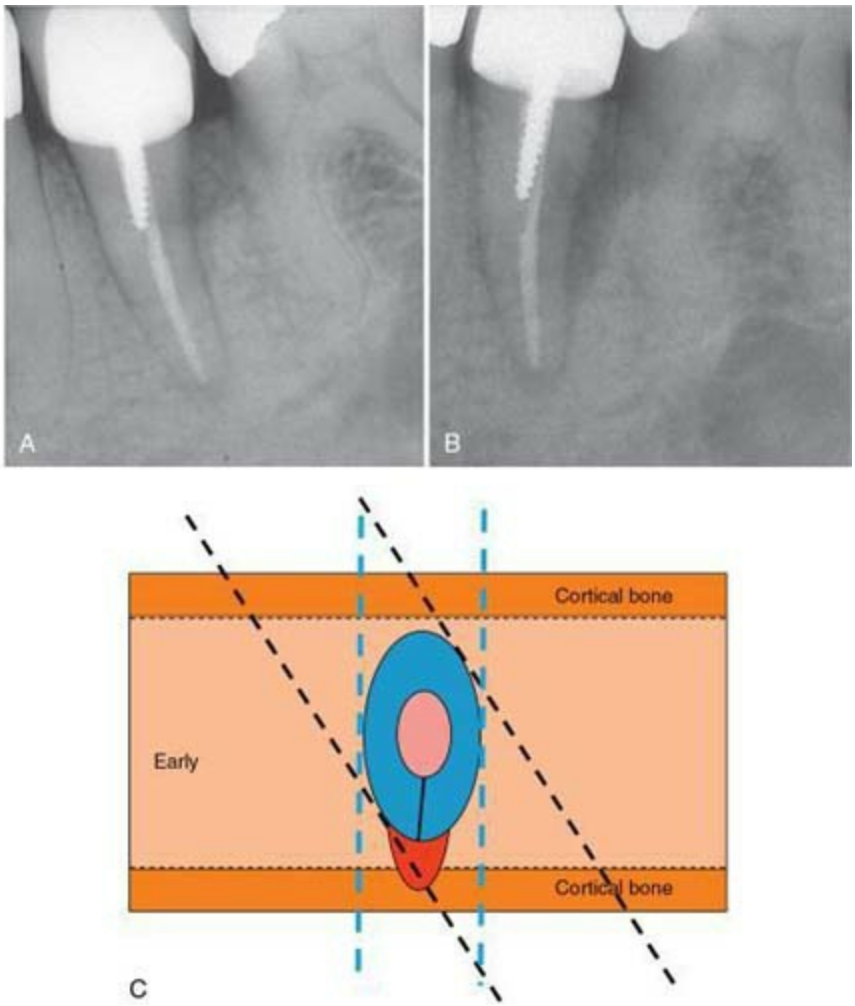

FIG. 22.19 Diagonal radiographs for detection of vertical root fracture (VRF). **A,** Orthoradial radiograph: very limited bone loss is seen. **B,** A different horizontal angulation reveals a radiolucent lesion along the root. **C,** Schematic presentation. Although a radiograph in an

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orthoradial direction *(blue lines)* cannot pick up the radiolucency along the root, diagonal angulation *(black lines)* may do so, as in **B.**

A) Radiograph shows the central tooth with filled root canal. The filling is comparatively less radiopaque from mid to tip of the root.

B) Radiograph shows the oblique space in the mid of the root canal. The tissue along the one side of root has less density.

C) Early detection shows the U-shaped outgrowth from tooth penetrates in the cortical bone. The tooth is placed inside two vertical parallel blue lines and two black diagonal lines.

In the early stages of a VRF, no radiolucent bone lesions may be observed[20] (see Fig. 22.12), which may be the reason why VRFs often remain undetected, delaying diagnosis and treatment. Rud and Omnell[73] correlated the direction of the fracture, the degree of bone destruction, and the radiographic appearance, and emphasized that the extent of bone destruction around a fractured root depends on the location of the root fracture and the time elapsed since the inception of the fracture. The significance of time was confirmed by Meister et al.,[59] who demonstrated that immediate radiographic detection is difficult due to the time required for bone resorption to occur or for the fractured segments to separate and be radiographically visible. In a study of the patterns of bone resorption in 110 VRF cases, Lustig et al.[58] found that in 72% of patients with either chronic signs and symptoms (i.e., pertaining to a sinus tract, osseous defect, or mobility) or acute exacerbations, there was greater bone loss recorded compared to patients for whom a VRF diagnosis was made at an early stage.[58]

Despite the difficulty of diagnosing *early stage* VRFs in endodontically treated teeth, there are often several radiographic signs associated with *later stages* that are strong indicators of VRF.

The J-shaped or halo radiographic appearance of bone loss, a combination of periapical and periradicular radiolucencies, was associated with a high probability of a VRF in a double-blind radiographic study involving 102 endodontically treated maxillary premolars (see Fig. 22.13).[83] An angular resorption of the crestal bone along the root on one or both sides, without the involvement of the periapical area, mimicking a “periodontal radiolucency” (see Fig. 22.13), was found in 14% of the cases. Tamse et al.[84] also reported

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the radiographic appearance of “halo” and “periodontal” radiolucencies in vertically fractured mesial roots of mandibular molars (37% and 29%, respectively). In that study, the use of these two variables, combined with bifurcation involvement (63%) and the presence of an amalgam dowel (67%), predicted fracture in 78% of the cases. Others have reported similar findings.[24,63] Despite the different sample sizes, study designs, and objectives, the most common radiographic feature in these studies was a lateral radiolucency appearing longitudinally along the root and a halo appearance.

Osseous radiolucency adjacent to the root.

The type of periradicular radiolucency associated with a VRF is not and should not be interpreted as a thickening of the PDL. Instead, it represents a substantial destruction of the cortical plate of the alveolar bone (see Fig. 22.12).[58] In the case of a VRF in the bucco-lingual plane, often the bone resorption is limited at early stages, and any associated radiolucency may be obscured by the superimposition of the root (see Fig. 22.12). As the bone loss increases, the radiolucency becomes greater than the dimensions of the root, allowing it to be detected more clearly in the previously mentioned manner (see Fig. 22.12). As the VRF progresses to an intermediate stage, radiographs taken at different horizontal angulations may detect bone resorption (see Fig. 22.19), whereas a single radiograph may not (see Figs. 22.12 and 22.19). This radiographic feature should be differentiated from a split tooth, in which the fracture plane is typically mesiodistal, with the bone resorption occurring in the earlier stages on the mesial or distal aspects of the root.

Radiograph of empty canal.

As mentioned previously, the direct clinical detection of an early stage VRF from a periapical radiograph is unlikely, especially when there has been endodontic treatment. Because most VRFs are in the bucco-lingual plane, the radiopaque obturation often obstructs the view of the hairline radiolucency of the fracture (see Fig. 22.11). When a VRF is suspected, one may initiate root canal retreatment, removing the root obturation, and exposing radiographs at two or three different horizontal angulations. The detection of a hairline radiolucency may provide a more definitive diagnosis of a VRF (see Fig.

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22.18).[81]

Cone-beam computed tomography in vertical root fracture diagnosis.

Modern cone-beam computed tomography (CBCT) has a much smaller radiation dose compared to traditional medical spiral CT imaging, thus rendering CBCT a reasonable diagnostic tool for use in selected endodontic cases.[6,31,32,66]

One of the unique features of CBCT is its ability to study the suspected tooth and associated bone in an axial plane (i.e., the horizontal sectioning of the root). Axial views may provide detailed information regarding the cross-sectional appearance of the tooth and its surrounding bone (Fig. 22.20). Considering the resolution of the current CBCT devices, the width of an unseparated fracture may be too small to be detectable (see Fig. 22.20) (SEDENTEXCT guidelines[31]). Traditional planar, periapical radiographs are also of limited value for the *early* detection of VRFs. More specifically, bone damage or separation of the fragments is only radiographically evident at a relatively late stage. Several studies suggested that the detection of early-stage VRFs by a CBCT scan set to an axial view may be possible.[42,43,60] Yet such detection may greatly depend on the resolution of the machine (i.e., the voxel size). At a voxel size of 0.3 mm, the detection of early, unseparated VRFs is not reliable; however, when smaller voxel sizes were used in these in vitro studies, the reliability greatly increased.[42,43,60] Although the detection level of a fracture is thought to be the width of twice the voxel size of the CBCT imaging, there is presently no literature available to support this theory. Therefore, given that the smallest voxel size currently available for a CBCT device is about 0.075 mm, CBCT imaging would not be able to visualize a root fracture unless the fracture width was greater than 0.15 mm. It should also be noted that the intracanal presence of gutta-percha or a metal post often causes artifacts that make it extremely difficult to discern a VRF.[60]

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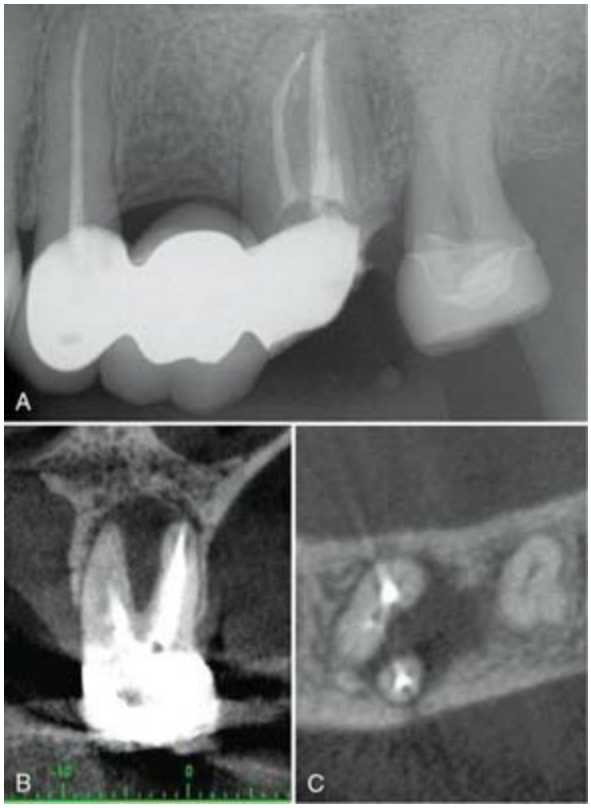

FIG. 22.20 **A,** Periapical radiograph of maxillary left first molar. **B,** Cone beam computed tomography (CBCT) coronal view revealing bone loss between the buccal and palatal roots, suggestive of a vertical root fracture. **C,** CBCT axial view revealing bone loss between the buccal and palatal roots, suggestive of a vertical root fracture.

Set of four radiographs, marked A through D, shows periapical, front, and transverse views of vertical root fracture in left first molar of upper jaw.

Source: (Courtesy Dr. R. Granik.)

Although the width of early VRFs may still be below the detection level of many CBCT devices, the *early destruction of the bone* along the suspected fracture may be visible in the cancellous bone (i.e., with an axial view) at relatively early stages, whereas this early bone destruction would not be detectable in traditional two-dimensional periapical radiographs; such bone resorption may help to establish a VRF diagnosis (see Fig. 22.20).

With likely increased resolution in the near future, CBCT may become a more important diagnostic tool for the detection of VRFs. For the present, neither the most updated Joint Position Statement of the American Association of Endodontists and American Academy of Oral and

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Maxillofacial Radiology (2010), nor the European Society of Endodontology position statement on the use of CBCT in endodontics (2014) recommend the use of CBCT for a definitive diagnosis of VRF.[6,32]

Improvements of CBCT imaging—such as achieving a better signal-to-noise ratio, obtaining a smaller voxel size, and by applying advanced algorithms to segment fracture lines—may promise the potential to enhance the ability to detect early-stage VRFs in the future.

Exploratory surgery.

When clinical and radiographic evaluations are equivocal in detecting a suspected VRF, exploratory surgery may be indicated. In the presence of a bony lesion, especially a bony dehiscence, the raising of a full-thickness flap and removal of the granulation tissue may help to facilitate the visualization of a VRF (Fig. 22.21; also see Fig. 22.12).[81] The bone resorption pattern associated with a VRF is most often seen as a bony dehiscence, with the greater bone destruction being present on the buccal cortical plate located over the offending root. In a small percentage of the cases, a fenestration can be seen.[58] Furthermore, it has been shown that the longer a VRF-related infection persists, the greater the resulting periradicular bone destruction.[58]

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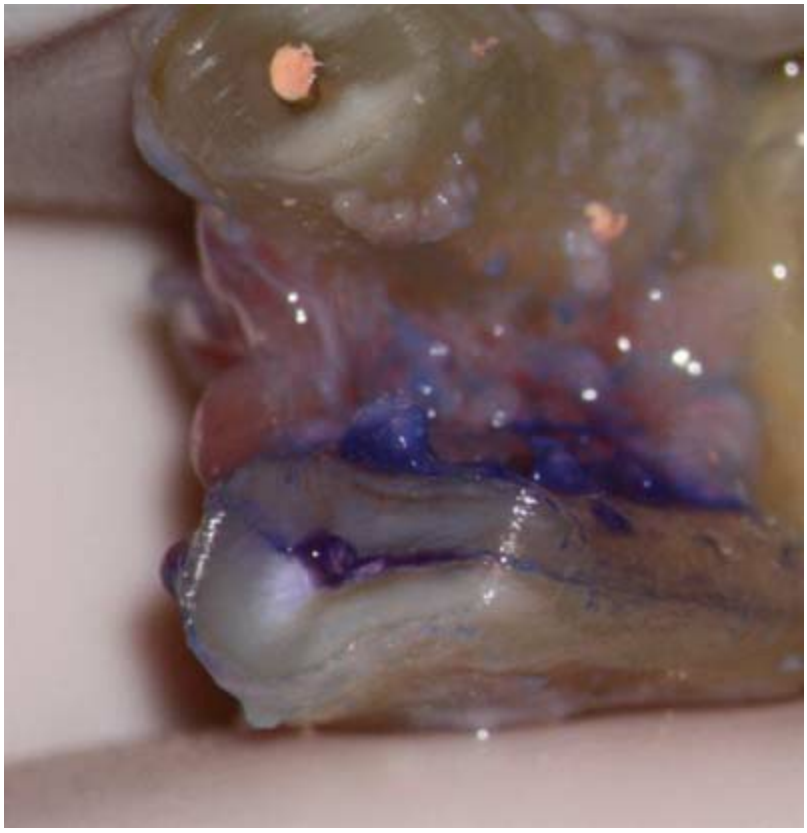

FIG. 22.21 Clinical image of a vertical root fracture, observed using applied dye.

Specimen of vertical root fracture has an ingrown and soft inflamed tissue stained in blue.

Source: (Courtesy Dr. S. Floratos, Athens, Greece.)

Etiology

VRFs may arise from a series of factors, some of which are natural whereas others are iatrogenic, arising from dental procedures such as endodontic treatment and restorative procedures. The most common dental procedure contributing to VRFs is endodontic treatment.[11]

Most VRFs occur in endodontically treated teeth.[11,24] VRFs usually do not occur during the actual obturation of the root canal, but rather long after the procedure has been completed.[82]

The etiology of VRFs is multifactorial,[34,80] arising in the presence of one or more predisposing factors, the repeated functional or parafunctional occlusal loads which may eventually lead, over months or even years, to the

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development of a VRF. Predisposing factors may include natural influences, such as the anatomy of the root, or iatrogenic causes, such as the excessive forces during root canal instrumentation, excessive tooth structure removal, or excessive obturation pressure.

Natural predisposing factors

Shape of root cross section.

One of the common anatomic features shared by teeth that typically develop VRFs is an oval cross section of the root, with a bucco-lingual diameter being larger than the mesiodistal diameter.[37,41] These teeth include the maxillary and mandibular premolars, the mesial roots of the mandibular molars, and the mandibular incisors (Fig. 22.22, *A* and *B*). Such anatomy is easily observed in the axial plane of a CBCT scan (see Fig. 22.22, *A* and *B*). The fracture in these teeth typically starts in the bucco-lingual plane, specifically at the highest convexity of the oval root (see Fig. 22.22, *A* and *B*).[21,81] This conclusion, derived from large case series, is also supported by finite element analysis.[65] Such analysis clearly demonstrated strain concentration on the inner side of the remaining dentin wall at the highest convexity point (i.e., the buccal and lingual sides of the oval roots) (Fig. 22.23).[55,56]

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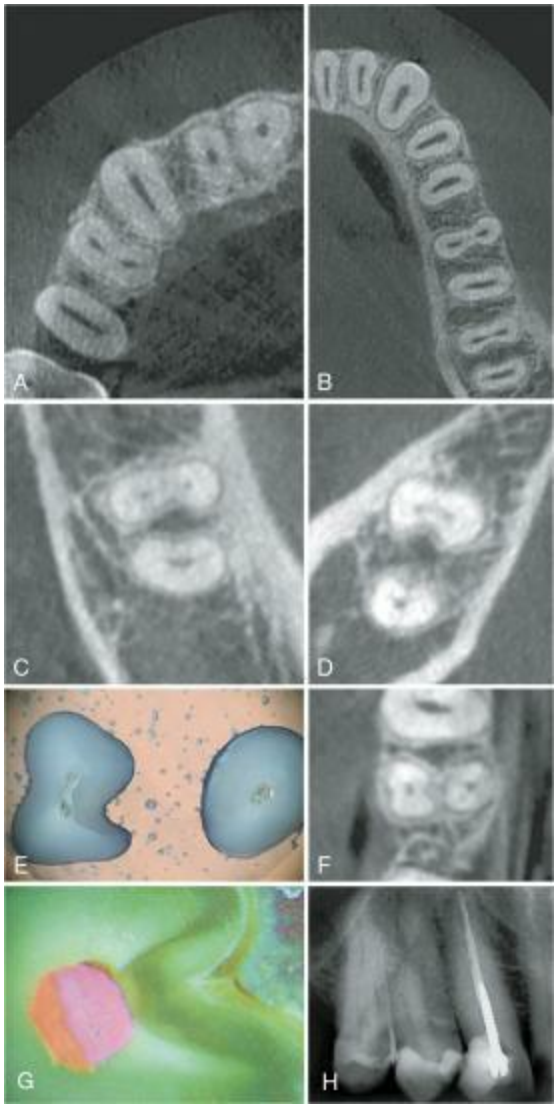

FIG. 22.22 Anatomic predisposing factors. **A,** An axial view of cone-beam computed tomography (CBCT) scan of a maxilla, revealing oval canals in the maxillary canine and second premolar. **B,** An axial view of a mandible, revealing oval canals in incisors, canine, premolars, and distal roots of the mandibular molars. Oval anatomy combined with endodontic treatment has been associated with a higher incidence of vertical root fractures (VRFs). **C** and **D,** Concavities on the distal aspect of the mesial roots of mandibular molar may establish a "danger zone" in which excessive instrumentation, combined with straightening of the canal, may result in a thinner dentin wall that may allow strain concentration. **E** and **F,** Concavities in the palatal side of the buccal root of maxillary first premolar (**E**, sections; **F**, axial view from a CBCT). These depressions may also represent a potential danger zone. Neither the concavity in **C** and **D** nor the concavity in **E** and **F** would be evident in a planar periapical radiograph. It should be noted that CBCT scans should not be used for routine screening but should be limited to the indications delineated in the joint statement of the American

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Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR).[5,6] **G,** Histology of VRF. **H,** Radiograph revealing peri-radicular bone loss.

A set of six radiographs and two micrographs, marked A through H, depicts oval canals in teeth resulting in less-density bone in periodontal ligament and cementum of root.

Source: (Courtesy Dr. R. Ganik.)

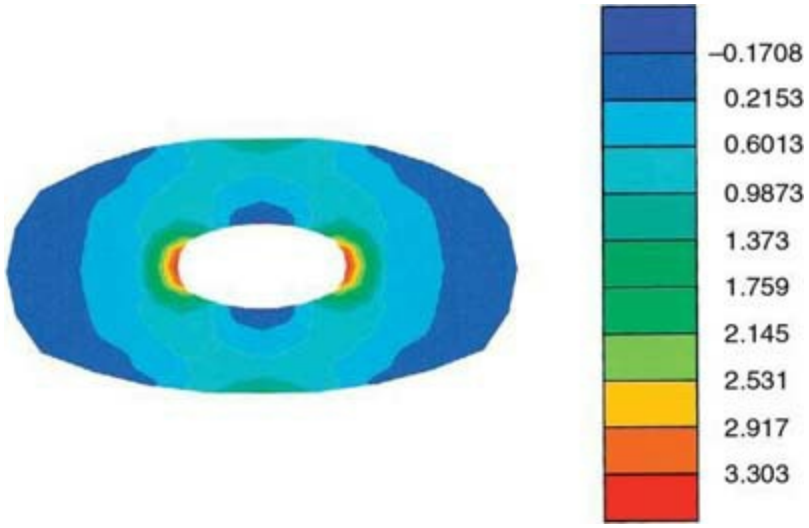

FIG. 22.23 Finite element analysis of the strain distribution in an oval root. Note the strain concentration on the inner side of the highest convexity of the remaining dentin wall. Red and orange represent areas of higher strains than blue areas.

Multicolored horizontal tooth shows oval root where left and right sides have higher strain in a range of 3.3 0 3 to 0.9 8 7 3. The top and bottom sides of the root have strain in the range of 0.2 1 5 3 to negative 0.1 7 0 8.

Source: (From Lertchirakarn V, Palamara J, Messer HH: Patterns of vertical fractures: factors affecting stress distribution in the root canal, *J Endod* 29:523, 2003.)

Occlusal factors.

Excessive occlusal loads or concentration of such loads may be another natural predisposing factor for VRFs. Load concentrations, such as those caused by occlusal prematurities in maxillary premolars, and excessive

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occlusal forces, specifically in the case of mandibular second molars, are good illustrations.[18] In combination with other natural and iatrogenic predisposing factors, excessive occlusal loads may, over time, lead to VRFs.

Preexisting microcracks.

Preexisting microcracks may be present in the radicular dentin, likely resulting from repeated forces of mastication or occlusal parafunction.[15,64] Such fractures were also recently reported by Barreto et al.,[10] who found these microcracks present in 40% of intact maxillary incisors and canines.

Iatrogenic predisposing factors

Root canal treatment.

VRFs mostly appear in endodontically treated teeth[5,81]; therefore, endodontic treatment per se may be considered an iatrogenic predisposing factor. Teeth were once thought to be more susceptible to fracturing after endodontic treatment because of a decrease in hydration.[44] However, later studies found no difference in the properties of dentin, as a material, after endodontic procedures.[47,76]

Although the physical characteristics of dentin, as a material, may not be compromised by endodontic treatment, the radicular dentin, as a structure, may be compromised by the accumulative or combined effect of several natural or iatrogenic factors associated with the endodontic treatment and the restoration of endodontically treated teeth. This may be the reason for the often-reported association of VRF with endodontically treated teeth.

It is incumbent for the clinician to recognize this effect and take efforts to minimize any steps that may contribute to the development of a root fracture during endodontic treatment.

Excessive root canal preparation.

Excessive root canal preparation may be a predisposing factor for VRF development.[90] In one study, cracks detected by transillumination were more frequent when the same teeth were subjected to a gradually increasing endodontic canal preparation.[90] To reduce the risk of VRFs, less invasive methods may be considered, such as minimally invasive endodontic

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instrumentation (see also Chapter 8).[61,67]

Microcracks caused by rotary instrumentation.

Shemesh et al.[77] and others[2,10,14,17,45,78,92] observed that root canal preparation using nickel-titanium rotary and reciprocating files often results in microcracks in the remaining radicular dentin (Fig. 22.24). This finding, which was originally noted for single rooted teeth, has been further supported by Yoldas et al.,[92] who studied microcrack formation from rotary files in the mesial roots of mandibular molars. Each of the rotary file systems examined in this study caused frequent microcracks in the dentin, whereas both hand instrumentation with files and the self-adjusting file (see Chapter 8) did not cause such cracks.

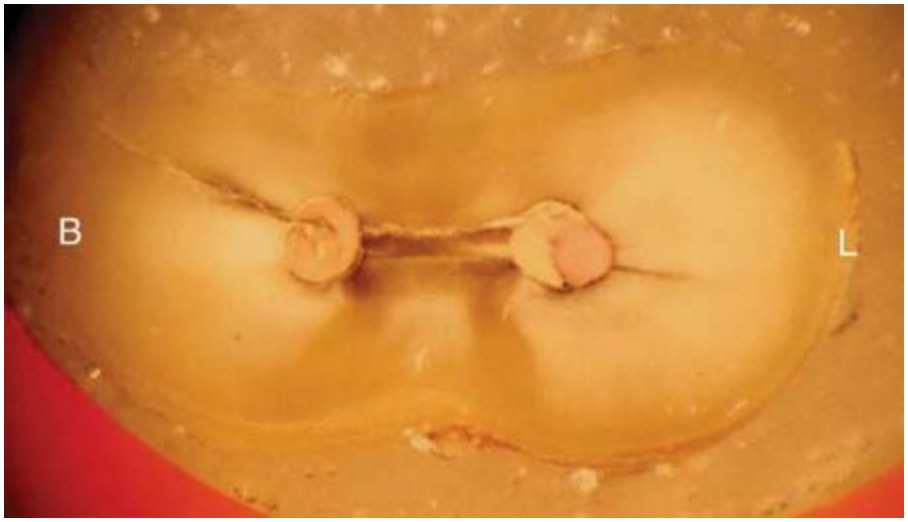

FIG. 22.24 Typical bucco-lingual fracture that includes the isthmus in a maxillary premolar with two canals.

Tooth shows a deep horizontal line with two circular structures on buccal and lingual sides.

A finite element analysis by Kim et al.[51] supports and may explain these findings. These researchers and others reported that rotary files induce strain on the dentin, as measured in the surface layers of the root dentin, which likely exceeds the elasticity of the dentin, causing subsequent microcracks.[2,10,14,17,45,77,78,92] Additional stress, either from root obturation with lateral compaction[10,77] or by nonsurgical endodontic retreatment,[78]

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caused some of the microcracks to propagate and become through-and-through fractures that were indistinguishable from VRFs.[10,77,78]

The relationship of these various findings to the subsequent clinical creation of a VRF has yet to be confirmed. In any event, considering the basic principles of fracture mechanics, the creation of microcracks should at least be considered as a potential predisposing factor.

Uneven thickness of remaining dentin.

The instrumentation of root canals often results in uneven thickness of the remaining dentin, particularly when curved canals are straightened by instrumentation.[68] Uneven dentin thickness can also occur upon excessive instrumentation of the mesial roots of the mandibular molars or first maxillary premolars, which may exhibit a distal or mesial concavity, respectively, that is not detectable in a common planar, periapical radiograph (see Fig. 22.22, *C* and *D*). These areas, which have been referred to as "danger zones,"[30] may be characterized by a decrease in the remaining dentin thickness in which the application of internal strain may potentially lead to a fracture. The anatomic groove that is often found on the palatal side of the buccal root of maxillary bifurcated premolars is another example of such a hidden danger zone (see Fig. 22.22, *E*–H).[49]

Lingual access, which is commonly used in incisors, may also result in a thinner buccal wall in the apical area as compared with the lingual wall. This phenomenon may be especially pronounced when thick, and thus rigid, instruments are used excessively. When lateral compaction was applied ex vivo in a similar case, the strain concentration was recorded on the thinner buccal side of the apical part of the tooth.

The use of flexible nickel-titanium files and minimally invasive instrumentation may reduce such risks (see also Chapter 8).

Methods of obturation.

Certain obturation techniques, such as lateral compaction, involve the application of internal pressure with a spreader, which may cause strains[27,74] and subsequent propagation of microcracks into fractures across the full dentin thickness.[10,77] Other obturation methods may create less pressure, such as thermoplasticized gutta-percha, and may reduce the risk of VRFs (see

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Chapter 9).

Type of spreader used.

The use of a more rigid and thick stainless-steel hand spreader may lead to increased strain in the radicular dentin and can result in an increased incidence of root fracture.[27,74] The introduction of more flexible finger spreaders, which have smaller diameter, may greatly reduce such risks.[27] Among the finger spreaders, devices composed of nickel-titanium allow insertion with less force than stainless-steel finger spreaders.[75] The nickel-titanium spreaders also allow a further reduction in the strain induced in the radicular dentin during obturation compared to traditional stainless-steel finger spreaders.[69]

Post design.

Post selection, design, and seating have a significant effect on the strain distribution in the root. Excessively long or thick posts are considered a predisposing factor for VRFs.[23,25,62] The use of posts carries an inherent risk of root fracture, particularly if excessive dentin is removed during preparation. Posts should only be used when essential for core retention and should be avoided whenever sufficient coronal tooth structure is available for the secure retention of the crown.[4,38]

Crown design.

When considering endodontically treated teeth, crowns with a ferrule margin (i.e., supported by a sound tooth structure all around and beyond the gingival margins of the core) provide better strain distribution than similar restorations that are supported by the post and core alone.[4] This design may help to avoid yet another potential predisposing factor for VRFs.[26]

Even though endodontic treatment may be necessary because of pulpal pathosis, the impact of each of the iatrogenic predisposing factors should be minimized as much as possible, as they are likely to have a cumulative deleterious effect in perpetuating VRFs.

Treatment planning

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Prevention is the key to managing VRFs. There are many predisposing factors and iatrogenic causes of these fractures, all of which should be minimized as much as clinically possible. Whereas VRFs may be present in teeth subjected to endodontic retreatment, they are rarely present in teeth that have never undergone endodontic treatment.[22] Therefore, comprehensive clinical, radiographic, and periodontal examination is imperative when evaluating any tooth that is planned for endodontic treatment or retreatment. A flexible periodontal probe is mandatory in such examinations.

When a VRF is determined to be present, extraction of the affected tooth or root is recommended as soon as possible. Any delay may increase the potential for additional periradicular bone loss and potentially compromise the placement of an endosseous implant. Attempts to "repair" a fracture by filling the crevice with a variety of restorative materials have been reported; however, none of these repairs is considered a reliable long-term solution.[8,50,54,85]

There are additional clinical and radiographic examples that can be seen of VRFs in Figs. 22.25 and 22.26.

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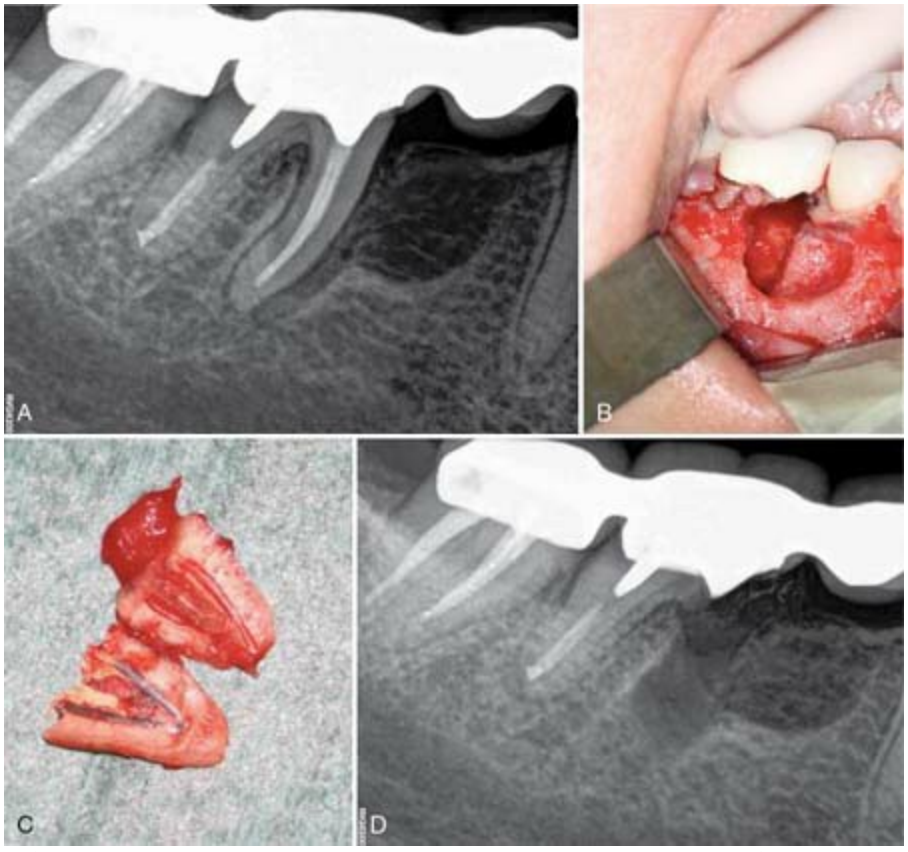

FIG. 22.25 **A,** Periapical radiograph revealing bone loss from crestal bone to apex. **B,** As viewed clinically after full thickness flap. **C,** Mesial root resection performed and can see how the root has split. **D,** Final radiograph after mesial root resection.

A) Radiograph shows three teeth with the center tooth having a radiolucent patch little above the first root canal. The adjacent tooth has no roots.

B) Close-up view of two teeth shows deep holes in the mass next to crowns.

C) Sections of tooth roots. One root has grey root canals. The other root has red root canals where one of the canals is split.

D) Radiograph shows the central tooth without one root canal.

Source: (Courtesy Dr. S. Floratos, Athens, Greece.)

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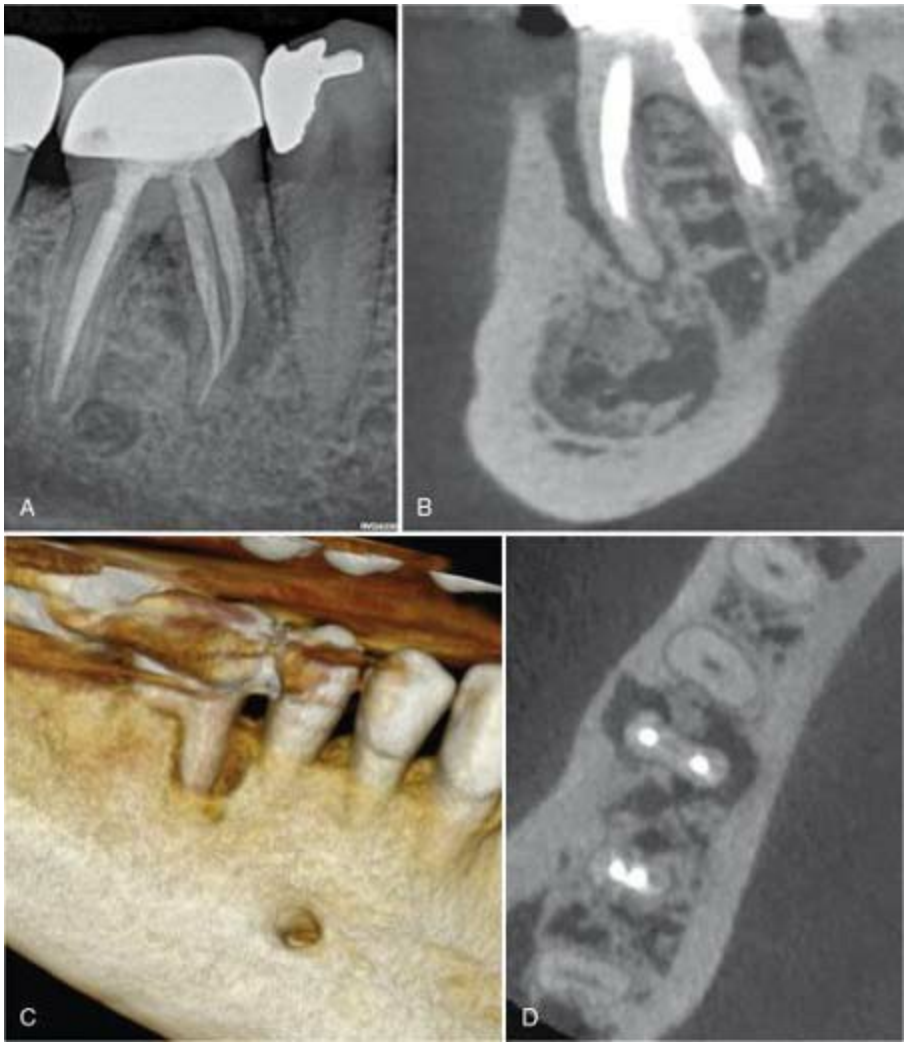

FIG. 22.26 **A,** Periapical radiograph of mandibular right first molar revealing suspected periradicular bone loss. **B,** Cone-beam computed tomography (CBCT) sagittal view of same tooth revealing bone loss from crestal bone to apex. **C,** CBCT anatomic view revealing mesial osseous defect. **D,** CBCT axial view revealing buccal and lingual bone loss midroot.

A) Radiograph shows a central tooth with a dark patch next to one root canal.

B) Radiograph shows the longitudinal view of tooth. One of the root canals has less bone-density at the root apex. The tissue in-between the root and surrounding is porous.

C) The model of tooth shows a lesion around one of the root canals.

D) Radiograph shows teeth arranged diagonally. The central tooth shows bone loss toward buccal and lingual regions.

Source: (Courtesy Dr. R. Kasem.)

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Summary

Because of the wide variety of different types of cracks and fractures in teeth, there may be a myriad of symptoms and presentations that may appear at different stages of the crack or fracture development, making their diagnosis often difficult. The awareness that many of these symptoms represent *stages in an evolving process* may make their interpretation easier for the clinician.

The extensiveness of a crack may directly alter the prognosis assessment and treatment plan for a given tooth; the presence of a fracture may lead to the demise of a tooth and may compromise the periradicular bone. Therefore, developing a diagnosis, prognosis assessment, and treatment plan for teeth with suspected cracks and fractures is essential, with an emphasis on early detection. In addition, endodontic and restorative procedures should focus on minimizing any offending and predisposing factors that may perpetuate cracks and fractures.

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References

1. Abbott PV. *Assessing restored teeth with pulp and periapical diseases for the presence of cracks, caries and marginal breakdown*, Aust Dent J 2004;49: 33.
2. Adorno CG, Yoshioka T, Suda H. *Crack initiation on the apical root surface caused by three different nickel-titanium rotary files at different working lengths*, J Endod 2011;37: 522.
3. Alior JE. *Managing incomplete tooth fractures*, J Am Dent Assoc 2000;131: 1186.

3a American Association of Endodontists. Glossary of endodontic terms ed 10 2020; American Association of Endodontists Chicago, IL.

4. American Association of Endodontists. Restoration of endodontically treated teeth 2004; American Association of Endodontists Endodontics; colleagues for excellence, Chicago, IL.
5. American Association of Endodontists. Cracking the cracked tooth code: detecting and treatment of various longitudinal tooth fractures 2008; American Association of Endodontists Endodontics; colleagues for excellence, Chicago, IL.
6. American Association of Endodontists and American Academy of Oral and Maxillofacial Radiology. *AAE and AAOMR joint position statement: use of cone-beam computed tomography in endodontics*, J Endod 2011;37: 274.
7. Anderson TL. Fracture mechanics: fundamentals and applications ed 3 2005; Taylor & Francis Oxford.
8. Arakawa S. *Treatment of root fractures by CO 2 and*

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www.konkur.in

Nd:YAG lasers, J Endod 1996;22: 662.

9. Bakland LK. *Tooth infractions*, Ingle JI Bakland LK Baumgartner JC Ingle's endodontics ed 6 2008; BC Decker Hamilton, ON.
10. Barreto MS, Moraes RA, da Rosa RA, et al: *Vertical root fractures and dentin defects: effects of root canal preparation, filling and mechanical cycling*, J Endod 2012;38: 1135.
11. Bender IB. *Adult root fracture*, J Am Dent Assoc 1983;107: 413.
12. Berman LH, Hartwell GR. *Diagnosis*, Hargreaves KM Cohen S Cohen's pathways of the pulp ed 10 2011; Elsevier New York 2.
13. Berman LH, Kuttler S. *Fracture necrosis: diagnosis, prognosis assessment, and treatment recommendations*, J Endod 2010;36: 442.
14. Bier CA, Shemesh H, Tanomaru-Filho M, et al: *The ability of different nickel-titanium rotary instruments to induce dentinal damage during canal preparation*, J Endod 2009;35: 236.
15. Boyarsky H, Davis R. *Root fracture with dentin retained post*, Am J Dent 1992;5: 11.
16. Brynjulfsen A, Fristad I, Grevstad T, et al: *Incompletely fractured teeth associated with diffuse longstanding orofacial pain: diagnosis and treatment outcome*, Int Endod J 2002;35: 461.
17. Bürklein S, Tsotsis P, Schäfer E. *Incidence of dentinal defects after root canal preparation: reciprocating versus rotary instrumentation*, J Endod 2013;39: 501.
18. Cameron CE. *Cracked tooth syndrome*, J Am Dent Assoc 1964;68: 405.
19. Cameron CE. *The cracked tooth syndrome: additional*

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www.konkur.in

findings, J Am Dent Assoc 1976;93: 971.
20. Caplan DJ, Weintraub JA. *Factors related to loss of root canal filled teeth*, J Public Health Dent 1997;57: 31.
21. Chai H, Tamse A. *Fracture mechanisms analysis of vertical root fracture from condensation of gutta-percha*, J Biomech 2012;45: 1673.
22. Chan CP, Lin CP, Tseng SC, et al: *Vertical root fracture in endodontically versus non-endodontically treated teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87: 504.
23. Cheung W. *A review of the management of endodontically treated teeth*, J Am Dent Assoc 2005;136: 611.
24. Cohen S, Blanco L, Berman L. *Vertical root fractures—clinical and radiographic diagnosis*, J Am Dent Assoc 2003;134: 434.
25. Cooney JP, Caputo AA, Trabert KC. *Retention and stress distribution of tapered-end endodontic posts*, J Prosthet Dent 1986;55: 540.
26. da Silva NR, Raposo LH, Versluis A, et al: *The effect of post, core, crown type, and ferrule presence on the biomechanical behavior of endodontically treated bovine anterior teeth*, J Prosth Dent 2010;104: 306.
27. Dang DA, Walton RE. *Vertical root fracture and root distortion effect of spreader design*, J Endod 1989;15: 294.
28. Davis R, Overton JD. *Efficacy of bonded and nonbonded amalgam in treatment of teeth with incomplete fractures*, J Am Dent Assoc 2000;131: 469.
29. Eakle WS, Maxwell EH, Braly BV. *Fractures of posterior teeth in adults*, J Am Dent Assoc 1986;112: 215.
30. El Ayouti A, Chu A-L, Kimionis I, et al: *Efficacy of rotary instruments with greater taper in preparing oval root canals*, Int Endod J 2008;41: 1088.

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www.konkur.in

31. European Commission for Radiation Protection: Radiation protection No 172, Cone Beam CT for dental and maxillofacial radiology: evidence-based guidelines, SEDENTEXCT project, 2012. www.sedentexct.eu.
32. European Society of Endodontology position statement. *The use of CBCT in endodontics*, Int Endod J 2014;47: 502.
33. Fuss Z, Lustig J, Katz A, et al: *An evaluation of endodontically treated vertically fractured roots: impact of operative procedures*, J Endod 2001;1: 46.
34. Fuss Z, Lustig J, Tamse A. *Prevalence of vertical root fractures in extracted endodontically treated teeth*, Int Endod J 1999;32: 283.
35. Geurdsen W, Schwarze T, Günay H. *Diagnosis, therapy and prevention of the cracked tooth syndrome*, Quintessence Int 2003;34: 409.
36. Gibbs JW. *Cuspal fracture odontalgia*, Dent Dig 1954;60: 158.
37. Gluskin AH, Radke RA, Frost SL, et al: *The mandibular incisor: rethinking guidelines for post and core design*, J Endod 1995;21: 33.
38. Goodcare CJ, Baba NZ. *Restoration of endodontically treated teeth*, Ingle JI Bakland LK Baumgartner JC Ingle's endodontics ed 6 2008; BC Decker Hamilton, ON 1431.
39. Graves DT, Oates T, Garlet GP. *Review of osteoimmunology and the host response in endodontic and periodontal lesions*, J Oral Microbiol 2011;3: 5304-doi:10.3402/jom.v3i0.5304.
40. Guthrie RC, DiFiore PM. *Treating the cracked tooth with full crown*, J Am Dent Assoc 1991;122: 71.
41. Gutmann JL. *The dentin-root complex: anatomic and biologic considerations in restoring endodontically treated*

Telegram: @dental_k

www.konkur.in

teeth, J Prosth Dent 1992;67: 458.
42. Hassan B, Metska ME, Ozok AR, et al: *Detection of vertical root fractures in endodontically treated teeth by a cone beam computed tomography scan*, J Endod 2009;35: 719.
43. Hassan B, Metska ME, Ozok AR, et al: *Comparison of five cone beam computed tomography systems for the detection of vertical root fractures*, J Endod 2010;36: 126.
44. Helfer AR. *Determination of the moisture content of vital and pulpless teeth*, Oral Surg 1972;34: 661.
45. Hin ES, Wu MK, Wesselink PR, et al: *Effects of self-adjusting file, Mtwo, and ProTaper on the root canal wall*, J Endod 2013;39: 262.
46. Homewood CI. *Cracked tooth syndrome: incidence, clinical findings and treatment*, Aust Dent J 1998;43: 217.
47. Huang TJ, Schilder H, Nathanson D. *Effects of moisture content and endodontic treatment on some mechanical properties of human dentin*, J Endod 1991;18: 209.
48. Kahler B, Moule A, Stenzel D. *Bacterial contamination of cracks in symptomatic vital teeth*, Aust Endod J 2000;26: 115.
49. Katz A, Wasenstein-Kohn S, Tamse A, et al: *Residual dentin thickness in bifurcated maxillary premolars after root canal and dowel space preparation*, J Endod 2006;32: 202.
50. Kawai K, Masaka N. *Vertical root fracture treated by bonding fragments and rotational replantation*, Dent Traumatol 2002;18: 42.
51. Kim HC, Lee MH, Yum J, et al: *Potential relationship between design of nickel-titanium rotary instruments and vertical root fracture*, J Endod 2010;36: 1195.
52. Krell KV, Rivera EM. *A six year evaluation of cracked*

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teeth diagnosed with reversible pulpitis: treatment and prognosis, J Endod 2007;33: 1405.

53. Kruzic JJ, Nalla RK, Kinney JH, et al: *Mechanistic aspects of in vitro fatigue-crack growth in dentin*, Biomaterials 2005;26: 1195.
54. Kudou Y, Kubota M. *Replantation with intentional rotation of complete vertically fractured root using adhesive resin*, Dent Traumatol 2003;18: 115.
55. Lertchirakarn V, Palamara J, Messer HH. *Patterns of vertical fractures: Factors affecting stress distribution in the root canal*, J Endod 2003;29: 523.
56. Lertchirakarn V, Palamara JEA, Messer HH. *Finite element analysis and strain-gauge studies of vertical root fracture*, J Endod 2003;29: 529.
57. Liu R, Kaiwar A, Shemesh H, et al: *Incidence of apical root cracks and apical dentinal detachments after canal preparation with hand and rotary files at different instrumentation lengths*, J Endod 2013;39: 129.
58. Lustig JP, Tamse A, Fuss Z. *Pattern of bone resorption in vertically fractured endodontically treated teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 224.
59. Meister F, Lommel TJ, Gerstein H. *Diagnosis and possible causes of vertical root fractures*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1980;49: 243.
60. Melo SLS, Bortoluzzi EA, Abreu M Jr, et al: *Diagnostic ability of a cone-beam computed tomography scan to assess longitudinal root fractures in prosthetically treated teeth*, J Endod 2010;36: 1879.
61. Metzger Z, Teperovich E, Zary R, et al: *The self-adjusting file (SAF). Part 1: respecting the root canal anatomy—a new concept of endodontic files and its implementation*, J Endod 2010;36: 679.

Telegram: @dental_k

www.konkur.in

62. Morando G, Leupold RJ, Reiers JC. *Measurements of hydrostatic pressure during simulated post cementation*, J Prosthet Dent 1995;74: 586.
63. Nikopoulou-Karayanni K, Bragger U, Lang NP. *Patterns of periodontal destruction associated with incomplete root fractures*, Dentomaxillofac Radiol 1997;26: 321.
64. Onnink PA, Davis RD, Wayman BE. *An in vitro comparison of incomplete root fractures associated with obturation technique*, J Endod 1994;20: 32.
65. Opdam NJ, Roeters JJ, Loomans BA, et al: *Seven-year clinical evaluation of painful cracked teeth restored with a direct composite restoration*, J Endod 2008;34: 808.
66. Patel S, Dawood A, Ford TP, et al: *The potential applications of cone beam computed tomography in the management of endodontic problems*, Int Endod J 2007;40: 818.
67. Peters OA, Paqué F. *Root canal preparation of maxillary molars with the self-adjusting file: A micro-computed tomographic study*, J Endod 2011;37: 53.
68. Peters OA, Peters CL, Schönenberg K, et al: *ProTaper rotary root canal preparation assessment of torque and force in relation to canal anatomy*, Int Endod J 2003;36: 93.
69. Pilo R: Development of strains and mechanical failure in dental roots undergoing root canal obturation and prosthetic rehabilitation. PhD thesis, Tel Aviv University, 2007.
70. Ritchey B, Mendenhall R, Orban B. *Pulpitis resulting from incomplete tooth fracture*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1957;10: 665.
71. Roh BD, Lee YE. *Analysis of 154 cases of teeth with cracks*, Dent Traumatol 2006;22: 118.

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www.konkur.in

72. Rosen H. *Cracked tooth syndrome*, J Prosthet Dent 1982;47: 36.
73. Rud J, Omnell KA. *Root fracture due to corrosion*, Scand J Dent Res 1970;78: 397.
74. Saw L-H, Messer HH. *Root strains associated with different obturation techniques*, J Endod 1995;21: 314.
75. Schmidt KJ, Walker TL, Johnson JD, et al: *Comparison of nickel-titanium and stainless steel spreader penetration and accessory cone fit in curved canals*, J Endod 2000;26: 42.
76. Sedgley CM, Messer HH. *Are endodontically treated teeth more brittle?*, J Endod 1992;18: 332.
77. Shemesh H, Bier CA, Wu MK, et al: *The effects of canal preparation and filling on the incidence of dentinal defects*, Int Endod J 2009;42: 208.
78. Shemesh H, Roeleveld AC, Wesselink PR, et al: *Damage to root dentin during retreatment procedures*, J Endod 2011;37: 63.
79. Snyder DE. *The cracked tooth syndrome and fractured posterior cusp*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1976;41: 698.
80. Tamse A. *Iatrogenic vertical root fractures in endodontically treated teeth*, Endod Dent Traumatol 1988;4: 190.
81. Tamse A. *Vertical root fractures of endodontically treated teeth*, Ingle JI Bakland LK Baumgartner JC Ingle's endodontics ed 6 2008; BC Decker Hamilton, ON 676.
82. Tamse A, Fuss Z, Lustig J, et al: *An evaluation of endodontically treated vertically fractured teeth*, J Endod 1999;25: 506.
83. Tamse A, Fuss Z, Lustig JP, et al: *Radiographic features of vertically fractured endodontically treated maxillary*

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premolars, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88: 348.

84. Tamse A, Kaffe I, Lustig J, et al: *Radiographic features of vertically fractured endodontically treated mesial roots of mandibular molars*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101: 797.
85. Taschieri S, Tamse A, del Fabbro M, et al: *A new surgical technique for preservation of endodontically treated teeth with coronally located vertical root fractures: a prospective study*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;110: 45.
86. Testori T, Badino M, Castagnola M. *Vertical root fractures in endodontically treated teeth: a clinical survey of 36 cases*, J Endod 1993;19: 87.
87. Tsesis A, Rosen E, Tamse A, et al: *A diagnosis of vertical root fractures in endodontically treated teeth based on clinical and radiographic indices: a systematic review*, J Endod 2010;36: 1455.
88. Walton RE, Michelich RJ, Smith GN. *The histopathogenesis of vertical root fractures*, J Endod 1984;10: 48.
89. Walton RE, Torabinejad M. Principles and practice of endodontics ed 3 2002; WB Saunders Philadelphia 516.
90. Wilcox LR, Roskelley C, Sutton T. *The relationship of root canal enlargement to finger-spreader induced vertical root fracture*, J Endod 1997;23: 533.
91. Yeh CJ. *Fatigue root fracture: a spontaneous root fracture in non-endodontically treated teeth*, Br Dent J 1997;182: 261.
92. Yoldas O, Yilmaz S, Atakan G, et al: *Dentinal microcrack formation during root canal preparations by different Ni-Ti rotary instruments and the self-adjusting file*, J Endod

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2012;38: 235.

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23: Restoration of the endodontically treated tooth

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CHAPTER OUTLINE

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Special features of endodontically treated teeth

Once endodontic therapy is completed, the tooth must be adequately restored. Indeed, given the large impact that poor or missing restorations have on the survival of endodontically treated teeth, one could make the argument that the restoration is actually the last step of endodontic therapy. However, it is important to realize that endodontically treated teeth are structurally different from nontreated vital teeth. Major changes following treatment include altered tissue physical characteristics, loss of tooth structure, and possibly discoloration. Research has analyzed these tissue modifications at different levels, including tooth composition, dentin microstructure, and tooth macrostructure. These studies indicate that it is critical to understand the implication of such features on tooth biomechanics, as they will largely influence the restorative approach and means (Table 23.1). Additional in vitro studies dealing with the complexity of the nonvital tooth substrate are reported in the literature; ultimately, clinical studies have documented the global effect of these changes on the long-term survival of endodontically treated teeth.

Table 23.1

Specific Tissue Modifications and Possible Clinical Implications Following Loss of Vitality or Endodontic Treatment

Alteration Level	Specific Changes	Possible Clinical Implication
Composition	Collagen structure Tooth moisture Mineral composition	Increased tooth fragility Reduced adhesion to substrate

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	and content	
Dentin structure	Elasticity modulus and behavior Tensile and shear strength Microhardness	Increased tooth fragility
Tooth macrostructure	Resistance to deformation Resistance to fracture Resistance to fatigue	Increased tooth fragility Reduced retention/stability of the prosthesis

Compositional changes in nonvital teeth and the influence of endodontic therapy

The loss of pulpal vitality is accompanied by a slight change in tooth moisture content. This loss of moisture (9%) is attributed to a change in free water but not in water bonded to the organic and inorganic components.[65,69] This alteration was associated with a slight change in values for the Young modulus and proportional limit.[75] However, no decrease in compressive and tensile strength values was associated with this change in water content.[75] Only one study showed no difference in moisture content between vital and nonvital teeth.[123] No difference in collagen cross-linkage was found between vital and nonvital dentin.[140] Thus nonvital teeth undergo rather minor changes in physical characteristics.

Sodium hypochlorite and chelators such as ethylenediamine tetra-acetic acid (EDTA), cyclohexane-1,2-diaminetetra-acetic acid (CDTA), ethylene glycol-bis-(β-amino-ethyl ether) *N*,*N*,*N*,N-tetra-acetic acid (EGTA), and calcium hydroxide ($Ca[OH]_2$) commonly used for canal irrigation and disinfection interact with root dentin, with either mineral content (chelators) or organic substrate (sodium hypochlorite).[78,116,119] Chelators mainly deplete calcium by complex formation and also affect noncollagenous proteins (NCPs), leading to dentin erosion and surface softening.[78,82,144] Depending on concentration, duration of exposure, and other factors, sodium hypochlorite can demonstrate a proteolytic action by hydrolysis of long peptide chains such as collagen.[68] These alterations are likely to impact dentin and root structure and alter bonding properties of this substrate.

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Dentin structure and properties in nonvital and endodontically treated teeth

It is important to know that dentin displays a range of normal variations in its physical properties, which must be distinguished from alterations related to loss of vitality or endodontic treatment. For instance, dentin microhardness and elasticity actually vary between peritubular and intertubular dentin and depend on tooth location. Peritubular dentin presents an elasticity modulus of 29.8 GPa, whereas intertubular dentin ranges from 17.7 GPa (close to pulp) to 21.1 GPa (close to the root surface).[70,85,101] Most if not all the decreases in hardness on approaching the pulp can be attributed to changes in hardness of the intertubular dentin.[84,85] Overall dentin elasticity modulus is considered to be in the range of 16.5 to 18.5 GPa.[15,32,50,86,121,138]

The changes in mineral density due to the variation in the number and diameter of tubules within the tooth may also contribute to variations in the properties of dentin. Unsurprisingly, dentin hardness values are inversely related to dentin tubule density.[124] Ultra microindentation measurements also demonstrated significantly higher values for hardness and elasticity modulus when forces were parallel to the tubules rather than perpendicular.[133] Differences in maximum strength and compressive strength were also found to vary according to tubule orientation.[121] The ultimate tensile strength (UTS) of human dentin is lowest when the tensile force is parallel to tubule orientation, showing the influence of dentin microstructure and anisotropy of the tissue.[93] No difference was found in the Young modulus of aged, transparent dentin (also called *sclerotic*) and normal dentin,[19,87,167] but the mineral concentration significantly increases and crystallite size is slightly smaller in transparent dentin, in relation with closure of the tubule lumens. Transparent dentin, unlike normal dentin, exhibits almost no yielding before failure. Its fracture toughness is also lowered by about 20%, whereas the fatigue lifetime is deleteriously affected.[87]

Interestingly, comparisons between vital and nonvital dentin of contralateral teeth demonstrate no or only minor differences in microhardness values after periods ranging up to 10 years after treatment.[94,154] Thus the literature does not support a widely held belief that attributes particular weakness or brittleness to nonvital dentin. Others have suggested that

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nonvital teeth in older patients may have greater risk for fracture because the age-related generation of secondary or tertiary dentin would be lost. However, this is not the case because the only impact of age-related tissue changes is the aforementioned reduction in fracture toughness and fatigue lifetime attributed to dentin sclerosis.[87]

The chemicals used for canal irrigation and disinfection, as already mentioned, interact with mineral and organic contents and then reduce dentin elasticity and flexural strength to a significant extent,[64,157] as well as microhardness.[33,77,146] On the contrary, disinfectants like eugenol and formocresol increase dentin tensile strength by way of protein coagulation and chelation with hydroxyapatite (eugenol). Dentin hardness, however, did not prove to be influenced by the latter products.[112]

In conclusion, a possible decrease in tooth strength can be attributed to dentin aging and to a smaller extent to dentin alteration by endodontic irrigants.

Fracture resistance and tooth stiffness of nonvital and endodontically treated teeth

In contrast to the aforementioned factors, the major changes in tooth biomechanics appear to be due to the loss of hard tissue following decay, fracture, or cavity preparation (including the access cavity prior to endodontic therapy).

The loss of hard tooth structure following a conservative access cavity preparation affects tooth stiffness by only 5%.[91,169] The influence of subsequent canal instrumentation and obturation lead only to a slight reduction in the resistance to fracture[91,169] and ultimately have little effect on tooth biomechanics.[91,137,169] From a clinical perspective, one can expect alteration of tooth biomechanics only in cases of nonconservative canal preparation or through the chemical or structural alteration triggered by endodontic irrigants, as previously mentioned.

In fact, the largest reduction in tooth stiffness results from excessive access preparation, especially the loss of marginal ridges. The literature reports a 20% to 63% and a 14% to 44% reduction in tooth stiffness following occlusal and MOD cavity preparations, respectively.[44,92,137] It has been shown that an

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endodontic access cavity combined with an MOD preparation results in maximum tooth fragilization. The cavity depth, isthmus width, and configuration are therefore highly critical to the reduction in tooth stiffness and risk of fracture (Fig. 23.1).[74,83,96,122] This important point has profound clinical implications.

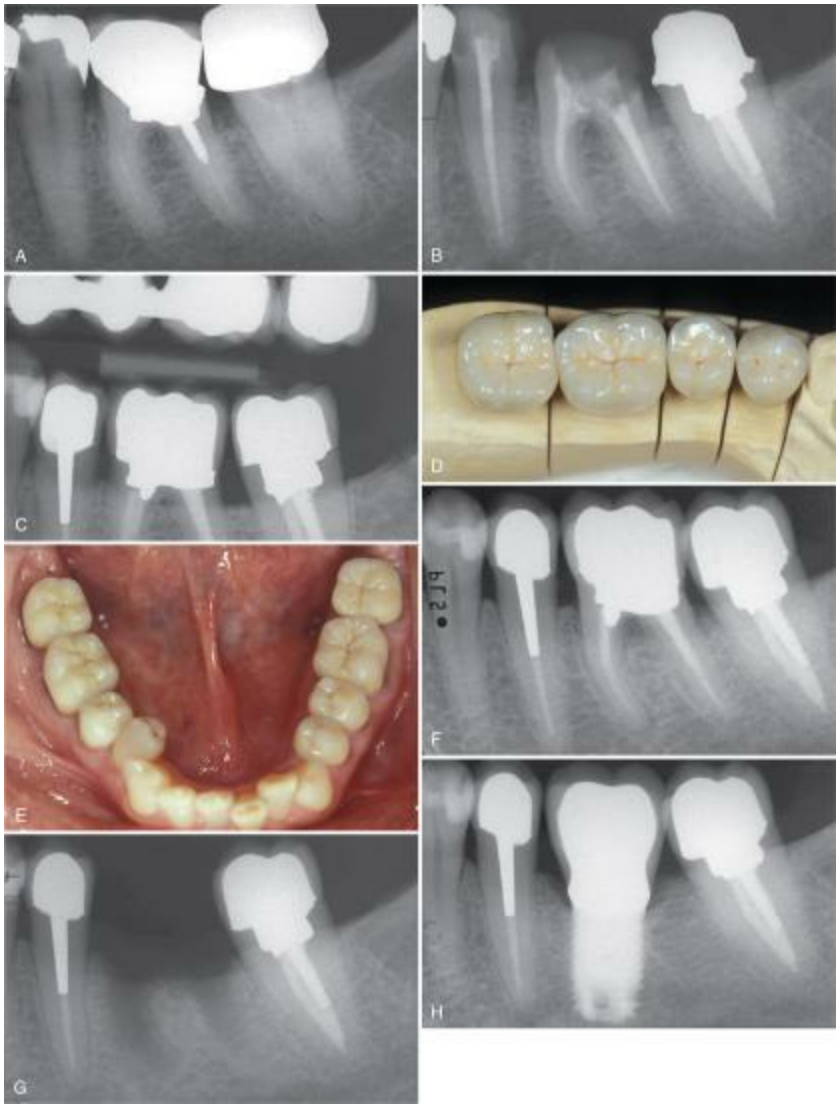

FIG. 23.1 The negative impact of poor initial biomechanical status on restoration success. **A** and **B,** Preoperative radiographic views following the removal of the old metallic foundation. **C,** A new amalgam core, using post and self-anchorage into mesial root structure, was performed. **D,** Prosthetic restorations on working model. **E,** Full arch view after 3 years. **F,** The tooth is symptomatic due to furcation involvement and periapical lesion. **G,** This untreatable tooth was finally

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extracted and replaced by an implant. **H,** Eight-year postoperative radiograph showing a stable situation. Other teeth with less extensive biomechanical damage overcame functional stresses.

A) Radiograph shows three teeth. The first tooth has radiolucent root canal with a hard-density patch on the enamel and a radiolucent round patch on the crown. The second and third teeth have radiopaque crowns.

B) Radiograph shows three teeth with filled root canals. Part of the crown of first and second teeth has less-density tissue.

C) Radiograph shows teeth with filled root canals.

D) Occlusal view of teeth with slight yellow stains.

E) Close-up view of lower jaw with slight yellow stains. Some teeth are not properly aligned.

F) Radiograph shows four teeth. The first tooth has radiolucent root canal with radiopaque patch on the crown. All other teeth have radiopaque crowns and root canals. The root canal of the second tooth is half-filled.

G) Radiograph shows the third tooth missing.

H) Radiograph shows the implantation of new tooth. Its U-shaped root is properly radiopaque with a hole at the base.

The presence of residual hard tissue in the cervical area (which constitutes the *ferrule* for restorations) and a larger amount of residual mineralized tissue in general increase tooth resistance to fracture. It actually allows the axial walls of the crown to encircle the tooth, providing restoration retention and stabilization and reducing cervical tensile stresses.[6,23,161] Crown preparations with as little as 1 mm coronal extension of dentin above the margin double the fracture resistance of preparations, compared to those where the core terminates on a flat surface immediately above the margin[103,161]; therefore a minimal 1-mm ferrule (and ideally 2 to 3 mm) is considered necessary to stabilize the restoration.[161] However, the widths of preparation shoulder and crown margin do not appear to influence fracture strength.[2] Taken together, the height of the ferrule is one of the most important elements in the long-term survival of restorations. These considerations have led to the conclusion that the most important part of the restored tooth is the tooth itself, and no contemporary restorative material or combination of materials will perfectly substitute for lost tooth structure.

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Aesthetic changes in nonvital and endodontically treated teeth

Several aesthetic changes may also occur in nonvital or endodontically treated teeth. For example, color change or the darkening of nonvital teeth is a common clinical observation (Fig. 23.2). In addition, incomplete endodontic treatment can contribute to discoloration. For instance, inadequate cleaning and shaping can leave necrotic tissue in coronal pulp horns, resulting in tooth darkening. In addition, root canal filling materials (gutta-percha and root canal sealer cements, MTA-like materials) retained in the coronal aspect of anterior teeth can detract from the aesthetic appearance. Opaque substances also adversely affect the color and translucency of most uncrowned teeth. Biochemically altered dentin modifies tooth color and appearance. It is generally accepted that organic substances present in dentin (e.g., hemoglobin) might play an important role in this color change and also food and drink pigment penetration triggered by the absence of pulpal pressure. However, the respective contribution of these two phenomena and precise physicochemical mechanisms leading to discoloration are poorly understood or described in the literature.[34,67,131]

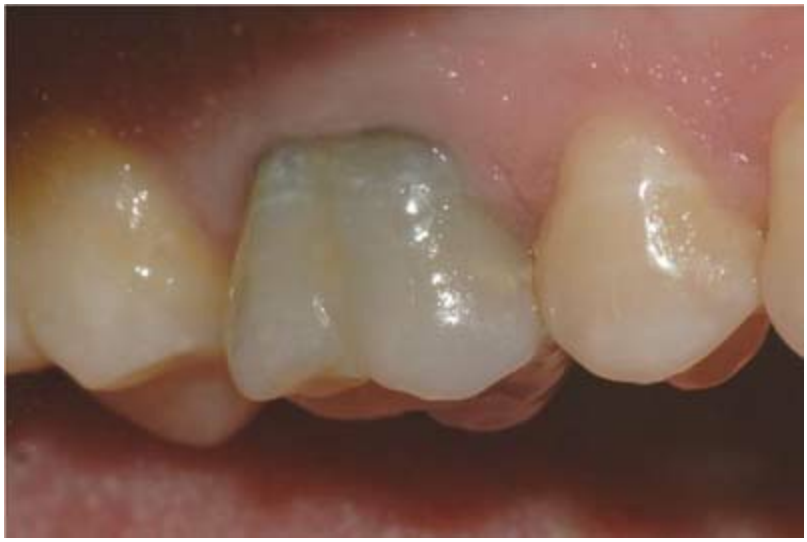

FIG. 23.2 Severe discoloration can significantly disturb aesthetics, even in the lateral area of the smile. When not treatable with bleaching agents or veneers, this condition might justify tooth preparation for a full crown.

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Close-up view shows teeth with yellow stains on them. The tooth at the center has grayish stain as well extending from the gum line.

Thin gingival tissue or, in general, thin biotype, is considered a negative factor for the aesthetic outcome of restorative and prosthetic treatment of discolored teeth.[110,111,118]

Endodontic treatment and subsequent restoration of teeth in the aesthetic zone require careful control of procedures and materials to retain a translucent, natural appearance. It is therefore strongly recommended that one avoid the use of potentially staining endodontic cements and clean all material residues left in the pulpal chamber and access cavity.

Restorative materials and options

As described previously, endodontic treatment, particularly excessive access preparations, can result in significant loss and weakening of tooth structure. Tooth structure lost during endodontic treatment increases the risk of crown fracture, with fatigue mechanisms mediating the fracture of roots over time. Restorations of endodontically treated teeth are designed to (1) protect the remaining tooth from fracture, (2) prevent reinfection of the root canal system, and (3) replace the missing tooth structure.

According to the amount of tissue to be replaced, restorations of endodontically treated teeth rely on different materials and clinical procedures. As a general rule, most structurally damaged teeth should be restored with an artificial crown.

Although the use of a crown built on post and core is a traditional approach, others have advocated the use of direct composite resins for restoring small defects in endodontically treated teeth. More recently, indirect restorations, such as overlays or endocrowns made of composite resins or ceramics, have also been used. The selection of appropriate restorative materials and techniques is dictated by the amount of remaining tooth structure. This is far more relevant to the long-term prognosis of endodontically treated teeth than any properties of post, core, or crown materials.

Direct composite restorations

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When a minimal amount of coronal tooth structure has been lost after endodontic therapy, a direct resin composite restoration may be indicated. Composite resins are a mixture of a polymerized resin network reinforced by inorganic fillers. Contemporary composites have compressive strengths of about 280 MPa, and the Young modulus of composite resins is generally about 10 to 16 GPa, which is close to that of dentin.[134]

When properly cured, resin composites are highly aesthetic, exhibit high mechanical properties, and can reinforce the remaining tooth structure through bonding mechanisms. Typically, 500 to 800 mW/cm^2 of blue light for 30 to 40 seconds is necessary to polymerize an increment of composite that must be 1 to 3 mm thick. Unfortunately, the shrinkage that accompanies polymerization of contemporary composite resins remains a significant problem to the long-term success of these restorations. The use of an incremental filling technique, which helps to reduce shrinkage stresses during polymerization, is highly recommended. The amount of shrinkage will also depend on the shape of the cavity preparation and the ratio of bonded to unbonded (or free) surfaces.[36] This so-called C-factor is a clinically relevant predictor of the risk of debonding and leakage; restorations with high C-factors (>3.0) are at greatest risk for debonding.[175] In other words, a direct composite restoration may be indicated when only one proximal surface of the tooth has been lost; using an incremental filling technique is mandatory.

Classically, direct composite restorations have been placed in anterior teeth that have not lost tooth structure beyond the endodontic access preparation. In such cases, the placement of a direct composite restoration offers an immediate sealing of the tooth, which prevents coronal leakage and recontamination of the root canal system with bacteria. In vitro studies have demonstrated that the fracture resistance of small bonded restorations is nearly as great as that of intact teeth.[57,136]

Although direct composite resins may also be used for small restorations in posterior teeth, they are contraindicated when more than a third of coronal tissue has been lost. In one study,[136] it was reported that the resistance to fracture of endodontically treated teeth is reduced by 69% in cases where MOD cavities are present.[137] Under such conditions, a direct composite restoration may not be appropriate to prevent the tooth structure from fracture and reinfection. Furthermore, resin composite materials may require the use

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of reinforcing in vitro fibers to increase their mechanical resistance. Although most studies on the clinical performance of direct composite restorations were conducted on vital teeth, one clinical report indicates that direct in vitro fiber-reinforced composite restorations may represent a valuable alternative to conventional restorations of endodontically treated teeth.[37] On the contrary, inserting an in vitro fiber post in the root canal of an endodontically treated tooth before bonding a direct MOD restoration significantly reduces its fracture resistance compared to the same composite restoration without a post.[160]

Indirect restorations: Composite or ceramic onlays and overlays

Ceramic or resin composite onlays and endocrowns can also be used to restore endodontically treated teeth. Whereas overlays incorporate a cusp or cusps by covering the missing tissue, endocrowns combine the post in the canal, the core, and the crown in one component.[88,142] Both onlays and endocrowns allow for conservation of remaining tooth structure, whereas the alternative would be to completely eliminate cusps and perimeter walls for restoration with a full crown.[58] Onlays and overlays are generally constructed in the laboratory from either hybrid resin composite or ceramics.

Ceramics are a material of choice for long-term aesthetic indirect restorations because their translucency and light transmission mimic enamel. Whereas traditional feldspathic porcelains were sintered from a slurry, new ceramic materials may be cast, machined, pressed, or slip-cast, in addition to being sintered. New materials either are variations of feldspathic porcelains (e.g., In-Ceram, Cerec, IPS Empress) or may be fabricated from other ceramic systems, including alumina, zirconia, or silica.[3,38] Among these newer compositions is lithium disilicate, which offers high strength, high fracture toughness, and a high degree of translucency. Physical properties of these materials have improved to the point where they can survive high stress-bearing situations such as posterior restorations in endodontically treated teeth.[46,73] Researchers have examined 140 partial Cerec restorations (Vita MKII, feldspathic porcelain) adhesively cemented to endodontically treated teeth and found this treatment approach satisfactory after an

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observation period of 55 months.[9] Their results indicate that survival rates are higher for molars than for premolars.

Onlays, overlays, and endocrowns can also be fabricated from resin composites processed in the laboratory. Using various combinations of light, pressure, and vacuum, these fabrication techniques are claimed to increase the conversion rate of the polymer and, consequently, the mechanical properties of the restorative material. Other investigations have described the application of glass fiber–reinforced composite endocrowns on premolars and molars as single restorations or as abutment for fixed partial dentures.[58,59] One in vitro study by another research team indicates that composite inlays can partially restore the resistance to fracture of endodontically treated molars and prevent catastrophic fractures after loading.[28] Other investigators reported that composite resin MZ100 increased the fatigue resistance of overlay-type restorations in endodontically treated molars when compared to porcelain MKII.[97] Another study used three-dimensional finite element analysis to estimate bone resorption around endocrowns made up of high- (alumina) or low-elastic modulus materials (resin composites). They concluded that the higher resilience of resin composite restorations acts positively against the risk of periodontal bone resorption by reducing the amount of force transferred to root dentin.[4]

Full crowns

When a significant amount of coronal tooth structure has been lost by caries, restorative procedures, and endodontics, a full crown may be the restoration of choice. In a few cases, the crown can be directly built on the remaining coronal structure, which has been prepared accordingly (see the Core Materials section). More frequently, the cementation of a post inside the root canal is necessary to retain the core material and the crown.[164] The core is anchored to the tooth by extension into the root canal through the post and replaces missing coronal structure. The crown covers the core and restores the aesthetics and function of the tooth.

An additional role of the post and core is to protect the crown margins from deformation under function and thereby to prevent coronal leakage. Because most endodontic sealers do not completely seal the root canal space, the coronal seal provided by the placement of a post and core will positively

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influence the outcome of the endodontic treatment.[148] The post's ability to anchor the core is also an important factor for successful reconstruction, because the core and the post are usually fabricated of different materials. Finally, the luting material used to cement the post, the core, and the crown to the tooth will also influence the longevity of the restoration. The post, the core, and their luting or bonding agents together form a *foundation restoration* to support the future crown.[106]

The foundation restoration: General considerations

Although many materials and techniques can be used to fabricate a foundation restoration, no combination of materials can substitute for tooth structure. As a general rule, the more tooth structure that remains, the better the long-term prognosis of the restoration. The coronal tooth structure located above the gingival level will help to create a ferrule.[6,100,128] The *ferrule* is formed by the walls and margins of the crown, encasing at least 2 to 3 mm of sound tooth structure. A properly executed ferrule significantly reduces the incidence of fracture in endodontically treated teeth by reinforcing the tooth at its external surface and dissipating forces that concentrate at the narrowest circumference of the tooth.[95,173] A longer ferrule increases fracture resistance significantly. The ferrule also resists lateral forces from posts and leverage from the crown in function and increases the retention and resistance of the restoration. To be successful, the crown and crown preparation together must meet five requirements:

1. The ferrule (dentin axial wall height) must be at least 2 to 3 mm.
2. The axial walls must be parallel.
3. The restoration must completely encircle the tooth.
4. The margin must be on solid tooth structure.
5. The crown and crown preparation must not invade the attachment apparatus.

Root anatomy can also have significant influence over post placement and selection. Root curvature, furcations, developmental depressions, and root concavities observed at the external surface of the root are all likely to be reproduced inside the root canal. Within the same root, the shape of the canal

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will vary between the cervical level and the apical foramen.[62] As a result, severe alteration of the natural shape of the canal is often necessary to adapt a circular post inside the root. This increases the risk of root perforation, especially in mesial roots of maxillary and mandibular molars that exhibit deep concavities on the furcal surface of their mesial root.[16,89] The tooth is also weakened if root dentin is sacrificed to place a larger-diameter post. A study using three-dimensional electronic speckle-pattern interferometry (ESPI) evaluated the effects of root canal preparation and post placement on the rigidity of human roots.[93] ESPI has the major advantage of being able to assess tooth deformation in real time and can be used repeatedly on the same root because of the nondestructive nature of the test. Study results indicate that root deformability increases significantly after the preparation of a post space. Thus preservation of root structure is also a guiding principle in the decision to use a post, the selection of the post, and the preparation of the post space. This is a reason why not every endodontically treated tooth needs a post and why more conservative approaches that do not rely on the use of a post are currently being developed. However, a post may be used in the root of a structurally damaged tooth in which additional retention is needed for the core and coronal restoration. Posts should provide as many of the following clinical features as possible:

- Maximal protection of the root from fracture
- Maximal retention within the root and retrievability
- Maximal retention of the core and crown
- Maximal protection of the crown margin seal from coronal leakage
- Pleasing aesthetics, when indicated
- High radiographic visibility
- Biocompatibility

From a mechanical point of view, an endodontic post should not break, should not break the root, and should not distort or allow movement of the core and crown. An ideal post would have an optimal combination of resilience, stiffness, flexibility, and strength. *Resilience* is the ability to deflect elastically under force without permanent damage. It is a valuable quality in endodontic posts, but too much flexibility in a narrow post

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compromises its ability to retain the core and crown under functional forces. *Stiffness* describes a material's ability to resist deformation when stressed. The stiffness of a material is an inherent physical property of that material, regardless of size. However, the actual flexibility of a post depends both on the diameter of a specific post and on the modulus of elasticity of the post's material. Posts with a lower modulus of elasticity are more flexible than posts of the same diameter with a higher modulus of elasticity. Posts made of nonstiff materials (low modulus of elasticity) are more resilient, absorb more impact force, and transmit less force to the root than stiff posts, but low-modulus posts fail at lower levels of force than do high-modulus posts.[99,120,143]

Excessive flexing of the post and micromovement of the core are particular risks in teeth with minimal remaining tooth structure, because these teeth lack their own cervical stiffness as a result of the missing dentin. Post flexion can also distort and open crown margins. Open margins can result in potentially devastating caries or endodontic leakage and apical reinfection. Extensive caries extending into the root can be as irreparable as root fracture. Because rigid posts flex and bend less than nonrigid posts, they can limit movement of the core and possible disruption of the crown margins and cement seal. However, the force must go somewhere. Force from a stiff post is transmitted to the root, next to the apex of the post. An attempt to strengthen a weak root by adding a stiff post can instead make the root weaker as a result of the force concentration of a stiff rod in a more flexible material. Stress concentration in the post/root complex can lead to the self-destructive processes of cracking and fracturing. Root fracture is particularly a risk in teeth with minimal remaining tooth structure to support a ferrule.

Roots also flex under force, which is a function of both the modulus of elasticity of dentin and the diameter of the root. Dentin is relatively flexible, and posts can be flexible or stiff. Although no material can behave exactly like dentin, a post with functional behavior similar to that of dentin is beneficial when the post must be placed next to dentin. Posts have been developed with a modulus of elasticity closer to dentin than that offered by traditional metal posts. But posts are significantly narrower than roots, and the actual deflection of a post within dentin is a function of both the modulus of elasticity and the diameter. The modulus of elasticity of various posts,

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compared with that of dentin, represents only one aspect of flexion.

In summary, an ideal post would be resilient enough to cushion an impact by stretching elastically, thereby reducing the resulting stress to the root. It would then return to normal without permanent distortion. At the same time, this ideal post would be stiff enough not to distort, permanently bend, or structurally fail under mastication forces. Finally, the perfect post would combine the ideal level of flexibility and strength in a narrow-diameter structure, which is dictated by root canal morphology. Current post systems are designed to provide the best compromise between the desired properties and inherent limitations of available materials.

Why roots fracture

Structures subjected to low but repeated forces can appear to fracture suddenly for no apparent reason. This phenomenon, also known as *fatigue failure,* occurs when a material or a tissue is subjected to cyclic loading. Fatigue may be characterized as a progressive failure phenomenon that proceeds by the initiation and propagation of cracks; many failures of teeth or materials observed in the mouth are fatigue-related. Because teeth are subjected to fluctuating cycles of loading and unloading during mastication, fatigue failure of dentin, posts, cores, crown margins, or adhesive components is likely to occur.[153] Mechanical loading will favor the propagation of microcracks that will progress from the coronal to the apical region of the tooth.

Initial failure of crown margins from fatigue loading is clinically undetectable. However, when measured in vitro, early failure resulted in significant leakage of crown margins, extending between the tooth, restoration, and post space. Particularly in teeth with minimal remaining tooth structure, fatigue can cause endodontic posts to bend permanently or break, or it can cause a fiber-matrix complex to disintegrate.

Fatigue failure of nonvital teeth restored with a post is more catastrophic because it may result in a complete fracture of the root. A post placed into a dentin root will function physically like any structural rod anchored in another material. This means that the forces applied on the post are transmitted to the root dentin with characteristic patterns depending on the modulus of elasticity of both the post and the dentin. If the post has a higher

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modulus than the dentin, the stress concentration is adjacent to the bottom of the post (Fig. 23.3). This is evident in clinical cases of root fracture originating at the apex of a rigid post.

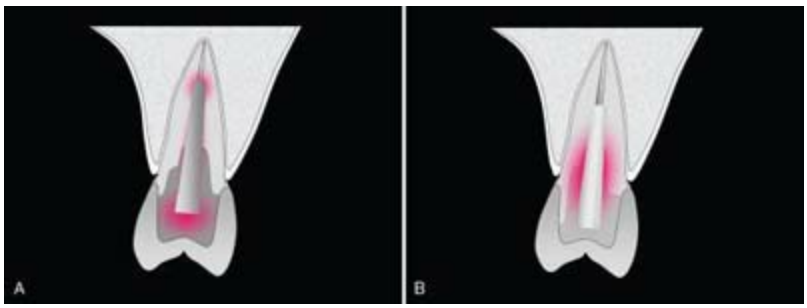

FIG. 23.3 **A,** Stress distribution within a metallic post and core foundation and residual tooth structure, according to photoelastic studies. The post is cemented and usually penetrates the apical portion of the root. Functional stresses accumulate inside the foundation, slightly around the post and further inside the canal, around the apex of the post; there is less stress buildup in the cervical area compared to that with a fiber post (as shown in Fig. 23.3, *B*). This configuration more ideally protects the coronocervical structures, but when failing, it results in severe untreatable root fractures. **B,** Stress distribution within a fiber post/composite foundation and residual tooth structure, according to photoelastic and FEM studies. The post is bonded to the canal walls and penetrates the canal less apically. Functional stresses mainly accumulate around the post in the cervical area. This configuration protects the cervical area less efficiently but tends to prevent untreatable root fractures. The presence of a ferrule is mandatory.

A) Tooth has grey root canal with pink spots at the root tip and pulp chamber.

B) Tooth has white root canal and pink spots along the lateral sides of the root canal.

Source: (Adapted from Dietschi D, Duc O, Krejci I, Sadan A: Biomechanical considerations for the restoration of endodontically treated teeth: a systematic review of the literature—part 1. Composition and micro- and macrostructure alterations, *Quintessence Int* 38:733, 2007.)

When the stiffness of the endodontic post is similar to that of dentin, stresses are not concentrated in the dentin adjacent to the apex of the post but rather are dissipated by both the coronal and the root dentin (see Fig. 23.3). A resilient post can also prevent a sudden blow by stretching elastically, which

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reduces the transient forces against the tooth, but a post that is too elastic becomes too flexible for retaining a core and a crown when the tooth cannot do so on its own. A resilient post that is overloaded fails with less force than a stiffer post. This limits the amount of resilience that can be designed into a post.

Direct foundation restorations

In general, the evolution of foundation restorations has been to diminish invasiveness and eliminate some components in selected cases. When a sufficient amount of tissue is present at the periphery of the prepared tooth, a direct foundation restoration is indicated. In the direct technique, a prefabricated post is cemented inside the root canal, and the core is built directly on the prepared tooth. For other clinical situations, an indirect custom-cast post and core may be indicated.

Various materials can be used to fabricate a direct foundation restoration. Although there is growing interest in using resin-based materials such as resin composites or fiber-reinforced resin posts, more traditional materials such as amalgam are still used for that purpose.[27] For clarity, the components used for fabricating a direct foundation restoration (e.g., the endodontic post and core material) are described individually.

Posts.

The large number of post designs and materials available on the market reflects the absence of consensus in that field. Based on what manufacturers or clinicians consider the most important properties, posts can be fabricated from metal (gold, titanium, stainless steel), ceramic, or fiber-reinforced resins. As a general rule, a post needs retention and resistance. Whereas *post retention* refers to the ability of a post to resist vertical forces, *resistance* refers to the ability of the tooth/post combination to withstand lateral and rotational forces. Resistance is influenced by the presence of a ferrule, the post's length and rigidity, and the presence of antirotational features. A restoration lacking resistance form is not likely to be a long-term success, regardless of the retentiveness of the post.

Prefabricated metallic posts.

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Prefabricated metallic posts are frequently used for the fabrication of a direct foundation restoration. These posts are classified several ways, including by alloy composition, retention mode, and shape. Materials used to fabricate metallic posts include gold alloys, stainless steel, or titanium alloys. Metallic posts are very strong and, with the exception of the titanium alloys, very rigid.[90] One study indicates that the flexural strength of stainless steel posts is about 1430 MPa and that flexural modulus approximates 110 GPa.[130] On the other hand, titanium posts are less rigid (66 GPa) but exhibit a flexural strength (1280 MPa) similar to stainless steel.

The retention of prefabricated posts inside the root canal is also essential for successful restorations. Two basic concepts have been used to promote the retention of endodontic posts: active posts and passive posts. Active posts derive their primary retention directly from the root dentin by the use of threads. Most active posts are threaded and are intended to be screwed into the walls of the root canal. A major concern about threaded posts has been the potential for vertical root fracture during placement. As the post is screwed into place, it introduces great stresses within the root, causing a wedging effect.[163] It is therefore generally accepted that *the use of threaded posts should be avoided.* Furthermore, the improved retention once offered by threaded posts can now be achieved with adhesive luting cements (discussed later).[117] Passive posts are passively placed in close contact to the dentin walls, and their retention primarily relies on the luting cement used for cementation. The shape of a passive post may be either tapered or parallel.[139] A parallel post is more retentive than a tapered post but also requires removal of more root dentin during the preparation of the post space. Parallel posts are reported to be less likely to cause root fractures than tapered posts, although they are less conforming to the original shape of the root.[79,152,161] Unfortunately, modern techniques for root canal preparation use tapered nickel-titanium (NiTi) rotary shaping files, which result in a very wide tapered and unretentive canal exhibiting a significant divergence from apical to coronal.[145] Longer posts are often necessary to accommodate this problem and offer adequate retention; adequate length in the root canal is considered to be greater than 6 mm. When teeth are protected by crowns with an adequate ferrule, longer posts do not further increase fracture resistance.[80] Posts designed with mechanical locking features in the heads and roughened

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surface texture can show better retention of the core.[29]

Fiber posts.

A fiber post consists of reinforcing fibers embedded in a resin-polymerized matrix. Monomers used to form the resin matrix are typically bifunctional methacrylates (Bis-GMA, UDMA, TEGDMA), but epoxies have also been used. Common fibers in today's fiber posts are made of carbon, glass, silica, or quartz but the type, volume content, and uniformity of the fibers and the matrix are proprietary and vary among fiber post systems. These differences in the manufacturing process may reflect the large variations observed among different types of fiber posts subjected to a fatigue-resistance test.[63] Fibers are commonly 7 to 20 μm in diameter and are used in a number of configurations, including braided, woven, and longitudinal. The original fiber posts consisted of carbon fibers embedded in epoxy resin, but quartz-fiber posts are currently preferred for their favorable mechanical properties, aesthetic qualities, and ability to chemically bond to the polymer matrix.[49] One study indicates that the flexural strength of glass-, silica-, or quartz-fiber posts approximates 1000 MPa and that flexural modulus is about 23 GPa.[35] Current fiber posts are radiopaque and may also conduct the light for polymerization of resin-based luting cements. A light-transmitting post results in better polymerization of resin composites in the apical area of simulated root canals, as measured by hardness values.[141,174] To enhance bonding at the post/core/cement interfaces, several physicochemical pretreatments such as silanization or sand blasting of the post surface have been described. Research indicates that silanization, hydrofluoric etching, and sandblasting (with 30 to 50 μm Al_2O_3) do not modify the mechanical properties of different glass-, silica-, or quartz-fiber posts.[5]

It is generally accepted that bonding fiber posts to root canal dentin can improve the distribution of forces applied along the root, thereby decreasing the risk of root fracture and contributing to the reinforcement of the remaining tooth structure.[12,18] A well-adapted adhesively cemented fiber post is considered the most retentive with the least stress generated on the canal walls. In a retrospective study that evaluated three types of bonded fiber posts, investigators reported a 3.2% failure of 1306 fiber posts in recalls of 1 to 6 years.[54] More recently, another study reported survival rates of 98.6%

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and 96.8% for parallel-sided and tapered fiber posts, respectively, placed in anterior teeth covered with full-ceramic crowns after a mean observation period of 5.3 years.[156]

Zirconia posts are composed of zirconium dioxide (ZrO_2) partially stabilized with yttrium oxide and exhibit a high flexural strength. Zirconia posts are aesthetic, partially adhesive, rigid, but also brittle. Zirconia posts cannot be etched, and available literature suggests that bonding resins to these materials is less predictable and requires substantially different bonding methods than conventional ceramics.[11] When a composite core is built on a zirconia post, core retention may also be a problem. Controversies exist about the efficiency of airborne particle abrasion at establishing a durable resin bond to zirconia posts treated or not treated with a coupling agent.[1,127] Overall, there are concerns about the rigidity of zirconia posts, which tends to make those posts too brittle. Other reports indicate that the rigidity of zirconia posts negatively affects the quality of the interface between the resin core material and dentin when subjected to fatigue testing.[40,43]

Core materials.

The core replaces carious, fractured, or missing coronal structure and helps to retain the final restoration. Desirable physical characteristics of a core include (1) high compressive and flexural strength, (2) dimensional stability, (3) ease of manipulation, (4) short setting time, and (5) the ability to bond to both tooth and post. Core materials include composite resin, cast metal or ceramic, amalgam, and sometimes glass-ionomer materials. The core is anchored to the tooth by extension into the coronal aspect of the canal or through the endodontic post. The importance of retention between the post, the core, and the tooth increases as remaining tooth structure decreases.

Composite resin core.

Composite core materials (see Figs. 23.12–23.14, located further in the chapter) take a number of strategies to enhance their strength and resistance; metal may be added, filler levels may be greater, or faster-setting ionomers may be used.[134] Composite core materials have been shown to exhibit slightly better mechanical values than conventional materials, but improvements are negligible.[176] However, they appear to be superior to

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silver-glass-ionomer cement and amalgam.[30] The advantages of composite core resins are adhesive bonding to tooth structure and many posts, ease of manipulation, rapid setting, and translucent or highly opaque formulations. Composite cores have been shown to protect the strength of all-ceramic crowns equally to amalgam cores. The bond strength of composite cores to dentin depends on a complete curing of the resin materials, so dentin bonding agents must be chemically compatible with composite core materials. Self-cure composite resins require self-cure adhesives and are mostly incompatible with light-cure adhesives.[26] However, no adhesive has been shown to completely eliminate microleakage at the margins of the restoration.[17] A degradation of the resin core or the marginal integrity of the crown can result in invasion of oral fluids. Therefore as with all buildup materials for decimated teeth, more than 2 mm of sound tooth structure should remain at the margin for optimal composite resin core function.

Composite core materials can be used in association with metallic, fiber, or zirconia posts. This is frequently observed in the presence of structurally compromised teeth. They may provide some protection from root fracture in teeth restored with metal posts compared with amalgam or gold cores. Loosening of the post, core, and crown with composite core can occur, but composite cores have been shown to fail more favorably than amalgam or gold.[129] A retrospective study of the clinical performance of fiber posts indicates that fiber posts and cores have a failure rate ranging between 7% and 11% after a service period of 7 to 11 years and that post loosening may also occur.[53] Composite core materials are typically two-paste, self-cured composites, but light-curing materials are also available. The use of light-curing composite core materials generally eliminates the risk of chemical incompatibility between adhesives and self-curing resin core materials. Bonding light-cured resin composites to the irregular structure of the pulp chamber and canal orifices might eliminate the need for a post when sufficient tooth structure remains. Research indicates that bonding to the dentin walls of the pulp chamber is easier and superior to resin dentin bonds made on dentin canal walls.[7]

Amalgam core.

Dental amalgam is a traditional core buildup material with a long history of

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clinical success. Although there are many variations in the alloy's composition, more recent formulations have high compressive strength (400 MPa after 24 hours), high tensile strength, and a high modulus of elasticity. High-copper alloys tend to be stiffer (60 GPa) than low-copper alloys.

Amalgam can be used with or without a post. In the 1980s, investigators described the amalcore.[113] With the amalcore technique, amalgam is compacted into the pulp chamber and 2 to 3 mm coronally of each canal. The following criteria were considered for the application of this technique: the remaining pulp chamber should be of sufficient width and depth to provide adequate bulk and retention of the amalgam restoration, and an adequate dentin thickness around the pulp chamber was required for the tooth-restoration continuum rigidity and strength. The fracture resistance of the amalgam coronal-radicular restoration with 4+ mm of chamber wall was shown to be adequate, although the extension into the root canal space had little influence.[81]

Amalgam can also be used in combination with a prefabricated metallic post when the retention offered by the remaining coronal tissue needs to be increased. Amalgam cores are highly retentive when used with a preformed metal post in posterior teeth; they require more force to dislodge than cast posts and cores.[102] Others have suggested the use of adhesive resins to bond amalgam to coronal tissue.[155]

Significant disadvantages of amalgam cores are the "nonadhesive nature" of the material, the potential for corrosion, and subsequent discoloration of the gingiva or dentin. Amalgam use is declining worldwide because of legislative, safety, and environmental issues.

Glass ionomer core and modified glass ionomer core.

Glass ionomer and resin-modified glass ionomer cements are adhesive materials useful for small buildups or to fill undercuts in prepared teeth. The rationale for using glass ionomer materials is based on their cariostatic effect resulting from fluoride release. However, their low strength and fracture toughness result in brittleness, which contraindicates the use of glass-ionomer buildups in thin anterior teeth or to replace unsupported cusps. They may be indicated in posterior teeth in which (1) a bulk of core material is possible, (2) significant sound dentin remains, and (3) caries control is indicated.[172]

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Resin-modified glass ionomer materials are a combination of glass ionomer and composite resin technologies and have properties of both materials. Resin-modified glass ionomers have moderate strength, greater than glass ionomers but less than composite resins. As a core material, they are adequate for moderate-sized buildups, but hygroscopic expansion can cause the fracture of ceramic crowns and fragilized roots.[158] The bond to dentin is close to that of dentin-bonded composite resin and significantly higher than traditional glass ionomers. Today, resin composites have replaced glass ionomer materials for core fabrication.

Indirect foundation restorations: Cast post and core

For many years, use of the cast metal post and core has been the traditional method for fabricating the foundation restoration of a prosthetic crown. Classically, smooth-sided, tapered posts conforming to the taper of the root canal are fabricated from high noble alloys, although noble and base-metal classes of dental alloys have also been used. Noble alloys used for post and core fabrication have high stiffness (approximately 80 to 100 GPa), strength (1500 MPa), hardness, and excellent resistance to corrosion.[31]

One advantage of the cast post/core system is that the core is an integral extension of the post and that the core does not depend on mechanical means for retention on the post. This construction prevents dislodgment of the core from the post and root when minimal tooth structure remains. However, the cast post/core system also has several disadvantages. Valuable tooth structure must be removed to create a path of insertion or withdrawal. Second, the procedure is expensive because two appointments are needed, and laboratory costs may be significant. The laboratory phase is technique sensitive. Metal casting of a pattern with a large core and a small-diameter post can result in porosity in the gold at the post/core interface. Fracture of the metal at this interface under function results in failure of the restoration. Most importantly, the cast post/core system has a higher clinical rate of root fracture than preformed posts.[47,159]

Studies on cast post retention have shown that the post must fit the prepared root canal as closely as possible to be perfectly retained. When a ferrule is present, custom cast posts and cores exhibit a higher fracture resistance compared to composite cores built on prefabricated metallic posts

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or carbon posts.[98] Cast posts are also known to exhibit the least amount of retention and are associated with a higher failure rate compared to prefabricated parallel-sided posts. In a classic retrospective study (1 to 20 years) of 1273 endodontically treated teeth in general practice, 245 (19.2%) were restored with tapered cast posts and cores. Among these, 12.7% were deemed failures. This failure rate was higher than that for the other passive post systems used. Of particular concern was the fact that 39% of the failures led to unrestorable teeth requiring extraction. Thirty-six percent of the failures were due to loss of retention, and 58% were due to the fracture of the root. It has been suggested that tapered smooth-sided posts have a “wedging” effect under functional loading, and it is this that leads to increased risk of root fracture.[162]

One 6-year retrospective study reported a success rate higher than 90% using a cast post and core as a foundation restoration.[8] The lower failure rate and fewer root fractures were attributed to the presence of an adequate ferrule and careful tooth preparation. Attention has also been drawn to the fact that the higher failure rate may be due to the fact that nearly half of the posts were shorter than recommended from the literature. A venting groove for the cement along the axis of the post results in less stress on residual tissues.

Luting cements

A variety of cements have been used to cement endodontic posts and include traditional cements, glass ionomer cements, and resin-based luting cements.

Traditional cements

Zinc phosphate cements or polycarboxylate cements are still used for cementation of posts and crowns. They are generally supplied as a powder and a liquid, and their physical properties are highly influenced by the mixing ratio of the components. Their compressive strength is about 100 MPa, and elastic moduli are lower than that of dentin (5 to 12 GPa). Zinc phosphate cement is mostly used for cementing metal restorations and posts; film thickness of the zinc phosphate cement is less than 25 μm. These cements provide retention through mechanical means and have no chemical bond to the post or to dentin but provide clinically sufficient retention for posts in teeth with adequate tooth structure.

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Glass ionomer luting cements

Glass ionomer cements are a mixture of glass particles and polyacids, but resin monomers may also be added. Depending on the resin content, glass ionomer cements can be classified as either conventional or resin-modified glass ionomer cements. Conventional glass ionomer cements have compressive strengths ranging between 100 and 200 MPa; the Young modulus is generally about 5 GPa. They are mechanically more resistant than zinc phosphate cements, and they can bond to dentin with values ranging between 3 and 5 MPa. Some authors still recommend the use of glass ionomer cements for the cementation of metallic posts. Major advantages of conventional glass ionomer cements are their ease of manipulation, chemical setting, and ability to bond to both tooth and post. On the contrary, resin-modified glass ionomer cements are not indicated for post cementation because these cements exhibit hygroscopic expansion that can promote fracture of the root.

Resin-based luting cements

Today there is a trend toward the use of adhesive cements for bonding endodontic posts during the restoration of nonvital teeth. The rationale for using adhesive cements is based on the premise that bonding posts to root canal dentin will reinforce the tooth and help retain the post and the restoration.[48] Contemporary resin-based luting cements have been shown to exhibit compressive strengths around 200 MPa and elastic moduli between 4 and 10 GPa.[24] These materials may be polymerized through a chemical reaction, a photopolymerization process, or a combination of both mechanisms. Photo-polymerization of these resin-based materials is often necessary to maximize strength and rigidity.

Most luting cements require a pretreatment of the root canal dentin with either etch-and-rinse or self-etching adhesives. Both types of adhesives have been shown to form hybrid layers along the walls of the post spaces.[10] However, bonding to root canal dentin may be compromised by the use of endodontic irrigants such as sodium hypochlorite, hydrogen peroxide, or their combination.[115] Because these chemicals are strong oxidizing agents, they leave behind an oxygen-rich layer on the dentin surface that inhibits the polymerization of the resin.[151] Previous research has shown that the bond

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strength of C&B Metabond to root canal dentin was reduced by half when the dentin was previously treated with 5% sodium hypochlorite (NaOCl) or 15% EDTA/10% urea peroxide (RC Prep, Premier Dental, Plymouth Meeting, PA).[107] Other reports indicate that the contamination of the dentin walls by eugenol diffusing from endodontic sealers may also affect the retention of bonded posts.[66,168] Furthermore, it is difficult to control the amount of moisture left in a root canal after acid etching, making impregnation of collagen fibers with etch-and-rinse adhesives problematic. The use of self-etching adhesives has been proposed as an alternative for the cementation of endodontic posts, because self-etching adhesives are generally used on dry dentin and do not require rinsing of the etchant. However, their efficiency at infiltrating thick smear layers like those produced during post space preparation remains controversial.[104,171] More recently, dual-curing adhesives have been developed to ensure a better polymerization of the resin deep inside the root canal. Dual-cured adhesives contain ternary catalysts to offset the acid-base reaction between the acidic monomers and the basic amines along the composite/adhesive interface.[105]

Although both self-curing and light-curing luting cements can be used for cementation of prefabricated endodontic posts, most resin cements have a dual-curing process that requires light exposure to initiate the polymerization reaction. Dual-curing cements are preferred because there are concerns as to whether light-curing materials are properly cured, especially in areas of difficult light access, such as the apical portion of the root canal. However, it has been reported that photocured composites generate more shrinkage stress and exhibit less flow than chemically cured composites.[52] Contraction stresses induced by polymerization also depend on the geometry of the post space and the thickness of the resin film. Previous research indicates that the restriction of flow of resin cements by the configuration of the root canal can significantly increase the contraction stress at the adhesive interface.[51,166]

In recent years, a number of techniques have been used to measure the adhesion of resin-based luting cements to root canal dentin. These methods include the pull-out tests, the microtensile bond strength tests, and the push-out tests.[45,61] Although laboratory tests confirmed that bond strengths ranging between 10 and 15 MPa can be obtained with modern resin-based luting cement, there is also evidence that frictional retention is a factor contributing

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to post retention.[14] It is generally accepted that bonding to dentin of the pulp chamber is more reliable than bonding to root canal dentin, especially at the apical level.[126] The lowered bond strength values recorded at the apical third of the root canal are likely to be related to the reduced number of dentinal tubules available for dentin hybridization. Shorter posts may be used when successful bonding occurs between fiber-reinforced posts and root dentin, because current adhesive luting cements can assist in the retention of posts in the root canal space.[132]

Another factor that may influence the performance of resin-based luting cements is the thickness of the cement layer. The cementation of endodontic fiber posts with thicker cement layers might be required when posts do not perfectly fit inside the root canal. Although a slight increase in cement thickness (up to 150 microns) does not significantly affect the performance of adhesive luting cements applied to root canal dentin, thicker layers may be detrimental to bond quality.[76,150]

One study indicates that bond strength to radicular dentin might be maximized by adopting procedures that compensate for polymerization stresses.[13] The bonding procedures are realized in two separate steps. The initial step allows optimal resin film formation and polymerization along the root canal walls, leading to more ideal resin-dentin hybridization without stresses imposed by the placement of the post. A second step bonds the post to the cured resin film. The polymerization shrinkage that occurs during the initial adhesive coating step reduces the effects of stress imposed when the resin-coated post polymerizes, thereby preserving the bond integrity.

Although the bonding performance of resin-based luting cements is well documented, other reports indicate that resin-dentin bonds degrade over time.[22,56] The loss of bond strength and seal are attributed to the degradation of the hybrid layer created at the dentin-adhesive interface. This is particularly true for etch-and-rinse adhesives, because the gelatinization of collagen fibers caused by phosphoric acids may restrict the diffusion of the resin within the interfibrillar spaces and may leave unprotected fibers available for degradation. Removing organic components from the demineralized dentin prior to bonding procedures has been suggested. The use of dilute NaOCl (0.5%) after acid etching or the conditioning of dentin smear layers with EDTA (0.1 M, pH 7.4) has been shown to produce more

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durable resin-dentin bonds made with single-step etch-and-rinse adhesives.[149]

Other research indicates that the degradation of denuded collagen fibrils exposed in incompletely infiltrated hybrid layers is driven by an endogenous proteolytic mechanism involving the activity of matrix metalloproteinases (MMPs).[21,125] The release of MMPs such as collagenases has been evidenced in both coronal and root dentin of fully developed teeth of young patients.[147] Researchers suggest that conditioning root canal dentin with a broad-spectrum protease inhibitor such as chlorhexidine (2 wt% chlorhexidine digluconate solution) might be useful for the preservation of dentin bond strength over time.[20]

Interestingly, these dentin-conditioning procedures, which may improve the resistance of the resin-dentin bond to chemical degradation, also act as antibacterial agents; this might be of interest in the endodontic context.

Self-adhesive cements

More recently, self-adhesive resin cements have been introduced as an alternative to conventional resin-based luting cements. Self-adhesive luting cements contain multifunctional phosphoric acid methacrylates that react with hydroxyapatite and simultaneously demineralize and infiltrate dental hard tissue.[108] They do not require any pretreatment of the tooth substrates, and their clinical application is accomplished in a single step. Therefore the self-etching capability of these new cements reduces the risk for incomplete impregnation of the conditioned tissue by the resins and reduces technique sensitivity. The elastic moduli of chemically cured self-adhesive resin cements are relatively low (4 to 8 GPa) but generally increase when a dual-curing process is used. It is therefore recommended that all dual-cured resin cements receive maximal light to achieve superior material properties wherever clinically possible.[135] Adhesion performance to dentin was found comparable to multistep luting cements but bonding to enamel without prior phosphoric acid etching is not recommended.[72] However, their long-term clinical performances need to be assessed before making a general recommendation for their use.

Pretreatment evaluation and treatment

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strategy

Before any therapy is initiated, the tooth must be thoroughly evaluated to ensure treatment success. Each tooth must be examined individually and in the context of its contribution to the overall treatment plan and rehabilitation. This assessment includes endodontic, periodontal, biomechanical, and aesthetic evaluations. Planning of the restoration for endodontically treated teeth brings together all aforementioned biomechanical and clinical factors, as well as the various materials and procedures designed to address them.

Pretreatment evaluation

Endodontic evaluation

The prerestorative examination should include an inspection of the quality of existing endodontic treatment. New restorations, particularly complex restorations, should not be placed on abutment teeth with a questionable endodontic prognosis. Endodontic retreatment is indicated for teeth showing radiographic signs of apical periodontitis or clinical symptoms of inflammation. Restorations that require a post need a post space, which is prepared by removal of gutta-percha from the canal. Canals obturated with a silver cone or other inappropriate filling material should be endodontically retreated before starting any restorative therapy. Because the probability for periapical tissue to heal after endodontic retreatment is reasonably high, the chances to retain a well-restored tooth in asymptomatic function over time are excellent.[119]

Periodontal evaluation

Maintenance of periodontal health is also critical to the long-term success of endodontically treated teeth. The periodontal condition of the tooth must therefore be determined before the start of endodontic therapy and restorative phase. The following conditions are to be considered as critical for treatment success:

- Healthy gingival tissue
- Normal bone architecture and attachment levels to favor periodontal health

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- Maintenance of biologic width and ferrule effect before and after endodontic and restorative phases

If one or more of the aforementioned conditions are not met, owing to preexisting pathology or structural defects, treatment success or even feasibility can be compromised, sometimes suggesting extraction of weak teeth and replacement with dental implants rather than conventional therapy.

Biomechanical evaluation

All previous events, from initial decay or trauma to final root canal therapy, influence the biomechanical status of the tooth and the selection of restorative materials and procedures. The biomechanical status can even justify the decision to extract extremely mutilated teeth that do not deserve extensive treatments and that carry a limited probability of success. Important clinical factors include the following:

- The amount and quality of remaining tooth structure
- The anatomic position of the tooth
- The occlusal forces on the tooth
- The restorative requirements of the tooth

Teeth with minimal remaining tooth structure are at increased risk for the following clinical complications[114,165,170] (Fig. 23.4; also see Fig. 23.1):

- Root fracture
- Coronal-apical leakage
- Recurrent caries
- Dislodgment or loss of the core/prosthesis
- Periodontal injury from biologic width invasion

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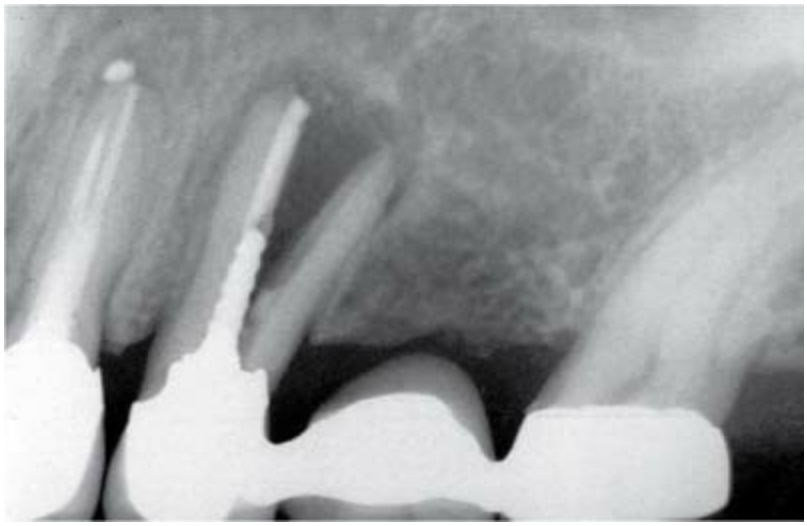

FIG. 23.4 Failure of prosthetic foundations can have dramatic consequences on both overlying restorations and surrounding tissues. A better understanding of compositional and structural changes that affect tooth resistance to repeated functional forces is mandatory to improve treatment success in endodontically treated teeth.

Radiograph shows four teeth. The first tooth has filled root canals with a globular mass close to the root tip. In the second tooth, only one root canal is filled and less-density tissue in-between the root canals. The roots of third tooth are missing. The fourth tooth has radiopaque crown and hard-density root.

The amount and quality of remaining tooth substrate are far more important to the long-term prognosis of the restored tooth than any restorative material properties. One must consider that no restorative material can truly substitute for dentin or enamel and that a minimal amount of intact structure is mandatory to justify tooth maintenance and its strategic importance to the overall treatment plan. The presence of healthy adjacent teeth available as abutments or the option of dental implants are additional factors to be analyzed when justifying the restoration of endodontically treated teeth.

Tooth position, occlusal forces, and parafunctions

Teeth are subjected to cyclic axial and nonaxial forces. The teeth and associated restorations must resist these forces to limit potential damages such as wear or fracture. The degree and direction of forces depend on the location of the tooth in the arch, the occlusal scheme, and the patient's functional status.

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In most occlusal schemes, anterior teeth protect posterior teeth from lateral forces through anterolateral guidance. In the context of very steep anterior guidance and deep vertical overbite, maxillary anterior teeth are sustaining higher protrusive and lateral forces from the mandibular anterior teeth. Restorations of damaged anterior teeth with heavy function should therefore be designed to resist flexion. Restorative components should be stronger than would be required for teeth with an edge-to-edge relationship and therefore vertical forces.

Posterior teeth normally carry more vertical forces, especially when maintaining canine and anterior guidance; they also sustain greater occlusal loads than anterior teeth, and restorations must be planned to protect posterior teeth against fracture. In the case of parafunctions, protection by anterior contacts is likely to be reduced or lost and posterior teeth then submitted to more lateral stresses, generating a higher demand for restorative materials.

The literature reports that average biting forces vary between 25 and 75 N in the anterior region and between 40 and 125 N for the posterior region of the mouth, depending on food type, dental status (dentate or edentulous), and patient anatomy and functional habits.[55,71] Those forces can easily reach 1000 N or above in case of parafunctions, showing how potentially destructive they can be for intact teeth and even more so for nonvital, fragilized teeth. Parafunctional habits (clenching and bruxism) are major causes of fatigue or traumatic injury to teeth, including wear, cracks, and fractures. Teeth that show extensive wear or sequelae from parafunctions, especially heavy lateral function, require components with the highest physical properties to protect restored teeth against fracture.

In general, modern strategy focuses on tissue preservation and on the use of adhesion to stabilize the restoration for improved short- and long-term service. However, in certain conditions like reduced tooth support, conventional materials are not obsolete.

Aesthetic evaluation and requirements

Anterior teeth, premolars, and often the maxillary first molar, along with the surrounding gingiva, compose the aesthetic zone of the mouth. Changes in the color or translucency of the visible tooth structure, along with thin soft tissues or biotype, diminish the chance for a successful aesthetic treatment

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outcome.

Potential aesthetic complications should be investigated before endodontic therapy is initiated. For instance, metal or dark carbon fiber posts or amalgam placed in the pulpal chamber can result in unacceptable aesthetic results, such as a grayish appearance of the overlying prosthetic restoration (especially with modern, more translucent full-ceramic crowns) or gingival discoloration from the underlying cervical area or root (see Fig. 23.1). All teeth located in the aesthetic zone also require critical control of endodontic filling materials in the coronal third of the canal and the pulp chamber to avoid or reduce the risk of discoloration. Careful selection of restorative materials, careful handling of tissues, and timely endodontic intervention are important for preserving the natural appearance of nonvital teeth and gingiva.

Treatment strategy

General principles and guidelines

The post, the core, and their luting or bonding agents together form the *foundation restoration* to support the coronal restoration of endodontically treated teeth. The evolution of foundation restorations has been to diminish invasiveness, to use adhesion rather than macromechanical anchorage, and to eliminate intraradicular components in selected cases. These changing clinical concepts derive from both an improved understanding of tooth biomechanics and advances in restorative materials.

The foundation and its different constituents are then aimed at providing the best protection against leakage-related caries, fracture, or restoration dislodgment. Therefore all aforementioned local and general parameters are to be systematically analyzed in order to select the best treatment approach and restorative materials. Prosthetic requirements are also to be taken into consideration to complete each case analysis. In general, abutments for fixed or removable partial dentures clearly dictate more extensive protective and retentive features than do single crowns, owing to greater transverse and torquing forces. This modern biomechanical treatment strategy is summarized in Fig. 23.5.

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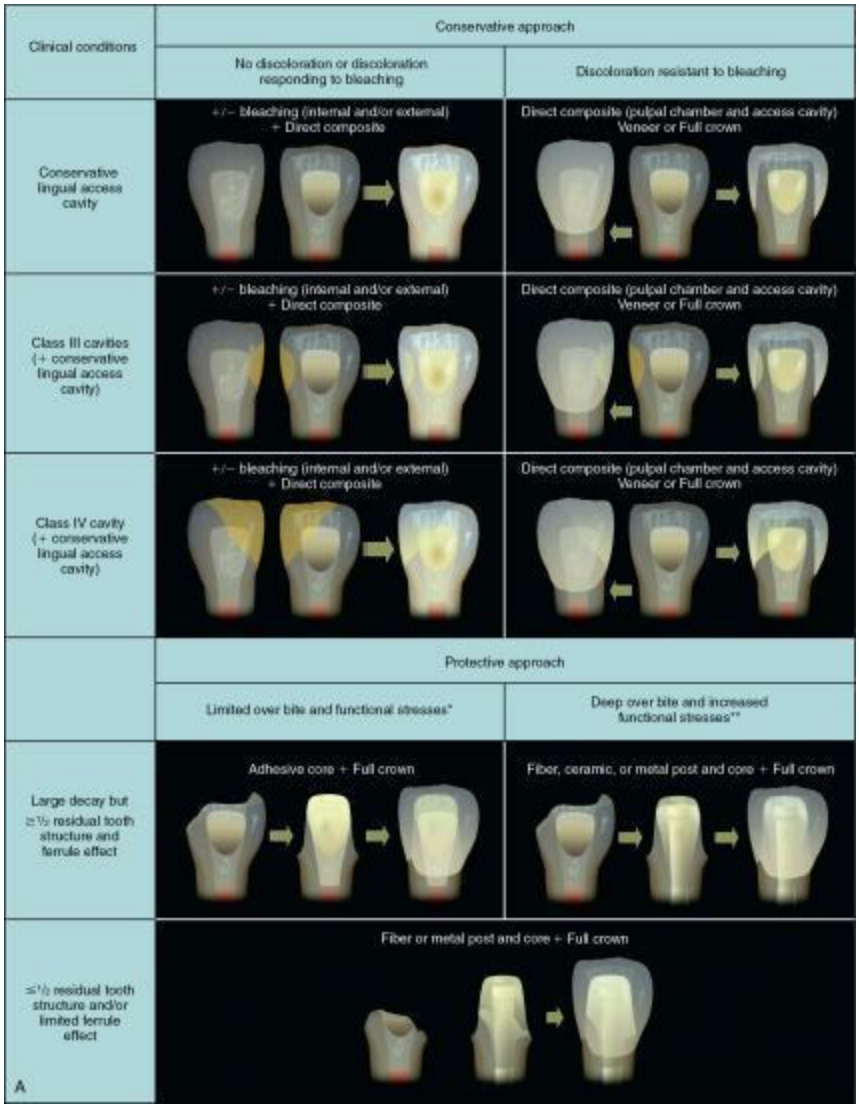

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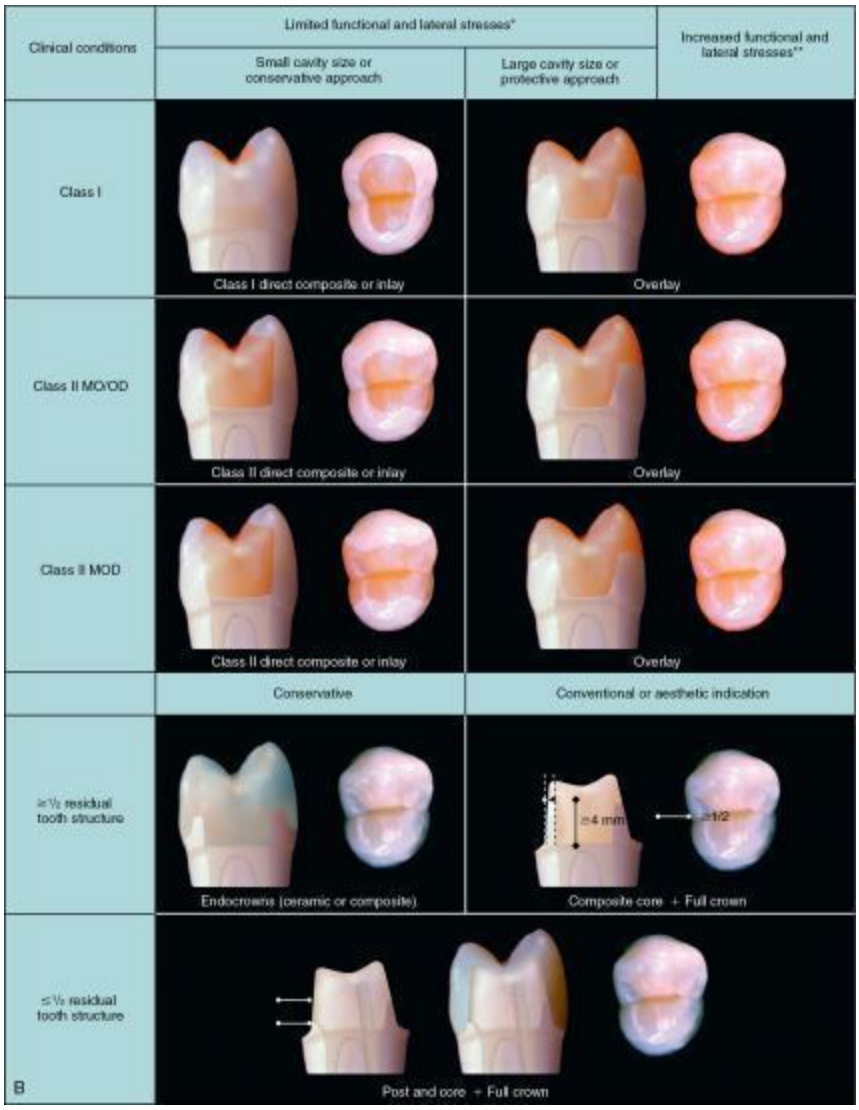

FIG. 23.5 **A,** Current recommendations for the treatment of nonvital anterior teeth. *Normal function and anterior guidance; **moderate to severe parafunctions and abnormal occlusion/anterior guidance. **B,** Current recommendations for the treatment of nonvital posterior teeth. *Relatively flat anatomy and canine guidance, normal function; **group guidance, steep occlusal anatomy, parafunctions.

Figure shows conservative and protective approach for teeth.

Conservative approach: The columns headers are Clinical conditions, No discoloration or discoloration or responding to bleaching, and Discoloration resistant to bleaching. The row headers are Conservative lingual access cavity, Class 3 cavities (conservative lingual access cavity), and Class 4 cavity (conservative lingual access cavity). Each cavity undergoes plus/minus bleaching (internal and/or external plus direct composite) and direct composite (pulpal chamber and access cavity) veneer or full crown.

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Protective approach: The column headers are limited over bite and functional stresses, and deep over bite and increased functional stresses. The row headers are large decay but greater than equal to half residual tooth structure and ferrule effect and smaller than equal to half residual tooth structure and/or limited ferrule effect. Large decay shows adhesive core plus full chamber in the first column and fiber, ceramic, or metal post and core plus full crown in the second column. Limited ferrule effect has fiber or metal post and core plus full crown.

A two-panel figure is as follows:

First panel: The column headers are clinical conditions, Small cavity size or conservative approach and large cavity size or protective approach under limited functional and lateral stresses; Increased functional and lateral stresses. The row headers under clinical conditions are class 1, class 2 MO or OD, and class 2 MOD. Class 1 shows class 1 direct composite or inlay in the first column and overlay in the second column. Class 2 and 3 each show class 2 direct composite or inlay in the first column and overlay in the second column.

Second column: The column headers are conservative and conventional or aesthetic indication. The row headers are greater than or equal to half residual tooth structure and smaller than or equal to residual tooth structure. The first row shows endocrowns (ceramic or composite) in the first column and composite core plus full crown in the second column. The second row shows post and core plus full crown.

Structurally sound anterior teeth

Anterior teeth can lose vitality as a result of a trauma with no or minimal structural damage. They generally do not require a crown, core, or post; restorative treatment is limited to sealing the access cavity and direct composite fillings. Discoloration, whenever present, is addressed by nonvital bleaching, or, for untreatable or relapsing ones, with conservative restorative approaches such as direct or indirect veneers (Table 23.2).

Table 23.2

Clinical Protocols for Restoring Nonvital Teeth With Partial Restorations (Most Likely Procedures)

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		INTERFACE TREATMENT			
Treatment Approach	**Indications**	**Tooth Preparation (Critical Guidelines)**	**Tooth**	**Restoration**	**Restoration Fabrication**
Composite restoration	Minimal tissue loss	None	DBA	—	Direct multilayer
Veneer	Limited tissue loss	≥1 mm Buccal reduction, lingual enamel present, minimal to moderate discoloration only	DBA	1. Sandblasting or etching 2. Silane 3. Bonding resin	CP direct multilayer *or In laboratory:* Etchable CER: fired, pressed, or CAD-CAM
Overlay (composite/ceramics)	Thin remaining walls	Minimum 2 mm occlusal reduction	DBA + composite lining	1. Sandblasting or etching 2. Silane 3. Bonding resin	*In laboratory:* CP: hand-shaped, light or heat cured, CAD-CAMEtchable CER: fired, pressed, or CAD-CAM
Endocrown (composite/ceramics)	Loss of occlusal anatomy	Minimum 2 mm occlusal reduction, extension into pulpal chamber	DBA + composite lining	1. Sandblasting or etching 2. Silane 3. Bonding resin	*In laboratory:* CP: hand-shaped, light or heat cured, CAD-CAMEtchable CER: fired, pressed, or CAD-CAM

CAD-CAM, Computer-aided design/computer-aided machined; *CER,* ceramic; *CP,* composite; *DBA,* dentin bonding agent.

Nonvital posterior teeth with minimal/reduced tissue loss

The loss of vitality in posterior teeth resulting from trauma, decay, or a restorative procedure does not necessarily lead to extreme biomechanical involvement and therefore allows, in certain conditions, for conservative restorations.

Occlusal cavities or mesio/disto-occlusal cavities can be restored with either direct- or indirect-adhesive intracoronal restorations, providing residual walls are thick enough (proximal ridges and buccolingual walls more than 1.5-mm thickness). The three additional clinical factors that must be analyzed to ensure optimal treatment success are the configuration factor (C-factor), cavity volume, and dentin quality. For instance, a large class I cavity with contaminated and sclerotic dentin would clearly be a contraindication to the direct approach, despite the fact that it apparently falls within the indications of direct techniques. Conservative options, however, must always be analyzed under the light of functional and occlusal environment. They can

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only be considered in the absence of parafunctions and with anterior guidance, which limits overall functional loading and lateral or flexural forces. In less favorable biomechanical conditions (e.g., group guidance, steep occlusal anatomy, bruxism, clenching), a protective approach with full occlusal coverage (onlay or overlay) is mandated to minimize the risk of fatigue failures (see Table 23.2).

Structurally compromised teeth

The decision for placing a post as well as the selection of a post system (rigid or nonrigid) depends once again on the amount and quality of remaining tooth structure and the anticipated forces sustained by this tooth (Table 23.3).

Table 23.3

Clinical Protocols for Restoring Nonvital Teeth With Full Prosthetic Restorations (Most Likely Procedures)

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		FOUNDATION			RESTORATION	
Treatment Approach	**Indications**	**Tooth Preparation (Critical Guidelines)**	**Post**	**Core**	**Fabrication**	**Luting**
Composite core	Reduced walls but >½ crown height	Maintain all residual structures >1 mm thickness (after core prep.)	—	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + ceramic post	More than ½ coronal structure lost, reduced wall height	Maintain all residual structures >1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + in vitro fiber post	More than ½ coronal structure lost, reduced wall height	Maintain all residual structures >1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Composite core + metal post	More than 2/3 coronal structure lost, reduced wall height	Maintain all residual structures >1 mm thickness (after core prep.)	Sandblasting or coating/silane + DBA + Dual cement or SA cement	DBA + composite Dual or LC, incremental	*In laboratory:* PFM or full ceramic restoration: slip-casting, pressed, or CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement
Amalgam core (± metal post)	Alternative to composite core with metal post	Maintain all residual structures >1 mm thickness (after core prep.)	No tt + nonadhesive cement or sandblasting/coating/silane + DBA + Dual cement or SA cement	Amalgam placement in retentive tooth preparation	*In laboratory:* PFM restoration	Coating, sandblasting, or etching + silane and Dual or SA cement
Cast gold post and core (± porcelain)	More than ¾ coronal structure lost	Maintain all residual structures >1 mm thickness (after core prep.) Internal walls are divergent	No tt/sandblasting + nonadhesive cement or sandblasting/coating/silane + DBA + Dual cement or SA cement	No tt + nonadhesive cement or DBA + Dual cement or SA cement	*In laboratory:* PFM or full ceramic restoration: Zirconia/CAD-CAM	Coating, sandblasting, or etching + silane and Dual or SA cement

CAD-CAM, Computer-aided design/computer-aided machined; *CER,* ceramic; *DBA,* dentin bonding agent; *Dual,* dual curing; *LC,* light curing; *PFM,* porcelain fused to metal; *SA,* self-adhesive.

In general, rigid posts made of stiff materials (metal and ceramics) are indicated for teeth with minimal tooth structure that rely on the post to hold the core and crown. Because rigid posts flex and bend less than other types of posts, they are supposed to limit movement of the core and possible disruption of the crown margins and cement seal. But one must remember that stiff posts transmit more stresses to the root, next to the post apex, when

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conventionally cemented. An attempt to strengthen a weak root by adding a stiff post can instead make the root weaker as a result of the force concentration behavior of a stiff rod in a more flexible material. Adhesion therefore plays a crucial role, because a well-bonded post can help absorb stresses more evenly throughout the remaining tooth structure. Benefit and increased risk of fissure and fracture must then be appropriately weighed against adhesion potential inside the root and post type, composition, and surface treatment.

In structurally sound teeth, nonrigid posts flex with the tooth under functional forces, reducing the transfer of force to the root and reducing the risk of root fracture. Flexion is of course related to post diameter. In structurally compromised teeth, which lack cervical stiffness from dentin and ferrule effect, excessive post flexion can be detrimental to the marginal seal and prosthesis longevity, so fiber posts are generally contraindicated.

White or translucent fiber posts are generally preferred underneath full-ceramic restorations, whereas stronger black carbon fiber posts, which can reflect through gingiva, tooth structure, or ceramic restorations, are usually used in teeth to be restored with gold or porcelain fused to metal crowns, as well as in zirconia-based restorations. The literature has largely overemphasized the impact of post color on restoration aesthetics. Metal or carbon post color can be masked with resin opaquer and gold post and core ceramized to enhance aesthetic integration. Such procedures can help to approach a more ideal restoration biomechanical behavior through the fabrication of rigid but more aesthetic foundations. Upper lateral and lower incisors, together with extremely thin biotype, are probably the only real aesthetic contraindication for metal or carbon fiber posts.

In cases of extreme tooth fragility secondary to caries, fracture, previous overenlargement of the root canal system, or immaturity, residual root structure can be unified and reinforced with adhesive bonding and composite before placing a normal-diameter post, forming an altogether cohesive unit, as previously described.

In conclusion, in a damaged tooth that is to be restored with a nonrigid post, 2 to 3 mm of cervical tooth structure must ideally remain to allow creation of a restoration as a whole that is resistant to flexion. Teeth with minimal tooth structure and limited ferrule effect need additional cervical stiffness from a more rigid post to resist distortion. In this situation, adhesive

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cementation is preferred to conventional cementation.

Structurally compromised anterior teeth

Restoration of endodontically treated teeth becomes more complex as teeth or supporting structures become increasingly affected. A nonvital anterior tooth that has lost significant tooth structure requires restoration with a crown, supported and retained by a core and possibly a post as well.

When less than half the core height is present, or when remaining walls are extremely thin (<1 mm on more than three-fourths of the tooth circumference), a post is needed to increase retention and stabilize and reinforce the foundation. Many post options are available nowadays, including titanium, fiber-reinforced resin, and ceramics. Adhesion is now the preferred mode of post cementation unless a long-term contamination of root dentin is obvious (e.g., with eugenol), making adhesion highly questionable.

In the latter situation or in the presence of flared canals (possibly as well when a limited ferrule effect is present), cast gold post and core are still considered feasible options. Actually, in this extremely unfavorable biomechanical environment, this traditional treatment approach provides a higher rigidity in the cervical area, which is mandatory for restoration stability. Here, a fiber-reinforced composite foundation having higher flexibility might present less favorable biomechanical behavior, such as suggested by FEM studies (see Fig. 23.3 and Table 23.2).[42] Tooth extraction and implant placement or bonded bridge (particularly for lateral incisors) are also to be considered in this situation.

In the aesthetic zone, the post should not detract from the aesthetics of the coronal tooth structure, ceramic crown, or gingiva. Current restorative procedures allow fabrication of highly aesthetic ceramic coronal restorations that have no metal substructure. When such restorations with remarkably lifelike color and vitality are selected, it usually implies the use of nonmetal aesthetic posts, either ceramic or resin fiber-reinforced ones.

Structurally compromised posterior teeth

Slightly decayed posterior teeth in the context of parafunctions or significantly fragilized premolars and molars require cuspal protection afforded by onlay restoration, endocrown, or a full crown. The need for a

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post and core depends on the amount of remaining tooth structure. When remaining walls (buccal and lingual) provide more than 3 to 4 mm height (from the pulpal chamber floor) and 1.5 to 2 mm thickness, core and restoration stability are granted through macromechanical retention or adhesion; then, posts are not needed (see Fig. 23.5). With current treatment strategy, *the post has become the exception rather than the rule for the restoration of nonvital posterior teeth*.

Additional procedures

Periodontal crown lengthening surgery or orthodontic extrusion can expose additional root structure to allow restoration of a severely damaged tooth. In the smile frame, crown lengthening might, however, be limited by aesthetically adverse consequences (proximal attachment reduction); basically, buccal crown lengthening can only be considered as a potential indication for this procedure. In the posterior region, crown lengthening is limited by tooth and furcation anatomy or by loss of bone structure, which complicates future implant placement. With regard to orthodontic extrusion, root length and anatomy are the limiting factors of this procedure; short roots or conical anatomy are contraindications to orthodontic extrusion. Once again, when a long-lasting, functional restoration cannot be predictably created, it might be better to extract the tooth than to pursue heroic efforts to restore an extremely weak tooth, using complex, expensive, and unpredictable procedures.

Clinical procedures

The restoration of a nonvital tooth may include several restorative components such as the post, the core, and the overlying restoration. Within the same tooth, several interfaces such as post to radicular dentin, core to coronal dentin, core to post, and core to overlying restoration will be created. According to the biomechanical status, some or all restorative components and interfaces will be present and need to be addressed.

General clinical guidelines for restoring endodontically treated teeth with either partial or full crown restorations are presented in Table 23.2, with specific procedures for tooth preparation and treatment of the different interfaces involved. Clinical steps for all recommended treatment options are

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presented in Figs. 23.6–23.14.

FIG. 23.6 Direct composite restorations on front teeth (following bleaching). Nonvital anterior tooth with sufficient amount of residual tissue and intact, healthy neighboring teeth, usually in rather young patients. **A,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha were removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. **B,** Bleaching procedures. Two sessions of internal bleaching were needed to adapt the color of both traumatized anterior teeth (tooth #11), using a mixture of 3% hydrogen peroxide and sodium perborate powder. Each bleaching session extended over a 10-day period. Between sessions and until the given delay to proceed with final restoration (1 to 2 weeks up to 6 to 8 weeks), access cavity is closed with either a temporary filling material or flowable resin composite. **C** and **D,** Restorative procedures. A layer of adhesive is applied over all cavity surfaces. An etch-and-rinse system is frequently preferred when the dentinal tissue is highly sclerotic or was contaminated (e.g., by eugenol). A hard cement such as glass ionomer

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(not a resin-modified glass ionomer because of water uptake and further expansion) was used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. Two or three layers are usually used to replace the missing tissue, preferably following the "natural layering concept," which aims to build up the restoration with two basic masses, namely dentin and enamel, with the help of a silicone index. **E** and **F,** Postoperative views. The two conservative restorations are shown after finishing and polishing, with the rubber dam still in place **(E)** and after several weeks **(F)**. Note that aesthetic integration cannot be evaluated before full rehydration has occurred (4 to 6 hours at least).

Six close-up views of teeth, marked A through F, show the changes taking place during the conversion of yellowish and chipped front teeth into healthy white teeth.

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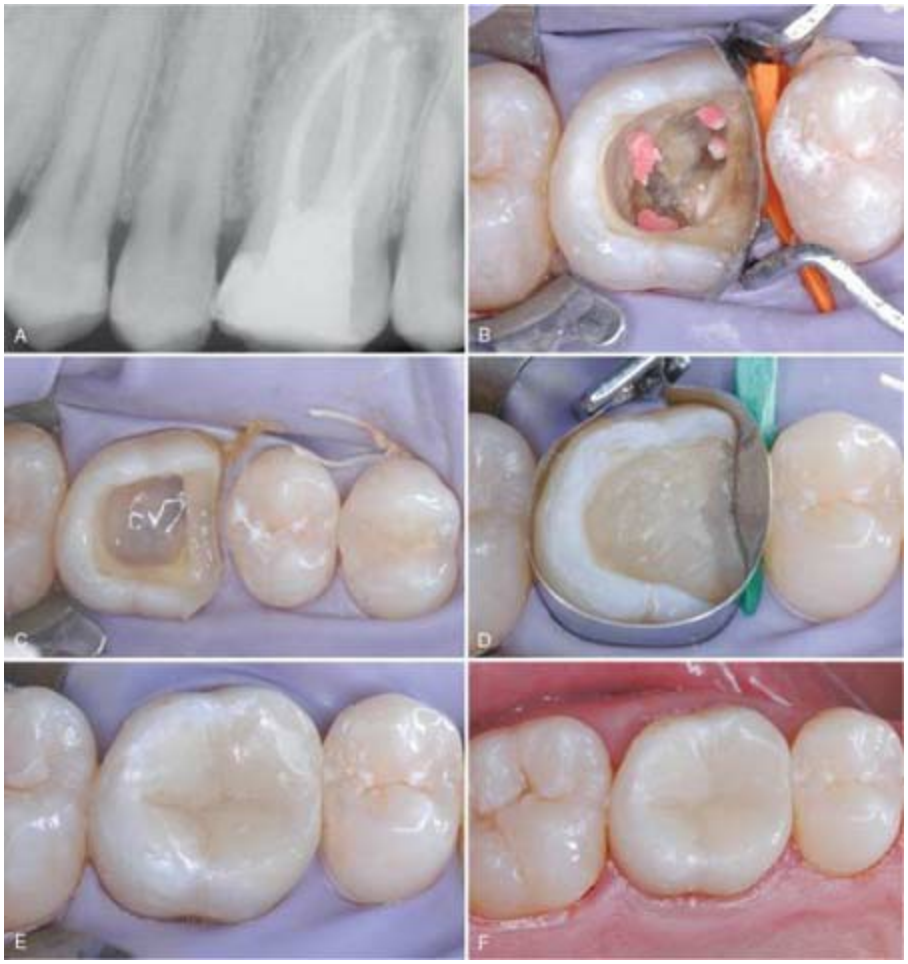

FIG. 23.7 Direct composite restoration on posterior tooth. Nonvital posterior tooth with sufficient amount of residual tissue and mostly intact, rather healthy, neighboring teeth. No significant discoloration of buccal tooth substrate and absence of severe parafunctions or group guidance or other unfavorable occluso-functional condition. **A,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for a later placement of a post. **B,** Adhesive procedure. An etch-and-rinse adhesive system was used here owing to the presence of sclerotic and contaminated dentin. **C** to **E,** Restoration base and layering technique. Resins or glass ionomer cements can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. A dentin composite mass is used to replace missing dentinal tissue, and enamel composite is used to build up proximal walls and the occlusal anatomy. **F,** The postoperative view demonstrates the advantage of this

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conservative approach in the context of teeth with appropriate biomechanical status.

A) Radiograph shows three teeth. The third tooth has radiopaque crown with three filled root canals.

B) Close-up view of teeth isolated with rubber dam clamp shows the central tooth with a large cavity that has pink residues and brownish tissue.

C) The cavity of tooth is clear without any residues and brownish tissue.

D) The tooth with cavity is isolated with rubber dam clamp.

E) The cavity of tooth is filled.

F) Close-up view of jaw shows clean and normal tooth with filled cavity at the center.

FIG. 23.8 Veneer. Nonvital anterior tooth with sufficient amount of residual tissue and rather healthy neighboring teeth. Discoloration

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resisted bleaching or relapsed, despite several retreatments. Preferably thick biotype to limit the risk of gingival recession and loss of aesthetic integration. **A,** Closing the lingual cavity. A hard cement such as glass ionomer (not resin-modified glass ionomer because of water uptake and possible expansion) can be used for dentin replacement, before adding enamel composite to close the lingual cavity. A full resin composite restoration can also be considered to fill in the lingual cavity (dentin + enamel masses) in consideration of the material's hydrophobic nature, which could presumably prevent or limit penetration of water-soluble pigments from the oral cavity. **B,** Preparation. A sufficient amount of buccal tooth structure must be removed to allow for a good aesthetic result (preparation depth is dictated by the discoloration severity). Cervical margins closely follow gingival contours, preventing soft-tissue impingement, but they assume complete coverage of discolored tooth structure. **C** and **D,** Adhesive procedure. Hydrophobic bonding systems are preferred to wet-etched enamel when no dentin is exposed. An etch-and-rinse adhesive system is preferred when dentin is exposed (here, in the presence of significant areas of sclerotic dentin). Restoration. Ceramic is etched and silanated. Just before cementation, a last layer of bonding resin is applied but not light-cured until placement. **E,** Luting technique. A light-curing, highly filled, translucent and fluorescent restorative material is preferably used for cementation. This allows better control of restoration placement and minimal wear and fatigue of the cement layer. **F,** Postoperative view. The postoperative view demonstrates satisfactory aesthetics. This approach is sometimes suitable to meet a patient's high aesthetic demands.

Set of six close-up views, marked A through F, shows the conversion of worn front teeth into normal healthy teeth without any crack by using veneer technique.

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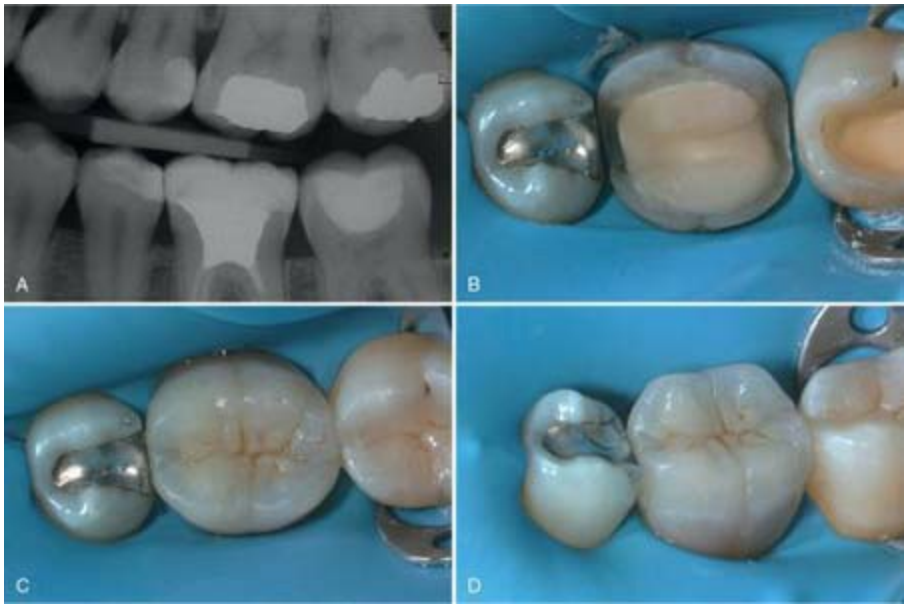

FIG. 23.9 Onlay/overlay. Nonvital posterior tooth with sufficient amount of residual tissue to justify partial restoration. Preparation involves both proximal surfaces or only two surfaces, but in a less favorable occluso-functional environment (thin walls). No significant discoloration of visible buccal tooth substrate. Option is favorable for short clinical crowns. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak occlusal walls are removed until reaching sufficient thickness for the restoration (1.5 to 2 mm in occlusal-free and occlusal areas, respectively). Adhesive procedure: an etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated dentin (a self-etch adhesive system can also be used). A resin composite base is made with a restorative composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **C** and **D,** After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. This treatment option provides optimal tissue conservation, function, and aesthetics and is a good alternative to full crowns.

A) Radiograph shows cavities in teeth.

B) Close-up view shows three teeth isolated with a rubber dam clamp. They are filled with metal, white, and pinkish materials.

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C and D) shows occlusal view of filled cavities.

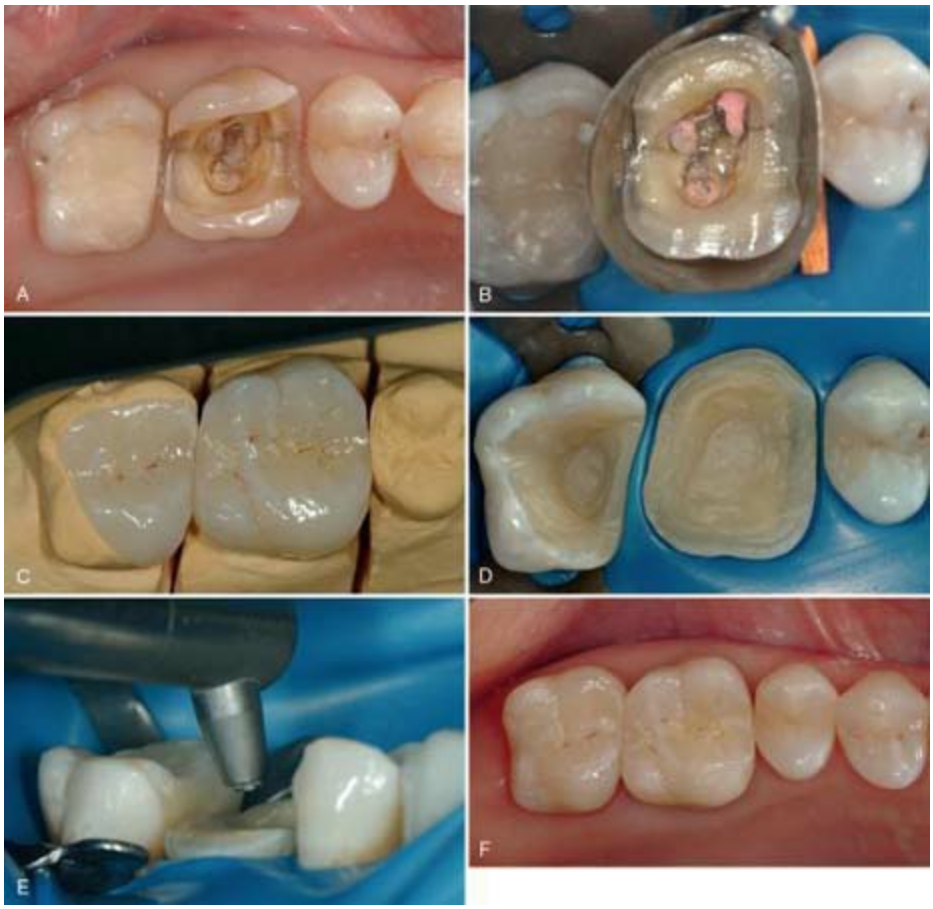

FIG. 23.10 Endocrown. Nonvital posterior tooth with reduced amount of residual cervical tissue but sufficient tissue to provide supragingival restoration margins and good stabilization within former pulpal chamber. No or limited discoloration in visible buccal tooth substrate. Favorable option for short clinical crowns. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Opaque cement can be used to mark the entrance of the roots to facilitate eventual retreatment or for the later placement of a post. Thin and weak walls are removed until reaching sufficient thickness (1.5 mm minimum); as much residual cervical structure as possible is then maintained to avoid restoration interference with periodontal tissues. **C,** Prosthetic restoration on working model. **D,** Restoration base. Adhesive procedures: An etch-and-rinse adhesive system is preferably used here owing to the presence of sclerotic and contaminated dentin (a self-etch adhesive system can also be used). Restoration base: A resin composite base is made with a restorative

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composite or preferably with flowable composite (for limited volume and thickness) to smooth internal angles, fill undercuts, and, when needed, relocate cervical margins occlusally. **E,** Impression and restoration fabrication. After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and aesthetics. The restoration can be made of different tooth-colored materials: ceramics (fired, pressed, or CAD-CAM generated) or resin composite. **F,** Postoperative result. Despite their relatively rare application, endocrowns combine biomechanical advantages with facilitation of clinical procedures and minimal if any involvement of biologic width. It is an interesting alternative to full crowns.

A) Close-up view shows the second tooth with dead and soft tissues in the cavity.

B) The tooth has pink residues along with some fluid in the cavity.

C) Occlusal view of two teeth shows brown lines in pits. The third tooth is covered.

D) Tooth teeth with deep and clear cavities.

E) The second tooth undergoes fabrication.

F) Occlusal view of four teeth with deep lines.

Source: (From Rocca GT, Bouillaguet S: Alternative treatments for the restoration of nonvital teeth, *Rev Odont Stomat* 37:259, 2008.)

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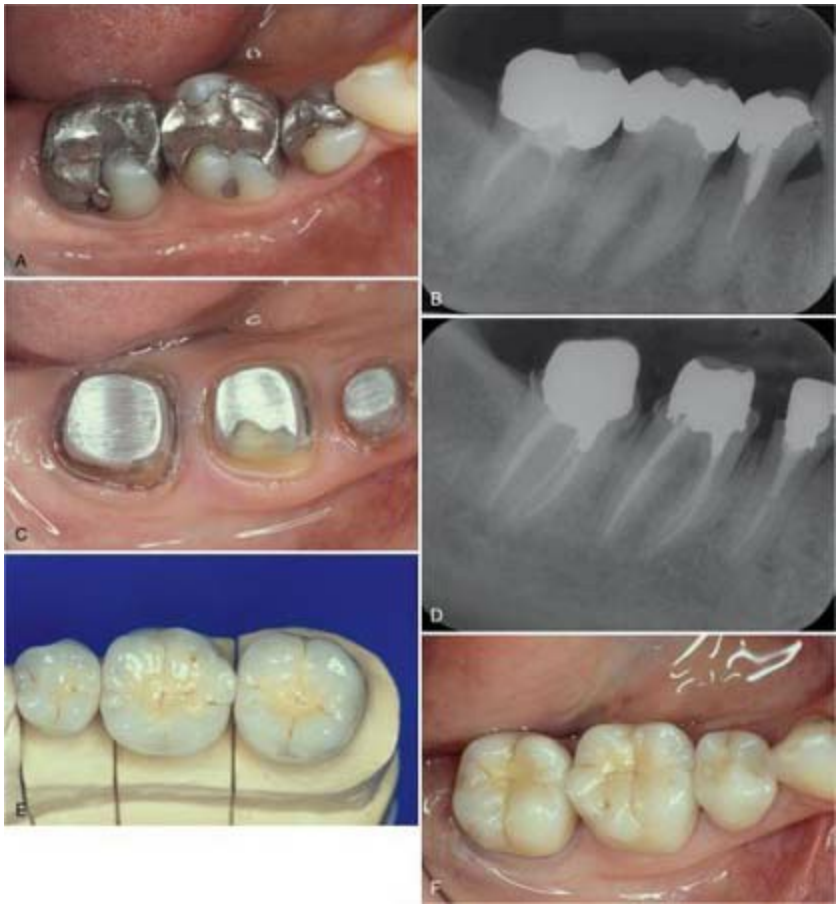

FIG. 23.11 Amalgam core. Nonvital posterior teeth with reduced tooth structure and wall height. Amalgam core stability is granted by extension of the restoration into the coronal portion of the canal (curved roots) and post (only if straight root anatomy) and remaining pulpal chamber undercuts. Today, this option is considered obsolete or historical because of the intrinsic staining of amalgam, the nonadhesive technique, and the less-than-ideal biomechanical behavior of amalgam. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. Thin and weak walls are removed until reaching sufficient thickness (1.5 to 2 mm); existing retentions are used to stabilize the amalgam core, together with a post. The metal post (with passive and anatomic design, preferably made of titanium) extends into the root for about the same length as its coronal extension. **C** to **E,** Restoration fabrication. After placing a matrix or cupper ring to envelop the preparation, amalgam is compacted into the cavity, superior root canal portion, and around the post until the appropriate coronal volume is restored. Preparation of cores is made to manage optimal space for porcelain fused to metal (PFM) crowns. **F,** Posttreatment result. This treatment approach is feasible for teeth already restored with amalgam cores and when PFM restorations are mandated. Satisfactory aesthetics can be attained with intrasulcular

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restoration margins.

A) Close-up view shows teeth with silver fillings.

B) Radiograph shows first and third teeth with partially filled root canals. The central tooth has unfilled root canals.

C) Close-up view shows smooth surface of silver fillings.

D) Radiograph shows three teeth with filled root canals.

E) Occlusal view of teeth where gums are covered.

F) Close-up view of teeth shows yellowish stains at the center with cracks.

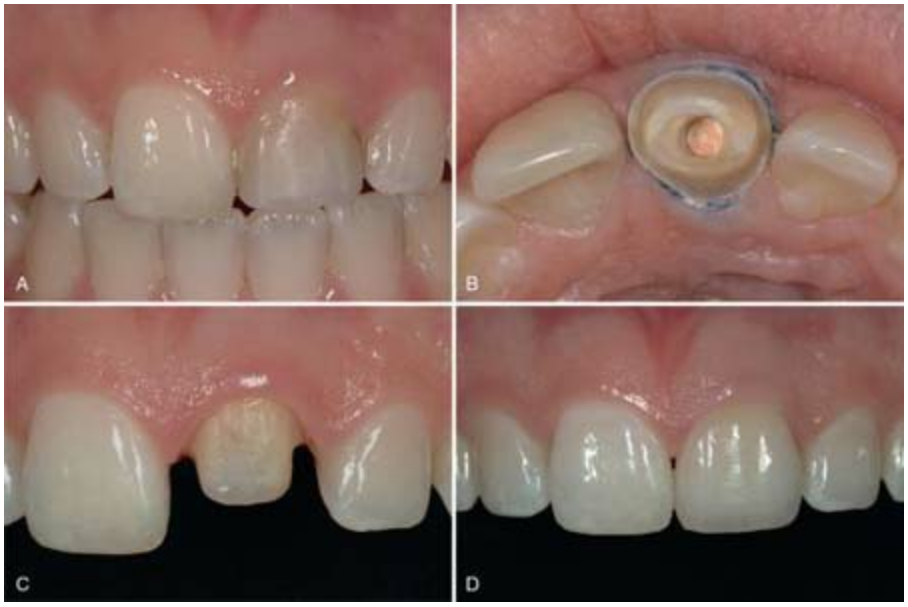

FIG. 23.12 Composite core (without post). Nonvital tooth with more than half the coronal structure left and sufficient wall thickness. Core stability is granted by adhesion, remaining pulpal chamber, and residual wall height. **A** and **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. Half of the abutment height and more than 1.5 mm wall thickness must be present to restore the tooth without a post. If aforementioned conditions are met, a thin layer of hard cement (calcium hydroxide, zinc phosphate, or glass ionomer; not resin-modified glass ionomer) is applied at the entrance of the root canal to enable possible retreatment in the future. **C,** Adhesive procedures and core fabrication. All walls are covered with the adhesive system (etch-

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and-rinse or self-etch) before applying in layers a direct light-curing restorative composite or a self-curing material, in case of limited restoration volume. **D,** Postoperative view. The advantage of this approach is the ease of application and excellent aesthetics. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with limited tissue loss and discoloration.

A) Panoramic view of closed teeth. The second left tooth on the upper jaw is yellowish near the gum line.

B) Occlusal view of three teeth where the second tooth has a cavity at the center.

C) Close-up view of three upper teeth where the second tooth is abnormally small.

D) Close-up view of normal and healthy upper teeth.

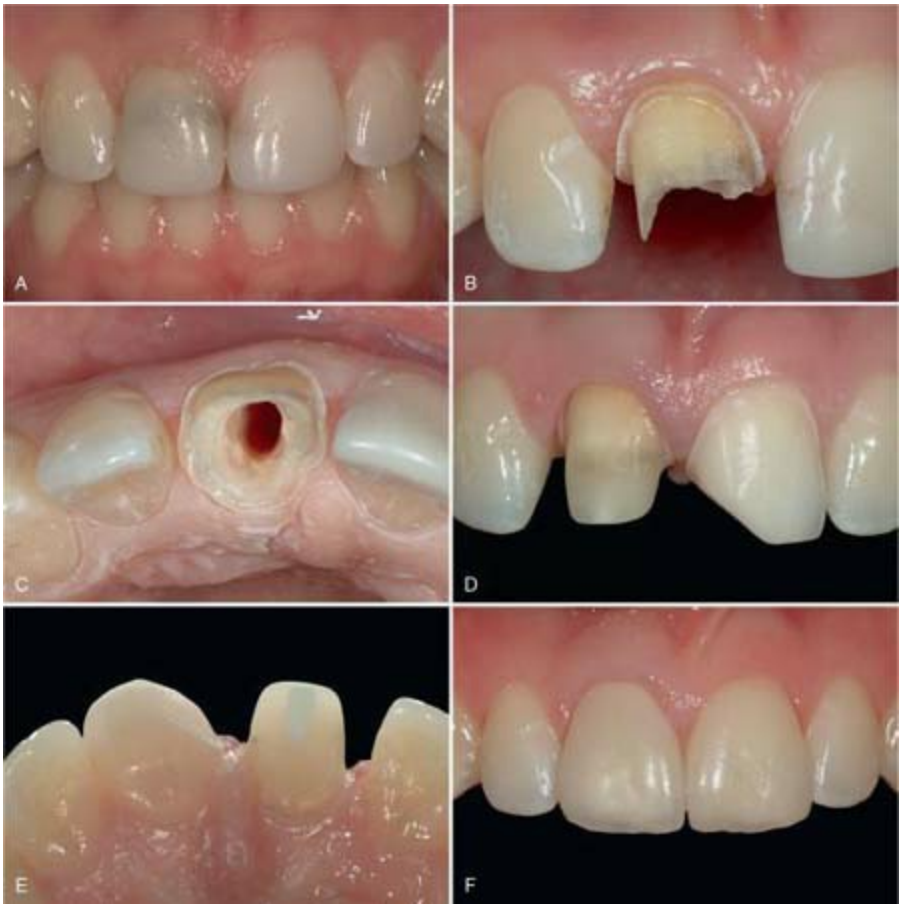

FIG. 23.13 Composite core with white post (resin-reinforced fiber or zirconia post). Nonvital tooth with less than half the coronal structure

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left or insufficient wall thickness. Core stability is improved through adhesion and a tooth-colored post (resin-reinforced fiber or ceramic posts). **A,** Preoperative view. **B** and **C,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose a clean dentin substrate. The tooth is prepared to evaluate precisely the height and thickness of residual tooth structure. If less than half of the abutment height or 1.5-mm wall thickness remain, the abutment requires a post to increase core retention and foundation stability. **D** and **E,** Post-and-core fabrication. Post placement: The post space is prepared with ad hoc instruments until reaching a length equivalent to core height or slightly less depending on remaining wall height. Increased length is not needed, because retention is predominantly achieved through adhesion. Any tooth-colored post can be used, but fiber posts are usually preferred to limit stress buildup into the root structure, even though they provide less rigidity to the foundation. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a direct light-curing restorative composite or with a single increment of a self-curing material when only limited volume is to be completed. **F,** Postoperative view. The primary advantage of this approach is the excellent aesthetic outcome. It is considered a good restorative solution for highly aesthetic ceramic crowns placed on teeth with more tissue loss but moderate discoloration.

Six close-up views are marked A through F.

A) Panoramic view of teeth shows a black stain on the center incisor.

B) Almost half of the crown in the central incisor is broken.

C) Occlusal view of three teeth shows a deep hole in the central tooth.

D) The central incisor on the right is comparatively smaller and has black stains. The central incisor on the left has an oblique fracture on one side.

E) Interior view of jaw shows the fractured edge of the left tooth and filling in the right tooth.

F) Upper jaw shows normal and clean teeth.

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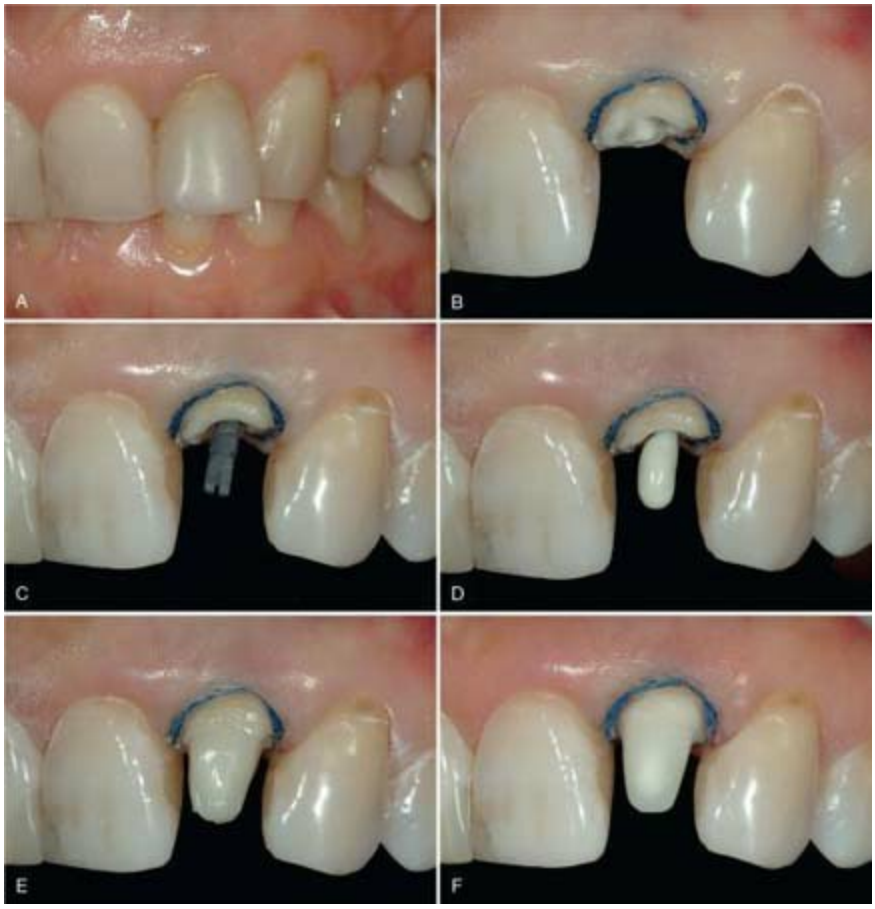

FIG. 23.14 Composite core with prefabricated metal post. Nonvital tooth with reduced coronal structure and ferrule effect or insufficient wall thickness. Core stability is improved through adhesion and a metal (opacified) post (titanium posts are usually preferred). **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. The absence of coronal tissue and ferrule effect mandates the use of a more rigid post (ceramic posts are considered too rigid and contraindicated in this situation). **C** to **F,** Post and core fabrication. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height. More length is not needed, because retention is predominantly achieved through adhesion. Post preparation: The metal post (titanium here) is sandblasted (silicoating can be used to increase adhesion; i.e., Rocatec or CoJet systems, 3M), followed by silane application. An opaque resin or flowable composite can be used to mask metal dark color and improve aesthetics. Post cementation: A self-curing adhesive system is applied on all cavity walls and into post preparation before luting the post with a dual-cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). Core fabrication: The remaining core volume is built up in layers, using a

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direct light-curing restorative composite or a single increment of a self-curing material when only limited volume is to be completed.

Six close-up views are marked A through F.

A) Closed teeth show yellow stains on left incisors.

B) The incisor marked in blue shows dentine tissue.

C) An instrument is implanted into the dentine.

D) The tooth shows an oval structure.

E) The oval structure resembles tooth but with decreased length.

F) It's similar to E, but has smooth edges.

Tooth preparation

The most important part of the restored tooth is the tooth itself. As described earlier, thickness and height of remaining dentin walls or cusps along with functional occlusal conditions are the determining factors in choosing the most appropriate restorative solution.

For partial intracoronal restorations, maximal tissue conservation is the only consideration for the clinician. In other situations, the selected restorative approach will usually necessitate some tooth preparation to comply with restoration design and thickness. Onlays, overlays, and endocrowns require about 1.5 to 2 mm occlusal space to guarantee restoration resistance to functional loads. For full crowns, a ferrule is needed to encircle the vertical walls of sound tooth structure above the restoration margin (1.5 to 3 mm), thus preventing a coincidence between core and restoration limits. Other preparation requirements presented in this chapter are to be respected to ensure treatment success. This means that a 4- to 5-mm height and 1-mm thickness of sound, suprabony tooth structure should be available to accommodate both the periodontal biologic width and the restorative ferrule.

Post placement

The post is an extension of the foundation into the root of structurally damaged teeth, which is necessary for core and coronal restoration stability and retention. The post is cemented or bonded into the root, according to

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tissue quality and post and core choice. The post performs both a mechanical and biologic function by protecting the apical seal from bacterial contamination in case of coronal leakage. These functions should not be attained at the expense of tooth strength; post space preparation should be as conservative as possible to avoid an increased risk of root fracture. It is important to point out again that a post does not strengthen or reinforce a tooth. This elusive goal would only be achieved in a case of perfect cohesion between post and tooth substrate, nowadays only partially achievable. The clinician must therefore keep in mind that inherent strength of the tooth and its resistance to root fracture comes mostly from the remaining tooth structure and the surrounding alveolar bone. The tooth is weakened if dentin is sacrificed to place a larger-diameter post.

The post should be long enough to meet aforementioned biomechanical demands without jeopardizing root integrity. The standard parameters for post placement in a tooth with normal periodontal support are as follows:

1. In case of nonadhesive cementation (metal posts only)[60]:
   - Two-thirds the length of the canal
   - A radicular extension at least equal to the coronal length of the core
   - One half the bone-supported length of the root
2. In case of adhesive cementation (fiber posts)[41,136]:
   - One-third to one half the length of the canal, maximum
   - A radicular extension about the coronal length of the core

The first step for all types of post and core restorations is removal of gutta-percha and endodontic cement from the future post space. This procedure generally is best accomplished by the clinician providing the endodontic service, because that person has a clear knowledge of the size and form of the canal system. The initial post space preparation procedure is similar for most standardized post and core systems. The space, cleared of gutta-percha, has the form of the canal after cleaning and shaping. Proprietary systems are supplied with a series of drills to prepare the internal surface of the canal. The goal of post space formation is to remove little or no dentin from the root canal.

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Adhesive procedures

In general, both self-etch and etch-and-rinse adhesive systems can be used successfully on root dentin, both systems having a well-documented proof of efficacy. However, the use of etch-and-rinse adhesive systems may be advantageous in the presence of highly sclerotic or contaminated dentin, because phosphoric acids will etch dentin more deeply than self-etching primers. When treating the canal in the perspective of post cementation, a dual-curing adhesive system is preferably used to optimize polymerization. Self-adhesive cements have also demonstrated satisfactory performance for post cementation.

Partial restorations

In the case of limited to moderate coronal substance loss, the restorative strategy for endodontically treated front teeth varies, from direct composite restoration using the same layering or application techniques as for vital teeth (possibly with the contribution of intracoronal or extracoronal bleaching) to full veneers, when discoloration is not fully treatable or tends to relapse. From a restorative standpoint, it is only necessary to mention that a 1- to 2-week delay is mandatory prior to adhesively restoring a bleached tooth. For more extensive restoration, it is even advised to wait up to 6 weeks or more (depending on the extent and duration of bleaching treatment) to allow for tooth final color stabilization and to proceed with shade selection.[39] Figs. 23.6 and 23.7 describe the clinical steps involved in the restoration of moderately decayed front teeth using direct composite and bleaching or veneers.

In posterior teeth exhibiting limited to moderate tissue loss, direct to indirect partial or full occlusal restorations are proposed. Their benefit is to take advantage of residual tooth structure to stabilize the restoration and minimize the potential impact of a prosthetic rehabilitation on periodontal tissues. For instance, second molars definitely profit from a conservative approach using adhesively luted intracoronal and full occlusal coverage restorations. An endocrown is a modification of the aforementioned concept, with extension of the restoration into the pulpal chamber as an additional retentive and stabilization structure. Figs. 23.8–23.10 describe the clinical steps involved in the restoration of moderately decayed posterior teeth using

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onlays, overlays, and endocrowns.

Foundation restoration underneath full crowns

If significant coronal substance loss justifies a full-tooth coverage, the strategy for foundation fabrication varies as follows:

- Amalgam core with/without metal post
- Composite core without post
- Composite post with fiber or ceramic post
- Composite with prefabricated metal post
- Cast gold post and core

Amalgam core

A coronal-radicular amalgam foundation may be realized with or without a post (see Fig. 23.11). The amalgam coronal-radicular core without a post is indicated for posterior teeth that have a large pulp chamber and lateral walls at least 4 mm high. This restoration extends slightly into the coronal portion of the canals (1 to 1.5 mm). The core is then retained by a combination of the divergence of the canals and natural undercuts in the pulp chamber. A single, homogeneous material is used for the entire restoration, rather than the dual phases of a conventional preformed post and core.

When shorter lateral walls are present, a post is mandatory to improve core stability and retention. The preferred roots for post placement for molars are the palatal root in the upper jaw and the distal root in the lower jaw. Other roots, thinner and presenting more pronounced curvatures, are normally not indicated for post placement. Coronal-radicular amalgam foundations are not indicated for premolars owing to their smaller dimensions; adhesive foundations or cast gold post and core are preferred.

Cast gold post and core

Cast gold post and core restorations can be fabricated with either direct or indirect techniques.

Direct technique.

In the direct technique, a castable post and core pattern is fabricated in the

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mouth on the prepared tooth. A preformed plastic post pattern is seated in the post space. To gain a path of withdrawal, undercuts are blocked out with resin composite rather than by removing healthy dentin structure. Acrylic resin is added to create a core directly attached to the post pattern. The finished pattern is removed from the tooth and cast in the laboratory.

Indirect technique.

With the indirect technique, a final impression of the prepared tooth and post space is made (Fig. 23.15). As with the direct technique, the path of withdrawal is made by undercut blockout, not by dentin removal. The castable final post and core pattern are fabricated on a die from this impression. The crown margins need not be accurately reproduced at this stage. Proprietary systems provide matched drills, impression posts, and laboratory casting patterns of various diameters. An impression post (preferably a repositionable one) is fitted to the post space, and a final impression is made that captures the form of the remaining coronal tooth structure and picks up the impression post. In the laboratory, a die reproduces the post space and the residual coronal tooth structure for fabrication of the post and core pattern.

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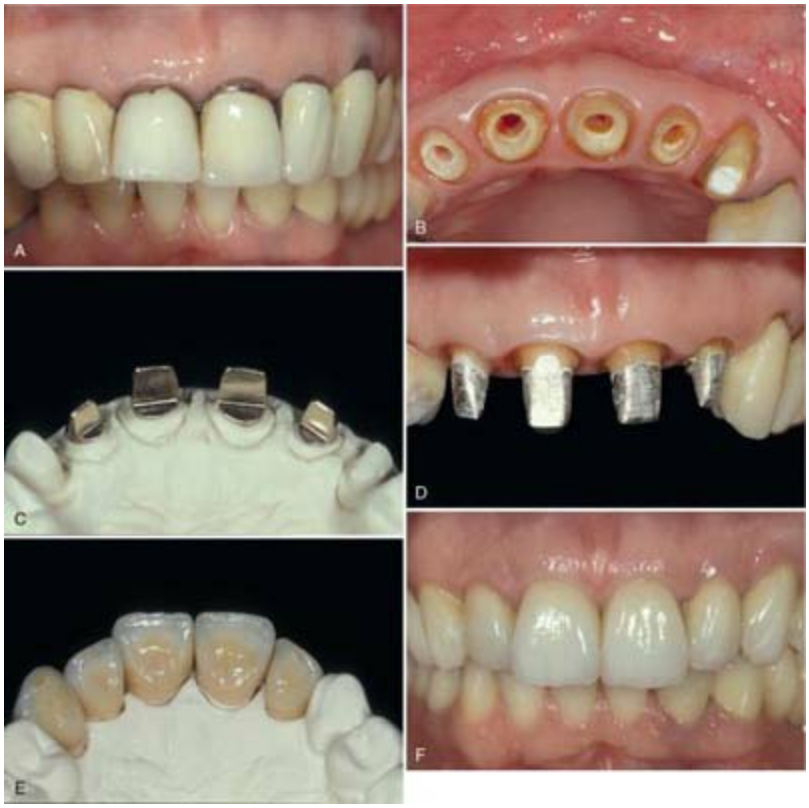

FIG. 23.15 Nonvital teeth with limited coronal structure or numerous abutments. Indirect post and core fabrication helps to achieve proper parallelism and core anatomy. Core retention is assumed by a conventional or adhesive cement. Today, this solution is considered mostly for extended metal-ceramic restorations or sometimes for multiple full-ceramic restorations. **A,** Preoperative view. **B,** Preparation. Existing restorative material and all remnants of endodontic sealer and gutta-percha are removed from the cavity to expose the remaining tooth structure and a clean dentin substrate. Despite the indirect approach, all healthy coronal structures must be maintained to improve foundation stability. Access cavity and coronal walls are prepared with minimal convergence (6 to 10 degrees) to allow for easy insertion. **C** and **D,** Cast gold post and core fabrication and luting. Post space preparation: The post space is prepared with ad hoc instruments until reaching a length equivalent to about core height or more. More length is needed here, as retention cannot always be achieved through adhesion (endodontic retreatment or highly sclerotic contaminated dentin). Then a conventional glass ionomer or zinc phosphate cement is used. Post and core preparation: The gold post is sandblasted (or eventually sandblasted and coated if adhesive procedures are possible) using Rocatec or CoJet systems, 3M + silane. Post cementation: The post and core is luted with the aforementioned conventional cements or adhesive technique. Next, a self-curing adhesive system is applied on all cavity walls and into post preparation (before the insertion of the post and core) and covered with a dual-

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cured composite cement or, as a simple alternative, a self-adhesive cement (then, no adhesive is used). **E** and **F,** Restoration fabrication and placement. A second impression is needed for the final prosthetic work to optimize restoration precision and quality. Lab-made PFM or full ceramic crowns are made with optimal foundation design and alignment.

Six close-up views are marked A through F.

A) Closed teeth show black gum line and yellow stains on the teeth.

B) Upper jaw shows teeth with removed crowns and cavities.

C) Metal tooth-like structures are implanted in the cavities.

D) Upper jaw shows front view of silver teeth.

E) Interior view of upper jaw shows teeth with little yellow stains.

F) Mouth shows restored and normal teeth.

With both techniques, the fabrication of a temporary crown with intraradicular retention is needed. This provisional restoration must stay in the mouth only for a limited time to prevent decementation or canal reinfection. This is why this approach is less popular today and is replaced by direct techniques in most cases.

The cast post and core is cemented at the second appointment. The cementation process must be passive in nature and an evacuation groove made on post side to facilitate cement expulsion and limit seating pressure. Rapid seating, excessive cement, and heavy seating pressure (e.g., occlusal force) can produce high hydraulic pressures inside the root that may be great enough to crack the root.

Crown preparation and temporary restoration

When a coronal restoration is indicated, the amount of tooth structure remaining after final preparation is the most important determinant of the design of the post and core. In addition, the underlying sound tooth structure provides greater resistance to fracture than any post and core type, design, or material. Natural tooth structure should always be carefully preserved during all phases of post space and crown preparation. Otherwise, preparation of endodontically treated teeth is not different from that for vital teeth, with the

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exception of severely discolored teeth, which require a slightly deeper chamfer to hide discoloration with the prosthetic structure and intrasulcular margins to reduce the visibility of a dark cervical tooth structure.

Provisional crowns for endodontically treated teeth must be used only for short periods of time because the loss of cement seal—which is, of course, symptom-free—can lead to leakage, canal reinfection, and even serious carious invasion, thereby jeopardizing the success of endodontic treatment and even resulting in tooth loss.

Summary

Endodontically treated teeth represent a singular situation because of the attendant qualitative and quantitative alterations of the dental substrate. Current literature indicates that treatment success relies on an effective coronal seal that will prevent canal reinfection and an adequate restoration that will resist functional stresses applied to the remaining tooth structure. The purpose of this chapter is to help the clinician make treatment planning decisions concerning the restorative options available for endodontically treated teeth.

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References

1. Akgungor G, Sen D, Aydin M. *Influence of different surface treatments on the short-term bond strength and durability between a zirconia post and a composite resin core material*, J Prosthet Dent 2008;99: 388.
2. Al-Wahadni A, Gutteridge DL. *An in vitro investigation into the effects of retained coronal dentine on the strength of a tooth restored with a cemented post and partial core restoration*, Int Endod J 2002;35: 913.
3. Anusavice KJ Phillips' science of dental materials ed 11 2003; Saunders St. Louis.
4. Aversa R, Apicella D, Perillo L, et al: *Non-linear elastic three-dimensional finite element analysis on the effect of endocrown material rigidity on alveolar bone remodeling process*, Dent Mater 2009;25: 678.
5. Balbosh A, Kern M. *Effect of surface treatment on retention of glass-fiber endodontic posts*, J Prosthet Dent 2006;95: 218.
6. Barkhodar RA, Radke R, Abbasi J. *Effect of metal collars on resistance of endodontically treated teeth to root fracture*, J Prosthet Dent 1989;61: 676.
7. Belli S, Zhang Y, Pereira PN, et al: *Regional bond strengths of adhesive resins to pulp chamber dentin*, J Endod 2001;27: 527.
8. Bergman B, Lundquist P, Sjögren U, et al: *Restorative and endodontic results after treatment with cast posts and cores*, J Prosthet Dent 1989;61: 10.
9. Bindl A, Richter B, Mormann WH. *Survival of ceramic computer-aided design/manufacturing crowns bonded to preparations with reduced macroretention geometry*, Int J

Telegram: @dental_k

www.konkur.in

Prosthodont 2005;18: 219.
10. Bitter K, Paris S, Mueller J, et al: *Correlation of scanning electron and confocal laser scanning microscopic analyses for visualization of dentin/adhesive interfaces in the root canal*, J Adhes Dent 2009;11: 7.
11. Blatz MB, Sadan A, Kern M. *Resin-ceramic bonding: a review of the literature*, J Prosthet Dent 2003;89: 268.
12. Boschian Pest L, Cavalli G, Bertanic P, et al: *Adhesive post-endodontic restorations with fiber posts: push-out tests and SEM observations*, Dent Mater 2002;18: 596.
13. Bouillaguet S, Bertossa B, Krejci I, et al: *Alternative adhesive strategies to optimize bonding to radicular dentin*, J Endod 2007;33: 1227.
14. Bouillaguet S, Troesch S, Wataha JC, et al: *Microtensile bond strength between adhesive cements and root canal dentin*, Dent Mater 2003;19: 199.
15. Bowen RL, Rodriguez MS. *Tensile strength and modulus of elasticity of tooth structure and several restorative materials*, J Am Dent Assoc 1962;64: 378.
16. Bower RC. *Furcation morphology relative to periodontal treatment*, J Periodontol 1979;50: 366.
17. Breschi L, Mazzoni A, Ruggeri A, et al: *Dental adhesion review: aging and stability of the bonded interface*, Dent Mater 2008;24: 90.
18. Butz F, Lennon AM, Heydecke G, et al: *Survival rate and fracture strength of endodontically treated maxillary incisors with moderate defects restored with different post-and-core systems: an in vitro study*, Int J Prosthodont 2001;14: 58.
19. Carrigan PG, Morse DR, Furst L, et al: *A scanning electron microscopic evaluation of human dentinal tubules according to age and location*, J Endod 1984;10: 359.

Telegram: @dental_k

www.konkur.in

20. Carrilho MR, Carvalho RM, de Goes MF, et al: *Chlorhexidine preserves dentin bond in vitro*, J Dent Res 2007;86: 90.
21. Carrilho MR, Tay FR, Donnelly AM, et al: *Host-derived loss of dentin matrix stiffness associated with solubilization of collagen*, J Biomed Mater Res B Appl Biomater 2009;90: 373.
22. Carrilho MR, Tay FR, Pashley DH, et al: *Mechanical stability of resin-dentin bond components*, Dent Mater 2005;21: 232.
23. Cathro PR, Chandler NP, Hood JA. *Impact resistance of crowned endodontically treated central incisors with internal composite cores*, Endod Dent Traumatol 1996;12: 124.
24. Ceballos L, Garrido MA, Fuentes V, et al: *Mechanical characterization of resin cements used for luting fiber posts by nanoindentation*, Dent Mater 2007;23: 100.
25. Deleted in page review.
26. Christensen G. *Core buildup and adhesive incompatibility*, Clin Res Assoc Newsl 2000;24: 6.
27. Christensen G. *Posts: a shift away from metal?*, Clin Res Assoc Newsl 2004;28: 1.
28. Cobankara FK, Unlu N, Cetin AR, et al: *The effect of different restoration techniques on the fracture resistance of endodontically-treated molars*, Oper Dent 2008;33: 526.
29. Cohen BI, Pagnillo MK, Newman I, et al: *Retention of a core material supported by three post head designs*, J Prosthet Dent 2000;83: 624.
30. Combe EC, Shaglouf AM, Watts DC, et al: *Mechanical properties of direct core build-up materials*, Dent Mater 1999;15: 158.
31. Council on Dental Materials, Instruments, and Equipment,

Telegram: @dental_k

www.konkur.in

American Dental Association. *Classification system for cast alloys*, J Am Dent Assoc 1984;109: 766.

32. Craig RG, Peyton FA. *Elastic and mechanical properties of human dentin*, J Dent Res 1958;52: 710.
33. Cruz-Filho AM, Souza-Neto MD, Saquy PC, et al: *Evaluation of the effect of EDTAC, CDTA and EGTA on radicular dentin microhardness*, J Endod 2001;27: 183.
34. Dahl JE, Pallesen U. *Tooth bleaching—a critical review of the biological aspects*, Crit Rev Oral Biol Med 2003;14: 292.
35. D'Arcangelo C, D'Amario M, Vadini M, et al: *Influence of surface treatments on the flexural properties of fiber posts*, J Endod 2007;33: 864.
36. Davidson CL, Feilzer AJ. *Polymerization shrinkage and polymerization shrinkage stress in polymer-based restoratives*, J Dent 1997;25: 435.
37. Deliperi S, Bardwell DN. *Reconstruction of nonvital teeth using direct fiber-reinforced composite resins: a pilot clinical study*, J Adhes Dent 2009;11: 71.
38. Denry IL. *Recent advances in ceramics for dentistry*, Crit Rev Oral Biol Med 1996;7: 134.
39. Dietschi D, Ardu S, Krejci I. *A new shading concept based on natural tooth color applied to direct composite restorations*, Quintessence Int 2006;37: 91.
40. Dietschi D, Ardu S, Rossier-Gerber A, et al: *Adaptation of adhesive post and cores to dentin after in vitro occlusal loading: evaluation of post material influence*, J Adhes Dent 2006;8: 409.
41. Dietschi D, Duc O, Krejci I, et al: *Biomechanical considerations for the restoration of endodontically treated teeth: a systematic review of the literature–Part 1 —Composition and micro- and macrostructure alterations*,

Telegram: @dental_k

www.konkur.in

Quintessence Int 2007;38: 733.
42. Dietschi D, Duc O, Krejci I, et al: *Biomechanical considerations for the restoration of endodontically treated teeth: a systematic review of the literature, Part II —Evaluation of fatigue behavior, interfaces, and in vivo studies*, Quintessence Int 2008;39: 117.
43. Dietschi D, Romelli M, Goretti A. *Adaptation of adhesive posts and cores to dentin after fatigue testing*, Int J Prosthodont 1997;10: 498.
44. Douglas WH. *Methods to improve fracture resistance of teeth*, Vanherle G Smith DC Proceedings of the international symposium on posterior composite resin dental restorative materials 1985; Peter Szulc Utrecht, Netherlands 433.
45. Drummond JL. *In vitro evaluation of endodontic posts*, Am J Dent 2000;13: 5B.
46. Drummond JL, King TJ, Bapna MS, et al: *Mechanical property evaluation of pressable restorative ceramics*, Dent Mater 2000;16: 226.
47. Drummond JL, Toepke TR, King TJ. *Thermal and cyclic loading of endodontic posts*, Eur J Oral Sci 1999;107: 220.
48. Duncan JP, Pameijer CH. *Retention of parallel-sided titanium posts cemented with six luting agents: an in vitro study*, J Prosthet Dent 1998;80: 423.
49. Duret B, Reynaud M, Duret F. *New concept of coronoradicular reconstruction: the Composipost (1)*, Chir Dent Fr 1990;542: 69.
50. Duret B, Reynaud M, Duret F. *Un nouveau concept de reconstitution corono-radiculaire: le composipost (1)*, Chir Dent Fr 1990;540: 131.
51. Feilzer A, De Gee AJ, Davidson CL. *Setting stress in composite resin in relation to configuration of the*

Telegram: @dental_k

www.konkur.in

restoration, J Dent Res 1987;66: 1636.

52. Feilzer A, De Gee AJ, Davidson CL. *Setting stresses in composite for two different curing modes*, Dent Mater 1993;9: 2.
53. Ferrari M, Cagidiaco MC, Goracci C, et al: *Long-term retrospective study of the clinical performance of fiber posts*, Am J Dent 2007;20: 287.
54. Ferrari M, Vichi A, Mannocci F, et al: *Retrospective study of the clinical performance of fiber posts*, Am J Dent Spec No, 2000;13: 9BB.
55. Fontijn-Tekamp FA, Slagter AP, Van Der Bilt A, et al: *Biting and chewing in overdentures, full dentures, and natural dentitions*, J Dent Res 2000;79: 1519.
56. García-Godoy F, Tay FR, Pashley DH, et al: *Degradation of resin-bonded human dentin after 3 years of storage*, Am J Dent 2007;20: 109.
57. Gelb MN, Barouch E, Simonsen RJ. *Resistance to cusp fracture in class II prepared and restored premolars*, J Prosthet Dent 1986;55: 184.
58. Göhring TN, Peters OA. *Restoration of endodontically treated teeth without posts*, Am J Dent 2003;16: 313.
59. Gohring TN, Roos M. *Inlay-fixed partial dentures adhesively retained and reinforced by glass fibers: clinical and scanning electron microscopy analysis after five years*, Eur J Oral Sci 2005;113: 60.
60. Goodacre CJ, Spolnik KJ. *The prosthodontic management of endodontically treated teeth: a literature review, II, Maintaining the apical seal*, J Prosthodont 1995;4: 51.
61. Goracci C, Tavares AU, Fabianelli A, et al: *The adhesion between fiber posts and root canal walls: comparison between microtensile and push-out bond strength measurements*, Eur J Oral Sci 2004;112: 353.

Telegram: @dental_k

www.konkur.in

62. Grandini S, Goracci C, Monticelli F, et al: *SEM evaluation of the cement layer thickness after luting two different posts*, J Adhes Dent 2005;7: 235.
63. Grandini S, Goracci C, Monticelli F, et al: *Fatigue resistance and structural characteristics of fiber posts: three-point bending test and SEM evaluation*, Dent Mater 2005;21: 75.
64. Grigoratos D, Knowles J, Ng YL, et al: *Effect of exposing dentin to sodium hypochlorite on its flexural strength and elasticity modulus*, Int J Endod J 2001;34: 113.
65. Gutmann JL. *The dentin root complex: anatomic and biologic considerations in restoring endodontically treated teeth*, J Prosthet Dent 1992;67: 458.
66. Hagge MS, Wong RD, Lindemuth JS. *Retention strengths of five luting cements on prefabricated dowels after root canal obturation with a zinc oxide/eugenol sealer: 1, Dowel space preparation/cementation at one week after obturation*, Prosthodont 2002;11: 168.
67. Hattab FN, Qudeimat MA, al-Rimawi HS. *Dental discoloration: an overview*, J Esthet Dent 1999;11: 291.
68. Hawkins CL, Davies MJ. *Hypochlorite-induced damage to proteins: formation of nitrogen-centered radicals from lysine residues and their role in protein fragmentation*, Biochem J 1998;332: 617.
69. Helfer AR, Melnick S, Shilder H. *Determination of the moisture content of vital and pulpless teeth*, Oral Surg Oral Med Oral Pathol 1972;34: 661.
70. Herr P, Ciucchi B, Holz J. *Méthode de positionnement de répliques destinée au contrôle clinique des matériaux d'obturation*, J Biol Buccale 1981;9: 17.
71. Hidaka O, Iwasaki M, Saito M, et al: *Influence of clenching intensity on bite force balance, occlusal contact*

Telegram: @dental_k

www.konkur.in

area, and average bite pressure, J Dent Res 1999;78: 1336.

72. Hikita K, Van Meerbeek B, De Munck J, et al: *Bonding effectiveness of adhesive luting agents to enamel and dentin*, Dent Mater 2007;23: 71.
73. Holand W, Rheinberger V, Apel E, et al: *Clinical applications of glass-ceramics in dentistry*, J Mater Sci Mater Med 2006;17: 1037.
74. Hood JAA. *Methods to improve fracture resistance of teeth*, Vanherle G Smith DC Proceedings of the international symposium on posterior composite resin dental restorative materials 1985; Peter Szulc Utrecht, Netherlands 443.
75. Huang TJ, Shilder H, Nathanson D. *Effect of moisture content and endodontic treatment on some mechanical properties of human dentin*, J Endod 1992;18: 209.
76. Huber L, Cattani-Lorente M, Shaw L, et al: *Push-out bond strengths of endodontic posts bonded with different resin-based luting cements*, Am J Dent 2007;20: 167.
77. Hulsmann M, Heckendorf M, Shafers F. *Comparative in-vitro evaluation of three chelators pastes*, Int Endod J 2002;35: 668.
78. Hulsmann M, Heckendorff M, Lennon A. *Chelating agents in root canal treatment: mode of action and indications for their use*, Int Endod J 2003;36: 810.
79. Isidor F, Brondum K. *Intermittent loading of teeth with tapered, individually cast or prefabricated, parallel-sided posts*, Int J Prosthodont 1992;5: 257.
80. Isidor F, Brøndum K, Ravnholt G. *The influence of post length and crown ferrule length on the resistance to cyclic loading of bovine teeth with prefabricated titanium posts*, Int J Prosthodont 1999;12: 78.

Telegram: @dental_k

www.konkur.in

81. Kane JJ, Burgess JO, Summitt JB. *Fracture resistance of amalgam coronal-radicular restorations*, J Prosthet Dent 1990;63: 607.
82. Kawasaki K, Ruben J, Stokroos I, et al: *The remineralization of EDTA-treated human dentine*, Caries Res 1999;33: 275.
83. Khera SC, Goel VK, Chen RCS, et al: *Parameters of MOD cavity preparations: a 3D FEM study*, Oper Dent 1991;16: 42.
84. Kinney JH, Balooch M, Marshall SJ, et al: *Atomic force microscope measurements of the hardness and elasticity of peritubular and intertubular human dentin*, J Biomech Eng 1996;118: 133.
85. Kinney JH, Balooch M, Marshall SJ, et al: *Hardness and Young's modulus of human peritubular and intertubular dentine*, Arch Oral Biol 1996;41: 9.
86. Kinney JH, Marshall SJ, Marshall GW. *The mechanical properties: a critical review and re-evaluation of the dental literature*, Crit Rev Oral Biol Med 2003;14: 13.
87. Kinney JH, Nallab RK, Poplec JA, et al: *Age-related transparent root dentin: mineral concentration, crystallite size, and mechanical properties*, Biomaterials 2005;26: 3363.
88. Krejci I, Lutz F, Füllemann J. *Tooth-colored inlays/overlays, Tooth-colored adhesive inlays and overlays: materials, principles and classification*, Schweiz Monatsschr Zahnmed 1992;102: 72.
89. Kuttler S, McLean A, Dorn S, et al: *The impact of post space preparation with Gates-Glidden drills on residual dentin thickness in distal roots of mandibular molars*, J Am Dent Assoc 2004;135: 903.
90. Lambjerg-Hansen H, Asmussen E. *Mechanical properties*

Telegram: @dental_k

www.konkur.in

of endodontic posts, J Oral Rehabil 1997;24: 882.

91. Lang H, Korkmaz Y, Schneider K, et al: *Impact of endodontic treatments on the rigidity of the root*, J Dent Res 2006;85: 364.
92. Larsen TD, Douglas WH, Geistfeld RE. *Effect of prepared cavities on the strength of teeth*, Oper Dent 1981;6: 2.5.
93. Lertchirakarn V, Palamara JE, Messer HH. *Anisotropy of tensile strength of root dentin*, J Dent Res 2001;80: 453.
94. Lewinstein I, Grajower R. *Root dentin hardness of endodontically treated teeth*, J Endod 1981;7: 421.
95. Libman WJ, Nicholls JI. *Load fatigue of teeth restored with cast posts and cores and complete crowns*, Int J Prosthodont 1995;8: 155.
96. Linn J, Messer HH. *Effect of restorative procedures on the strength of endodontically treated molars*, J Endod 1994;20: 479.
97. Magne P, Knezevic A. *Simulated fatigue resistance of composite resin versus porcelain CAD/CAM overlay restorations on endodontically treated molars*, Quintessence Int 2009;40: 125.
98. Marchi GM, Mitsui FH, Cavalcanti AN. *Effect of remaining dentine structure and thermal-mechanical aging on the fracture resistance of bovine roots with different post and core systems*, Int Endod J 2008;41: 969.
99. Martinez-Insua A, da Silva L, Rilo B, et al: *Comparison of the fracture resistances of pulpless teeth restored with a cast post and core or carbon fiber post with a composite core*, J Prosthet Dent 1998;80: 527.
100. McLean A. *Criteria for the predictably restorable endodontically treated tooth*, J Can Dent Assoc 1998;64: 652.

Telegram: @dental_k

www.konkur.in

101. Meredith N, Sheriff M, Stechell DJ, et al: *Measurements of the microhardness and Young's modulus of human enamel and dentine using an indentation technique*, Arch Oral Biol 1996;41: 539.
102. Millstein PL, Ho J, Nathanson D. *Retention between a serrated steel dowel and different core materials*, J Prosthet Dent 1991;65: 480.
103. Milot P, Stein RS. *Root fracture in endodontically treated teeth related to post selection and crown design*, J Prosthet Dent 1992;68: 428.
104. Miyasaka K, Nakabayashi N. *Combination of EDTA conditioner and Phenyl-P/HEMA self-etching primer for bonding to dentin*, Dent Mater 1999;15: 153.
105. Monticelli F, Ferrari M, Toledano M. *Cement system and surface treatment selection for fiber post luting*, Med Oral Patol Oral Cir Bucal 2008;13: E214.
106. Morgano SM, Brackett SE. *Foundation restorations in fixed prosthodontics: current knowledge and future needs*, J Prosthet Dent 1999;82: 643.
107. Morris MD, Lee KW, Agee KA, et al: *Effect of sodium hypochlorite and RC-Prep on bond strengths of resin cement to endodontic surfaces*, J Endod 2001;27: 753.
108. Moszner N, Salz U, Zimmermann J. *Chemical aspects of self-etching enamel-dentin adhesives: a systematic review*, Dent Mater 2005;21: 895.
109. Deleted in page review.
110. Muller HP, Eger T. *Gingival phenotypes in young male adults*, J Clin Periodontol 1997;24: 65.
111. Muller HP, Eger T. *Masticatory mucosa and periodontal phenotype: a review*, Int J Periodontics Restorative Dent 2002;22: 172.
112. Nakano F, Takahashi H, Nishimura F. *Reinforcement*

Telegram: @dental_k

www.konkur.in

mechanism of dentin mechanical properties by intracanal medicaments, Dent Mater J 1999;18: 304.

113. Nayyar A, Zalton RE, Leonard LA. *An amalgam coronal-radicular dowel and core technique for endodontically treated posterior teeth*, J Prosthet Dent 1980;43: 511.
114. Nicopoulou-Karayianni K, Bragger U, Lang NP. *Patterns of periodontal destruction associated with incomplete root fractures*, Dentomaxillofac Radiol 1997;26: 321.
115. Nikaido T, Takano Y, Sasafuchi Y, et al: *Bond strengths to endodontically-treated teeth*, Am J Dent 1999;12: 177.
116. Nikiforuk G, Sreebny L. *Demineralization of hard tissues by organic chelating agents at neutral pH*, J Dent Res 1953;32: 859.
117. Nissan J, Dmitry Y, Assif D. *The use of reinforced composite resin cement as compensation for reduced post length*, J Prosthet Dent 2001;86: 304.
118. Olsson M, Lindhe J. *Periodontal characteristics in individuals with varying form of the upper central incisors*, J Clin Periodontol 1991;18: 78.
119. Orstavik D Pitt Ford T Essential endodontology: prevention and treatment of apical periodontitis ed 2 2008; Munksgaard Blackwell.
120. Ottl P, Hahn L, Lauer HCH, et al: *Fracture characteristics of carbon fibre, ceramic and non-palladium endodontic post systems at monotonously increasing loads*, J Oral Rehabil 2002;29: 175.
121. Palamara JE, Wilson PR, Thomas CD, et al: *A new imaging technique for measuring the surface strains applied to dentine*, J Dent 2000;28: 141.
122. Panitvisai P, Messer HH. *Cuspidal deflection in molars in relation to endodontic and restorative procedures*, J Endod 1995;21: 57.

Telegram: @dental_k

www.konkur.in

123. Papa J, Cain C, Messer HH. *Moisture content of vital vs endodontically treated teeth*, Endod Dent Traumatol 1994;10: 91.
124. Pashley D, Okabe A, Parham P. *The relationship between dentin microhardness and tubule density*, Endod Dent Traumatol 1985;1: 176.
125. Pashley DH, Tay FR, Yiu C, et al: *Collagen degradation by host-derived enzymes during aging*, J Dent Res 2004;83: 216.
126. Perdigão J, Lopes MM, Gomes G. *Interfacial adaptation of adhesive materials to root canal dentin*, J Endod 2007;33: 259.
127. Phark JH, Duarte S Jr, Blatz M, et al: *An in vitro evaluation of the long-term resin bond to a new densely sintered high-purity zirconium-oxide ceramic surface*, J Prosthet Dent 2009;101: 29.
128. Pierrisnard L, Hohin F, Renault P, et al: *Coronoradicular reconstruction of pulpless teeth: a mechanical study using finite element analysis*, J Prosthet Dent 2002;88: 442.
129. Pilo R, Cardash HS, Levin E, et al: *Effect of core stiffness on the in vitro fracture of crowned, endodontically treated teeth*, J Prosthet Dent 2002;88: 302.
130. Plotino G, Grande NM, Bedini R, et al: *Flexural properties of endodontic posts and human root dentin*, Dent Mater 2007;23: 1129.
131. Plotino G, Grande NM, Pameijer CH, et al: *Nonvital tooth bleaching: a review of the literature and clinical procedures*, J Endod 2008;34: 394.
132. Pontius O, Hutter JW. *Survival rate and fracture strength of incisors restored with different post and core systems and endodontically treated incisors without coronoradicular reinforcement*, J Endod 2002;28: 710.

Telegram: @dental_k

www.konkur.in

133. Poolthong S, Mori T, Swain MV. *Determination of elastic modulus of dentin by small spherical diamond indenters*, Dent Mater 2001;20: 227.
134. Powers JM, Sakaguchi RL. Craig's restorative dental materials ed 12 2006; Mosby St. Louis.
135. Radovic I, Monticelli F, Goracci C, et al: *Self-adhesive resin cements: a literature review*, J Adhes Dent 2008;10: 251.
136. Reeh ES, Douglas WH, Messer HH. *Stiffness of endodontically treated teeth related to restoration technique*, J Dent Res 1989;68: 540.
137. Reeh ES, Messer HH, Douglas WH. *Reduction in tooth stiffness as a result of endodontic and restorative procedures*, J Endod 1989;15: 512.
138. Rees JS, Jacobsen PH, Hickman J. *The elastic modulus of dentine determined by static and dynamic methods*, Clin Mater 1994;17: 11.
139. Ricketts DN, Tait CM, Higgins AJ. *Post and core systems, refinements to tooth preparation and cementation*, Br Dent J 2005;198: 533- review.
140. Rivera EM, Yamauchi M. *Site comparisons of dentine collagen cross-links from extracted human teeth,* Arch Oral Biol 1993;38: 541.
141. Roberts HW, Leonard DL, Vandewalle KS, et al: *The effect of a translucent post on resin composite depth of cure*, Dent Mater 2004;20: 617.
142. Rocca GT, Bouillaguet S. *Alternative treatments for the restoration of non-vital teeth*, Rev Odont Stomat 2008;37: 259.
143. Rosentritt M, Furer C, Behr M, et al: *Comparison of in vitro fracture strength of metallic and tooth-coloured posts and cores*, J Oral Rehabil 2000;27: 595.

Telegram: @dental_k

www.konkur.in

144. Rosentritt M, Plein T, Kolbeck C, et al: *In vitro fracture force and marginal adaptation of ceramic crowns fixed on natural and artificial teeth*, Int J Prosthodont 2000;13: 387.
145. Ruddle CJ. *Nickel-titanium rotary instruments: current concepts for preparing the root canal system*, Aust Endod J 2003;29: 87.
146. Saleh AA, Ettman WM. *Effect of endodontic irrigation solutions on microhardness of root canal dentin*, J Dent 1999;27: 43.
147. Santos J, Carrilho M, Tervahartiala T, et al: *Determination of matrix metalloproteinases in human radicular dentin*, J Endod 2009;35: 686.
148. Saunders WP, Saunders EM. *Coronal leakage as a cause of failure in root-canal therapy: a review*, Endod Dent Traumatol 1994;10: 105.
149. Sauro S, Mannocci F, Toledano M, et al: *EDTA or H3PO4/NaOCl dentine treatments may increase hybrid layers' resistance to degradation: a microtensile bond strength and confocal-micropermeability study*, J Dent 2009;37: 279.
150. Schmage P, Pfeiffer P, Pinto E, et al: *Influence of oversized dowel space preparation on the bond strengths of FRC posts*, Oper Dent 2009;34: 93.
151. Schwartz RS. *Adhesive dentistry and endodontics, Part 2: bonding in the root canal system—the promise and the problems: a review*, J Endod 2006;32: 1125.
152. Schwartz RS, Robbins JW. *Post placement and restoration of endodontically treated teeth: a literature review*, J Endod 2004;30: 289.
153. Scotti R, Malferrari S, Monaco C. *Clarification on fiber posts: prosthetic core restoration, pre-restorative*

Telegram: @dental_k

www.konkur.in

endodontics, Proceedings from the 6th International Symposium on Adhesive and Restorative Dentistry 2002; 7.

154. Sedgley CM, Messer HH. *Are endodontically treated teeth more brittle?*, J Endod 1992;18: 332.
155. Setcos JC, Staninec M, Wilson NH. *Bonding of amalgam restorations: existing knowledge and future prospects*, Oper Dent 2000;25: 121- review.
156. Signore A, Benedicenti S, Kaitsas V, et al: *Long-term survival of endodontically treated, maxillary anterior teeth restored with either tapered or parallel-sided glass-fiber posts and full-ceramic crown coverage*, J Dent 2009;37: 115.
157. Sim TP, Knowles JC, Ng YL, et al: *Effect of sodium hypochlorite on mechanical properties of dentine and tooth surface strain*, Int Endod J 2001;33: 120.
158. Sindel J, Frandenberger R, Kramer N, et al: *Crack formation in all-ceramic crowns dependent on different core build-up and luting materials*, J Dent 1999;27: 175.
159. Sirimai S, Riis DN, Morgano SM. *An in vitro study of the fracture resistance and the incidence of vertical root fracture of pulpless teeth restored with six post-and-core systems*, J Prosthet Dent 1999;81: 262.
160. Soares CJ, Soares PV, de Freitas Santos-Filho PC, et al: *The influence of cavity design and glass fiber posts on biomechanical behavior of endodontically treated premolars*, J Endod 2008;34: 1015.
161. Sorensen JA, Engelman MJ. *Ferrule design and fracture resistance of endodontically treated teeth*, J Prosthet Dent 1990;63: 529.
162. Sorensen JA, Martinoff MD. *Intracoronal reinforcement and coronal coverage: a study of endodontically treated*

Telegram: @dental_k

www.konkur.in

teeth, J Prosthet Dent 1984;51: 780.

163. Standlee JP, Caputto AA, Holcomb JP. *The Dentatus screw: comparative stress analysis with other endodontic dowel designs*, J Oral Rehabil 1982;9: 23.
164. Summit JB, Robbins JW, Schwart RS. Fundamentals of operative dentistry—a contemporary approach ed 3 2006; Quintessence Hanover Park, IL.
165. Tamse A, Fuss Z, Lustig J. *An evaluation of endodontically treated vertically fractured teeth*, J Endod 1999;25: 506.
166. Tay FR, Loushine RJ, Lambrechts P, et al: *Geometric factors affecting dentin bonding in root canals: a theoretical modeling approach*, J Endod 2005;31: 584.
167. Tidmarsch BG, Arrowsmith MG. *Dentinal tubules at the root ends of apisected teeth: a scanning electron microscopy study*, Int Endod J 1989;22: 184.
168. Tjan AH, Nemetz H. *Effect of eugenol-containing endodontic sealer on retention of prefabricated posts luted with adhesive composite resin cement*, Quintessence Int 1992;23: 839.
169. Trope M, Ray HL. *Resistance to fracture of endodontically treated roots*, Oral Surg Oral Med Oral Pathol 1992;73: 99.
170. Vire DE. *Failure of endodontically treated teeth: classification and evaluation*, J Endod 1991;17: 338.
171. Watanabe I, Saimi Y, Nakabayashi N. *Effect of smear layer on bonding to ground dentin—relationship between grinding conditions and tensile bond strength*, Shika Zairyo Kikai 1994;13: 101.
172. Wiegand A, Buchalla W, Attin T. *Review on fluoride-releasing restorative materials—fluoride release and uptake characteristics, antibacterial activity and influence*

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on caries formation, Dent Mater 2007;23: 343.

173. Wu MK, Pehlivan Y, Kontakiotis EG, et al: *Microleakage along apical root fillings and cemented posts*, J Prosthet Dent 1998;79: 264.

174. Yoldas O, Alaçam T. *Microhardness of composites in simulated root canals cured with light transmitting posts and glass-fiber reinforced composite posts*, J Endod 2005;31: 104.

175. Yoshikawa T, Sano H, Burrow MF, et al: *Effects of dentin depth and cavity configuration on bond strength*, J Dent Res 1999;78: 898.

176. Yüzügüllü B, Ciftçi Y, Saygili G, et al: *Diametral tensile and compressive strengths of several types of core materials*, J Prosthodont 2008;17: 102.

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24: Vital pulp therapy

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CHAPTER OUTLINE

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"There is nothing permanent except change."

—Heraclitus

Vital pulp therapy aims to preserve and maintain pulpal health in teeth challenged by trauma, caries, restorative procedures, and anatomic anomalies. The treatment can be completed for permanent teeth that show reversible pulpal injuries, and outcomes depend on several factors.[87,192,252] The primary objective is to initiate the formation of new mineralized tissue. This procedure is essential for the preservation of involved immature permanent teeth where root development is incomplete, and maintenance of arch integrity is critical during maxillofacial development.[73]

From the first description of direct pulp capping using gold foil by Philipp Pfaff in 1756, dentistry has recognized the ability of the injured pulp to

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repair.[308] New materials now provide superior sealing properties to protect the pulp from microorganisms and their toxic by-products. Mineral trioxide aggregate (MTA) and other hydraulic calcium silicate cements (CSCs) partnered with advanced treatment strategies have changed the long-held concept that pulp capping after carious exposure should be avoided. The perception that outcomes for direct pulp capping in a carious field are inconsistent is based on traditional protocols and materials that did not generate a favorable environment for hard tissue formation.[168,229,271,382] This has encouraged clinicians towards alternative treatments, such as pulpotomy or pulpectomy, particularly in immature permanent teeth.[74,396] This is further complicated by diagnostic difficulties, because clinical signs, symptoms, and radiographs may not accurately reflect the histologic condition of the pulp (Fig. 24.1).[196] However, based on a better understanding of pulp physiology, caries microbiology, and the inflammatory mechanisms responsible for irreversible changes, teeth with the potential for repair and continued vitality can now be more readily identified and predictably treated.[54]

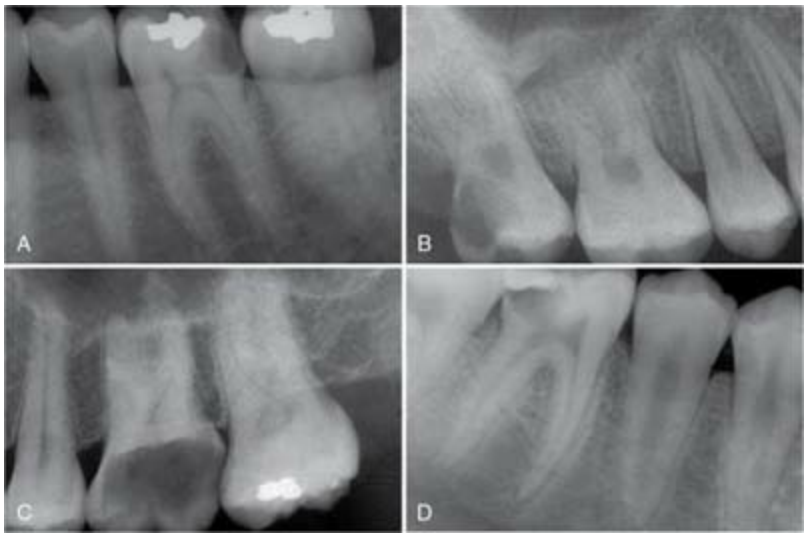

FIG. 24.1 Radiographs of carious molars in patients aged 12 to 38 years. **A,** Mandibular left first molar in a 23-year-old with minor symptoms. **B,** Asymptomatic maxillary right second molar in a 16-year-old. **C,** Asymptomatic maxillary left first molar in a 38-year-old. **D,** Deep caries in the mandibular right first molar of a 12-year-old. All patients were referred to the endodontist for root canal treatment based on radiographic appearance. They exhibited normal vitality with cold testing, and all were treated successfully with vital pulp therapy.

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Four radiographs are marked A through D.

A) Three teeth are shown. Two teeth have radiolucent root canals. One tooth on the left is blurred.

B) Right tooth has radiolucent patch on lateral side of the crown. All teeth have radiolucent root canals.

C) The central tooth has large radiolucent patch covering most of the part of crown.

D) Three teeth are shown with radiolucent root canals. The enamel is removed from one side of the right tooth.

Source: (© Dr. George Bogen.)

Maintaining pulp vitality is in contrast to root canal treatment where remaining pulp tissue is removed and the canal system shaped, disinfected, and obturated using root canal filling materials. Although success rates of over 90% can be achieved after approximately 5 years with careful treatment after vital extirpation the complete loss of function of the pulp tissue occurs, which can be disadvantageous.[21] Proprioceptive protection is partially lost after orthograde treatment, permitting more than double the normal occlusal load before a provoked neuromuscular response occurs.[32,314] This proprioceptive deficit can threaten long-term tooth function and survival. In contrast, vital tooth therapy provides a conservative option that exploits bioactive materials and strategies to save the dentition.

This chapter describes the microbiology of caries and the associated physiologic reactivity of pulp tissue. Understanding that pulp tissue has an innate potential for repair in the absence of bacterial contamination, new treatments in vital pulp therapy for the preservation of the pulpally involved permanent tooth are discussed.[97,201]

The living pulp

Vital pulp tissue comprises cells including fibroblasts, undifferentiated mesenchymal cells, odontoblasts, macrophages, dendritic cells, and other immunocompetent cells. The cells of the subodontoblastic layer and odontoblasts form a thin border between the dentin and the periphery of the pulp known as the *Höhl cell layer*.[106,158] The odontoblasts are elongated

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columnar-shaped cells separated from the mineralized dentin by predentin and characterized by processes that extend into the dentin and possibly to the dentinoenamel junction.[167,348,372,403] Odontoblasts form the mineralized predentin-dentin matrix, which includes phosphoproteins, glycoproteins, proteoglycans, and sialoproteins.[220] Repair mechanisms in the pulp are similar to those in normal connective tissue injured by trauma. When the enamel and dentin are challenged and the pulp is exposed to advancing microorganisms, inflammatory changes can induce pulp necrosis, which precedes problems including infection and its complications.[50,61] The pulp undergoes physiologic, pathologic, and defensive changes during its life.[240,276,381] These include continued dentin apposition, causing reduction of coronal pulp volume and circumference and canal lumen dimensions.[125,277] Atrophy results in fibrosis, dystrophic calcification, degeneration of odontoblasts, and increased cellular apoptosis.[46,125] Aging of human pulp cells is primarily characterized by the formation of reactive oxygen species and senescence-related beta (β)-galactosidase activity.[233] Pain sensitivity is reduced due to a decrease in fast-conducting A-δ fibers and diminished pulp repair, partly attributed to decreases in the levels of substances such as alkaline phosphatase.[254,345]

A comparison analysis of gene expression levels reflecting cell function, proliferation, differentiation, and development found them markedly higher in young pulps.[381] Young dental pulps show greater cell and tissue differentiation, proliferation, and development of the lymphatic, hematologic, and immune systems compared to older pulps where the apoptosis pathway is highly expressed.[381] Although A-β fiber function remains constant with aging, a decrease in A-δ fibers with age may reduce the perception of pain by these fibers.[254]

In summary, the main functions of the dental pulp include dentin formation during development and life of the tooth, the transmission of stimuli via proprio- and pain receptors, and immune responses. The pulp also produces reparative dentin as a defense mechanism against external stimuli, and tissue during the formation and closure of the root apex.

Loss of pulp vitality

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Pulpless teeth with minimal remaining tooth structure that undergo root-canal-filling procedures and are heavily restored become more vulnerable to irreparable fracture because of the loss of proprioceptive mechanisms.[32,262,314,358] Moisture depletion from dentin and reduction of tooth stiffness are minimal after root canal treatment.[186,204,241] Although root canal treatment can prolong tooth survival, the cumulative loss of tooth structure from endodontic and restorative treatment weakens teeth.[64,76,118,393] Changes in root canal geometry are inevitable in the course of canal preparation and may lead to a higher incidence of fractures.[151,235] Other potential problems are tooth discoloration and increased caries risk because of plaque accumulation and altered microflora or due to the absence of the defenses of the pulp-dentin complex.[224,263] Root-canal-filled teeth demonstrate an increased susceptibility to recurrent caries because of poor marginal integrity of restorations or modification of their biologic environment.[78,263] Root canal treatments may be more complex than initially thought, while procedures for maintaining vitality are conservative, easy to implement, and often cost-effective.[198,339]

Pulpal response to caries[a]

The microbial advance in carious lesions is the primary cause of pulpal inflammation and potential necrosis. Acidogenic gram-positive bacteria, predominately oral streptococci and lactobacilli, produce lactic acid as the main metabolic by-product in active lesions that demineralizes enamel and dentin.[136,176] Immune responses and inflammation occur when the caries front is within 1.5 mm of the pulp and bacterial antigens and metabolites diffuse through dentinal tubules.[43,136,203,293] If bacterial challenge continues, immune cell responses lead to increased inflammation and edema, initially characterized clinically by pulpal pain. In the low-compliance environment of the pulp space, inflammation eventually leads to pulp disintegration and apical pathosis.[176,376]

Advancing caries is initially confronted by protective innate immune responses, progressing to an adaptive response when bacteria directly approach the pulp.[175] The pioneer microbes first encounter a positive outward flow of dentinal fluid, characterized by the deposition of immunoglobulins

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and serum proteins that slow the diffusion of antigens.[176] Potent microbial metabolites, such as lipoteichoic acid and lipopolysaccharide, also activate the innate immune system. The by-products stimulate signaling by Toll-like receptors in odontoblasts when these cells first encounter the carious front. They also stimulate proinflammatory cytokines, including interleukin-1, interleukin-8, interleukin-12, tumor necrosis factor alpha, vascular endothelial growth factor (VEGF), and transforming growth factor beta (TGF-β).[137,187,411] VEGF promotes vascular permeability and angiogenesis. Dentin mineralization and matrix metalloproteinase secretion are also induced by the increased expression of TGF-β. Cariogenic bacteria also activate complement pathways and induce the proinflammatory cytokine interferon γ, responsible for killing phagocytosed bacteria by activated macrophages.[190] Odontoblasts also participate in the adaptive response by the synthesis of reactionary dentin, which features diminished tubularity.[86] As microorganisms advance, the modified helical structure in reparative dentin constricts lumen diameter, forming a barrier against pathogens.[85]

As pulpitis progresses, vasoactive neuropeptides contribute to increased vascular permeability and intrapulpal blood flow. Increases in neuropeptide concentration and nerve sprouting characterize neurogenic inflammation, which can cause a transient increase in interstitial tissue pressure and contribute to painful pulpitis.[65,323] Immune cells attempt to control neurogenic inflammation with the secretion of peptides such as somatostatin and β-endorphin.[280] The primary effector cells in innate responses include natural killer cells, neutrophils, monocytes, and macrophages. Immature dendritic cells and T cells contribute to immunosurveillance during the progression of caries. Macrophages eliminate both pathogens and senescent cells while contributing to tissue homeostasis by repairing and remodeling tissue after inflammation.[175]

Cytokines are small, cell-signaling proteins secreted by innate immune cells that induce phagocyte extravasation during inflammation. Chemokines secreted by odontoblasts, fibroblasts, immature dendritic cells, and macrophages stimulate leukocyte recruitment by directing monocyte and neutrophil migration extravascularly to sites of infection.[175] Prompt intervention, through removal of caries and bacterial antigens before irreversible pulpitis commences, can resolve inflammation (Fig. 24.2).

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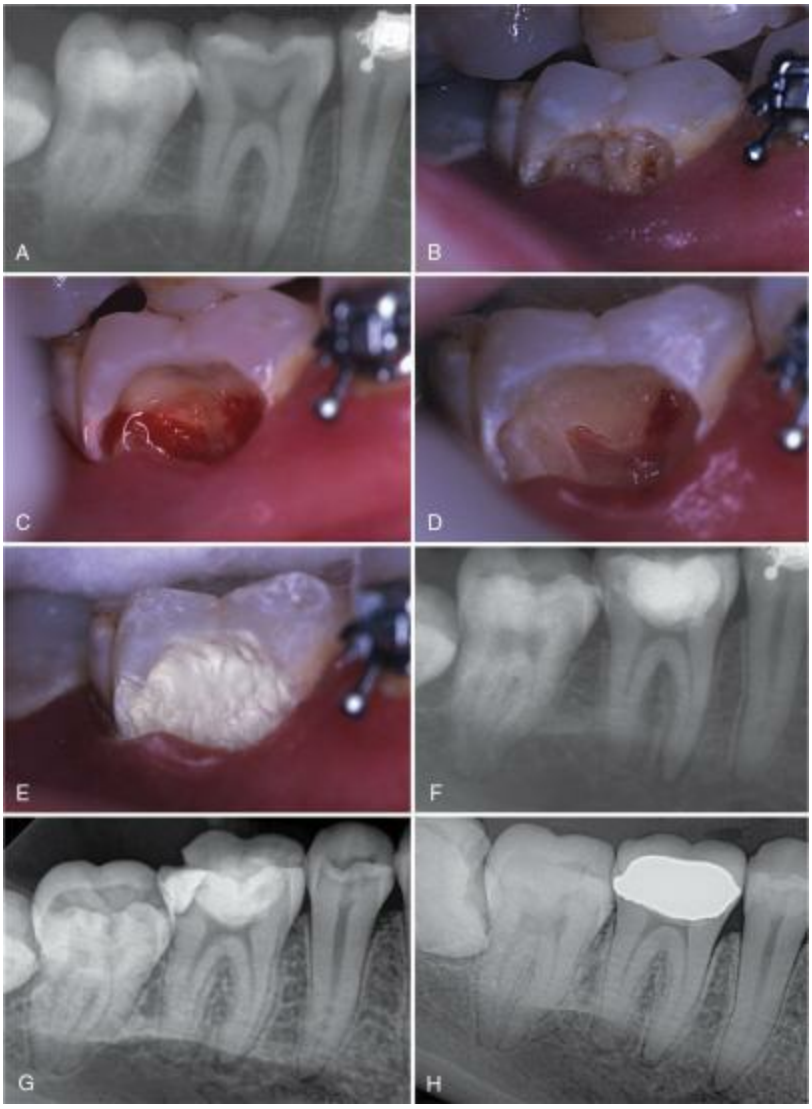

FIG. 24.2 A 15-year-old female patient presented with full orthodontic banding and deep cervical caries of the mandibular right first molar (tooth #30). The tooth was sensitive to cold and biting pressure. **A,** Periapical radiograph revealing large carious lesion and normal periapical structures. **B,** Clinical photograph showing advanced cervical caries extending below crestal gingiva. **C,** View of initial pulp exposure during caries excavation guided by caries detector dye. **D,** Photograph after complete caries removal, large pulp exposure and 6% sodium hypochlorite hemostasis. **E,** Clinical photograph after placement of thick white mineral trioxide aggregate aliquot over entire pulp and surrounding dentin. **F,** Radiograph showing pulp capped molar with temporary restoration (Photocore). **G,** Four-year radiographic control showing recurrent caries and fractured distal marginal ridge tooth #30. **H,** Five-year radiographic review showing no detectable apical pathosis and recently cemented full coverage porcelain fused to metal crown. The molar was asymptomatic and responded normally to sensibility testing.

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Radiograph A shows 30th tooth with radiolucent crown, pulp chamber, and root canal. Three close-up views, marked B to D, show 30th tooth with white soft tissue, blood, and clean cavity, respectively. Close-up view E shows white filling in the cavity. Three radiographs, marked F to H, show gradual changes in the cavity after filling. Radiograph F has rough radiopaque surface in crown, whereas radiograph H has smooth surface.

Source: (© Dr. George Bogen 2019.)

Dentin regeneration and reparative tissue formation

Mammalian dentin can be subdivided into primary, secondary, and tertiary types. Primary dentin is regular, tubular, and develops before eruption. Secondary dentin is also regular and formed throughout life. Tertiary dentin is more or less irregular and forms locally in response to caries or cavity preparation.[228] It is also referred to as irritation dentin. Secondary dentin formation is slower than that of primary dentin as odontoblast activity is presumably downregulated by autophagocytosis after eruption.[95] A mild external stimulus to the odontoblasts leads to upregulated activity and tertiary dentin formation.[350,353] Unlike bone, which is able to adapt to physiological changes throughout life, dentin is neither remodeled nor replaced once lost.[319]

Tertiary dentin formation

Once dentin is exposed by caries, trauma, or cavity preparation, the tubules are opened and odontoblasts injured or even destroyed. If these challenges are of moderate intensity, they lead to a brief inflammation, which subsides when the irritant is removed. Thereafter, tertiary dentin is formed in the affected region.[319,353] Minor trauma to the pulp stimulates undamaged primary odontoblasts to form new dentin.[236,353,354] This tertiary dentin is therefore also referred to as a reactionary dentin, defined as dentin whose matrix is secreted by primary postmitotic odontoblasts in response to an adequate stimulus.[236,353,354]

Injury to the dentin and odontoblast processes leads to a release of growth factors, which in turn leads to the upregulation of odontoblast activity.[350,353]

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The probable control mechanism includes a series of tissue factors or signaling molecules, mainly from the TGF-β family released from the dentin matrix following injury.[156,157] These growth factors can also be secreted by odontoblasts, promoted by acidogenic bacteria, or removed from the dentin by different rinsing solutions used during treatment such as ethylenediaminetetraacetic acid (EDTA), sodium hypochlorite (NaOCl), or citric acid.[352,353] In addition, the application of calcium hydroxide (CH) or silicate cements (e.g., MTA or Biodentine) to dentin or onto pulp tissue leads to TGF-β1 release.[164,232,374] Residual dentin thickness has a significant influence on dentin formation. Even if the dentin above the pulp remains intact, not all odontoblasts will survive. In cavities with a residual dentin thickness of less than 0.25 mm, approximately 50% of cells survive; in shallower cavities, this figure is 85% or more.[281] Despite odontoblast death, hard tissue formation can be observed. The reactionary dentin formation by the primary, postmitotic odontoblasts under these conditions is therefore a specific form of tertiary dentin.

Mineralized tissue formation after loss of the primary odontoblasts

The formation of mineralized tissue after loss of the odontoblasts is more complex than that of reactionary dentin. Dentin formation is not possible without odontoblasts. Nevertheless, after direct capping of a healthy, noninflamed pulp, hard tissue formation occurs. This mineralized tissue is heterogeneous, amorphous, and atubular. Therefore it differs histologically from primary and secondary dentin, but also from reactionary dentin. It is questionable whether this tissue can be called a “dentin bridge,” as it is not dentin from a histological viewpoint. If it really was further formation of dentin, a new type of cell would have to replace the destroyed primary odontoblast. The origin and differentiation of these initial cells remains unclear.[319]

Recent investigations have found no histological evidence for the formation of replacement odontoblasts or new odontoblast-like cells after direct pulp capping (DPC) of human teeth with either CH or MTA.[111,319,320] Secondary odontoblasts or odontoblast-like cells are therefore histologically not detectable in human teeth. It is uncertain whether the hard tissue

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formation is really dentin or just a dystrophic intrapulpal mineralization in response to inflammation.[111,320] However, it is important to emphasize that both reactionary dentin (formed by primary odontoblasts) and hard mineralized repair tissue can form simultaneously in the same lesion.[319,353]

Indications for vital pulp therapy

The penetration of microorganisms and their metabolic products through dentin leads to an inflammation in the pulp. Mediated via cell receptors on odontoblasts, dendritic cells, and pulp fibroblasts, an immune response is initiated. This leads to hyperemia; the reaction is characterized by a decrease in cell number, a flattening of the odontoblasts, and the immigration of lymphocytes and plasma cells.[321] Clinically, this correlates with the development of reversible pulpitis, where healing could be facilitated by therapeutic intervention. If the stimulus persists, bacterial colonization in the pulp chamber can be detected, microabscesses and tissue necrosis lined with polymorphonuclear neutrophilic granulocytes become present, and inflammatory infiltrates are found in the periphery.[321] This is considered to be irreversible pulpitis.

Signs of a reversible pulpitis are a positive response to cold sensibility testing with radiating pain. An irreversible pulpitis is diagnosed on the (enhanced) positive sensibility test, on radiating irritant-persistent pain or lingering pain, pain on heat, and possibly on the patient's inability to localize the problem tooth. However, irreversible pulpitis may be asymptomatic.[14] The correct diagnosis of pulp condition before DPC plays an important role in the outcome. Therefore, assessing the possible the degree of inflammation and/or infection is required.[211] It is challenging to assess pulp health accurately via clinical tests and to distinguish between altered and healthy pulps.[11]

A positive sensibility test (cold) can give a variety of different diagnoses, depending on the severity and duration of response, guiding clinicians to different diagnoses. Teeth responding to cold test may lead to a preoperative diagnosis of normal pulp, reversible pulpitis, or irreversible pulpitis dependent on subjective responses.[301] Sensibility testing does not precisely mirror the condition of the pulp, with 10% to 16% of results being false.[19]

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Besides pain history and sensibility testing, hemorrhage after pulp exposure should be assessed. This may be a more reliable way to determine the status of pulpal infection than clinical signs and symptoms or sensibility testing.[252] Bleeding may reflect the level of inflammation of the pulp, with excessive hemorrhage usually indicating a pulp with a decreased or no chance of recovery.[88] The inflammatory response extends deeper into the pulp tissue when carious dentin is present during exposure, with possible bacterial penetration, compared to superficial inflammation when the pulp is just mechanically exposed.[229]

Pulps with profuse and lingering hemorrhaging had a significantly poorer outcome than those with modest bleeding or bleeding of short duration.[252] Clinically, bleeding should be controlled within 5 to 10 minutes.[211] According to current recommendations in cases of irreversible pulpitis, the healing of the pulp tissue cannot be achieved predictably after removal of the triggering stimulus (e.g., caries). The diagnosis “irreversible pulpitis” often leads to the initiation of pulpectomy and root canal treatment. Although there is evidence that the histological observations described earlier correlate well with the clinical diagnosis, it should be noted that the clinical pattern of the complaint does not indicate the tissue’s ability to regenerate.[321] In a paradigm shift, current clinical investigations using CSCs for full pulpotomies in adult teeth diagnosed with irreversible pulpitis and demonstrating radiographic periapical rarefaction have been treated successfully without pulpectomy and conventional orthograde treatments.[22,23,25,211,242,363,364,386] Refer to the section Pulpotomy With Calcium Silicate Cements.

Maintaining pulp vitality can only be successful if bacterial infection of the tissue during and after the therapy is eliminated. Vital pulp treatments should generally be performed on teeth that do not show pronounced and unprovoked pain symptoms or severe pain on percussion. Vital pulp therapy should not be performed without a response to the sensibility test (in which the pulp status must be verified after pulp exposure) alongside radiographic evidence of periapical disease. In cases diagnosed with irreversible pulpitis, advanced treatment options such as CSC pulpotomy should be considered, especially in younger patients. All vital pulp treatment procedures require a quality bacteria-tight coronal restoration to preclude infection, with dental dam placement, the use of sterile instruments and complete excavation of

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caries. If not, root canal treatment should be given preference or extraction for teeth deemed unrestorable.[107]

Favorable conditions exist in young patients with pulps without previous damage.[334] With increasing age, a reduced ability for repair is expected due to pulpal changes characterized by a cell-poorer and more fibrous pulp tissue.[161,285] Nevertheless, the age of the patient seems to play only a minor role in treatment success.[23,101–103,206,211,242,243,248,258,363] The same applies to factors such as the sex of the patient, tooth position (posterior or anterior), and the size or location of the pulp exposure.[110,206,244,260,301] The success rates of vital pulp therapies in the literature vary widely, especially in the case of DPC after caries excavation. Early clinical failures (within days or weeks) are multifactorial, but certainly correlate with improper pulpal diagnosis or inability to identify and remove necrotic pulp tissue. An inaccurate pulpal diagnosis can underestimate the inflammatory status of the pulp, leading to irreversible pulpitis and pulp necrosis with postoperative pain.

The introduction of new bioactive materials, along with modified protocols, make more teeth with deep caries, traumatic injuries, and mechanical exposures viable candidates for innovative pulp therapies designed to maintain pulp survival. Outcomes depend on case selection, hemostatic agents, choice of pulp capping material, and the integrity of the sealed permanent restoration. The purpose in vital pulp therapy is to avoid or delay root canal treatment and advanced restorative care because these procedures, together, reduce long-term tooth survival compared to teeth with vital pulps.[76,77,118,270,289,328,366,380,385]

Procedures generating mineralized tissue barriers

Indirect pulp capping

Indirect pulp capping is defined by the American Association for Pediatric Dentistry (AAPD) as “a procedure performed in a tooth with a deep carious lesion approximating the pulp but without signs or symptoms of pulp degeneration. Indirect pulp treatment is indicated in a permanent tooth diagnosed with a normal pulp with no signs or symptoms of pulpitis or with a

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diagnosis of reversible pulpitis."[13] Hence indirect pulp capping is the permanent capping of a thin, pulp-nigh, cariously altered dentin layer, without complete excavation of the caries.[33] This may differ from the definition in other countries, for example, in the German-language literature, "indirect pulp capping" is defined as the treatment of a thin layer of caries-free dentin remaining above the pulp chamber with a medication.[332] The French definition of indirect pulp capping implies the covering of both healthy or carious dentin.[230] These definitions may lead to confusion when interpreting the literature.

Clinically, this situation usually arises in the excavation of an extensive carious lesion, so that indirect capping is also called *treatment of deep carious lesions*. A more general term would be "treatment of the pulp-nigh dentin," since indirect pulp capping may be necessary even in caries-free teeth, for example, after dental trauma.[357] Because only a minimal dentin layer remains above the pulp tissue, there is a risk of irreversible inflammation of the pulp via the dentinal tubules.[284] This can result from remaining bacteria or microorganisms actively entering the tissue and by cytotoxic components from restorative materials diffusing across thin residual dentin. Pulp-nigh dentin should be disinfected and bacteria-sealed with a capping material, stimulating the formation of tertiary dentin.[319] The indirect capping thus serves to protect the vital pulp, especially after caries removal. If there is already reversible pulpitis, a favorable environment for pulpal healing should be generated by indirect pulp capping (Fig. 24.3).

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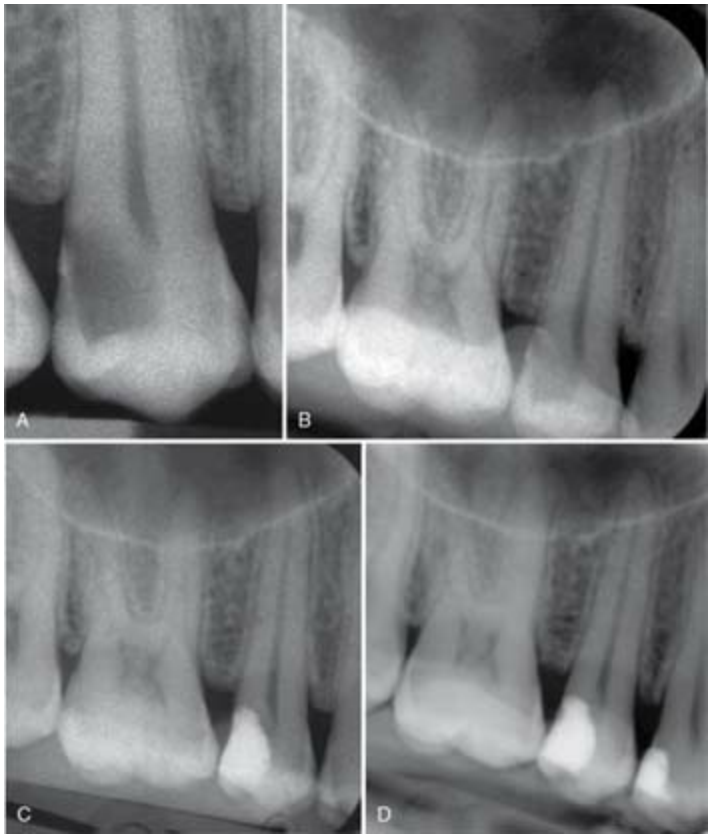

FIG. 24.3 A 24-year-old male patient presented with a deep distal carious lesion extending to the inner pulpal quarter in a maxillary right second premolar. The tooth was symptomatic but responded normally to sensibility testing. **A,** Magnified bitewing radiograph showing extension of advanced caries close to coronal pulp. **B,** Preoperative radiograph with period indentical device for precise comparison. **C,** Eighteen-month radiographic review after indirect pulp capping completed with calcium hydroxide. **D,** Five-year radiographic control. Pulp sensibility test response was positive with a well-defined lamina dura demonstrated on the periapical radiograph.

Set of four radiographs, marked A through D, shows the filling of radiolucent patch on crown of 4th tooth with calcium hydroxide.

Source: (Reprinted with permission, © Lars Bjorndal 2019.)

Advancing microorganisms and their by-products during the carious process pose a threat to the pulp.[322] Therefore, during caries excavation, the number of microorganisms in the cavity and near the pulp should be reduced, with indirect capping completed under dental dam isolation. To prevent spread of microorganisms, it is recommended to disinfect the clinical crown with NaOCl (1% to 5%) or chlorhexidine digluconate (CHX, 2%) prior to

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excavation. After caries excavation, the cavity should be cleaned with NaOCl or CHX and water spray.[53,75] Damage to vital tissue when using NaOCl is not of concern.[31] Nevertheless, it remains unclear how much altered dentin can be left while allowing the pulp to heal.[50,338] Hence indirect capping materials should eliminate potential residual microorganisms, neutralize any acidic (due to the carious defect) tissues, remineralize dentin, and stimulate the pulp to form tertiary dentin. Due to the disadvantages of aqueous CH suspensions, the use of CSCs can be seen as an improved alternative for indirect capping.[9]

Direct pulp capping

Direct pulp capping is defined as "placing a dental material directly on a mechanical or traumatic vital pulp exposure" and "sealing the pulpal wound to facilitate the formation of reparative dentin and maintenance of a vital pulp."[14] The procedure is indicated for pulp exposures incurred as a result of caries removal, trauma, or tooth preparation. When mechanical exposures occur during tooth preparation, the exposed tissue is generally not inflamed. However, in cases of trauma or carious exposure, the degree of inflammation is the key predetermining prognostic factor. According to the American Association of Endodontists (AAE), "In a carious pulp exposure, underlying pulp is inflamed to a varying or unknown extent."[14] The major challenge in DPC is the proper identification and removal of the acutely inflamed or necrotic tissue compromised by longstanding proximity to oral microorganisms.[247] Direct capping is therefore geared towards the healing of the pulp and the preservation of the tooth. If the pulp survives this treatment (remains vital), this can be considered a clinical success. Direct capping is a noninvasive, relatively simple and inexpensive treatment technique that, unlike root canal treatment, often does not require extensive restorative care.[198]

Since Hermann's first publications in the late 1920s, CH has been used as an aqueous suspension for direct capping.[189] However, the published success outcomes of DPC in a carious field show significant variation and the procedure remains controversial. Some recent publications therefore recommend selective excavation and leaving carious tissue in the tooth, suggesting improved performance over direct capping. For a direct

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comparison of the two treatments, there are currently only three clinical studies available, two comparing 1- and 5-year data and the third showing 1-year data.[25,51,52] In the second study, the prognosis of the two-stage excavation was compared with complete caries excavation treated by DPC or partial pulpotomy.[51] Five years after two-step caries excavation, the pulp was vital in 60% of the cases, whereas in the same period the success rates after direct capping were only 6% and after partial pulpotomy 11%.[51] The reason for lower success rates may include the absence of disinfection after pulp exposures, the choice of Dycal (Dentsply, Konstanz, Germany) as capping material, and the use of long-term provisional restorations.[181] However, in the third study, where no pulp exposures were detected after complete caries removal, indirect pulp capping resulted in a 100% success rate at 12 months. The results indicate that CSCs may be preferable in indirect pulp capping procedures and that hard setting CH cements do not provide long- or short-term pulpal protection.[181]

Partial pulpotomy

Partial pulpotomy (shallow or Cvek pulpotomy) is defined as the removal of a small portion of the vital coronal pulp as a means of preserving the remaining coronal and radicular pulp tissues.[14] During clinical treatment, the decision to remove a small or large portion of the coronal pulp is based on visual inspection of the tissue after pulp exposure during either caries excavation or exposure as a result of trauma (partial or shallow pulpotomy).[145] The incorporation of the dental operating microscope (DOM) is highly recommended. The coronal pulp is reduced from the exposed site by approximately 2 to 3 mm in order to remove necrotic or potentially inflamed and irreversibly damaged tissue, keeping the remaining pulp vital.[49] Partial pulpotomy is preferably performed with a high-speed handpiece and a small round diamond bur with copious water spray or continuous rinsing with physiological saline solution.[135,140,165]

After partial tissue removal, if bleeding cannot be controlled after 5 to 10 minutes of direct exposure to NaOCl, complete removal of the coronal pulp to the pulp floor is the preferred option.[211] NaOCl is a valuable diagnostic as well as clinical tool to differentiate irreversible from reversible pulpitis in the

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coronal pulp in order to help determine whether to proceed with partial pulpotomy, complete pulpotomy, or pulpectomy. This decision can be of paramount importance in young permanent teeth with open apices, in which removal of tissue contaminated by microorganisms can reverse symptoms and stabilize inflamed tissue.[89,130,390]

A recent study that examined MTA complete pulptomies suggested that a hemorrhage "control time" parameter is not an accurate indicator of pulpal inflammatory conditions, contrary to other investigations.[242] However, the study did not benefit from recommended protocols including verified necrotic tissue removal using the DOM. Investigations have shown that the inflammatory response of pulp tissue challenged by bacterial by-products can progress several millimeters into the coronal pulp or advance further, thus affecting the radicular pulp tissue distant from the injury site.[320] The removal of 2 to 3 mm of exposed coronal tissue or radicular tissue to access the deeper, healthy tissue, in cases of trauma, carious exposures or complete pulptomies can ensure pulp survival (Fig. 24.4).[101]

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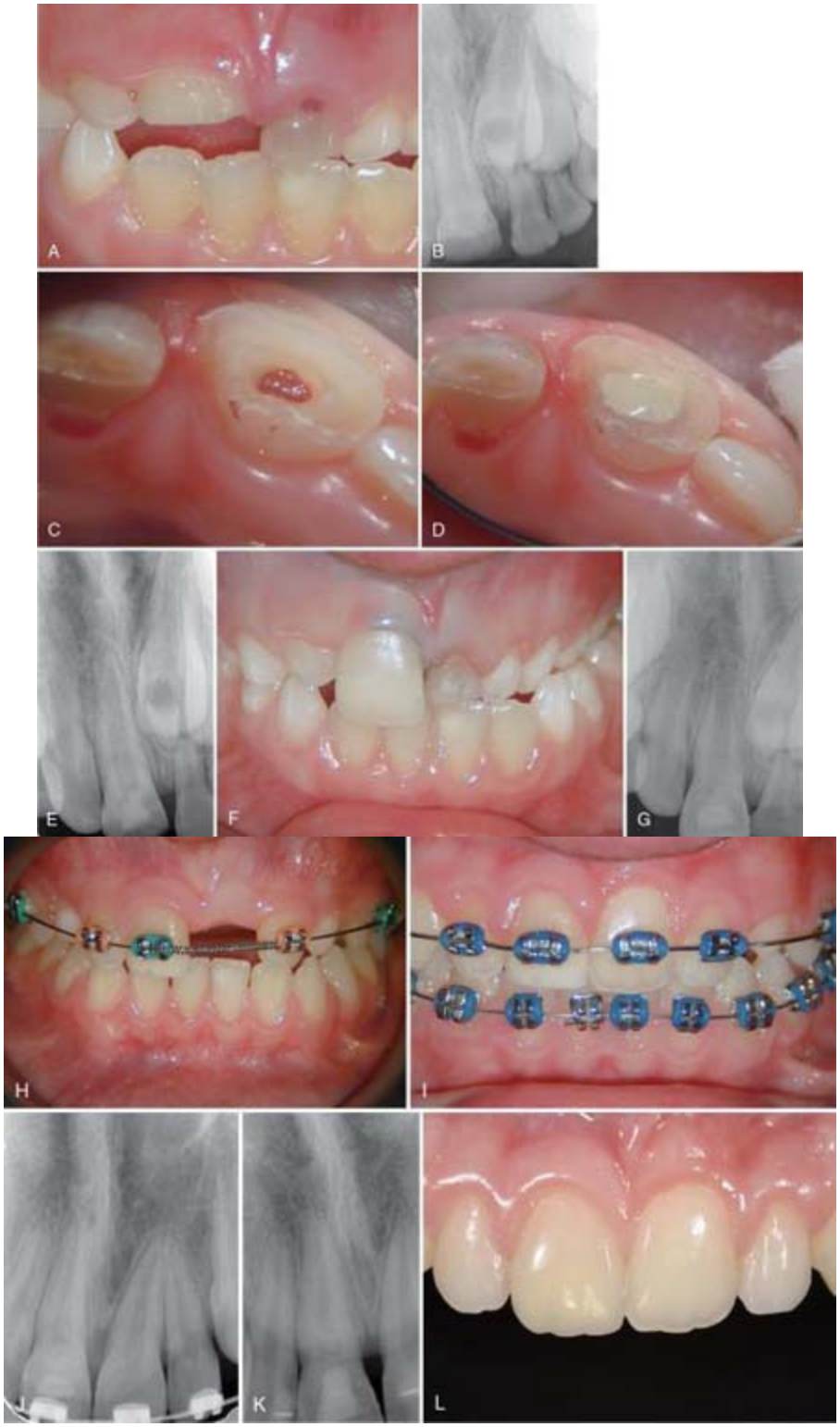

FIG. 24.4 An 7-year-old male patient referred after trauma displaying a horizontal coronal fracture of his maxillary right central incisor #8 with a pulp exposure covered with a glass ionomer cements by the general dentist and a retained deciduous left central incisor *(F)*. **A,** Clinical photograph showing horizontally fractured right maxillary incisor and

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retained deciduous left central incisor. **B,** Periapical radiograph reveals open apex #8 and unerupted maxillary left permanent incisor #9. In addition, a mesiodens is visible. **C,** Photograph after shallow Cvek coronal pulpotomy. **D,** View of access cavity after sodium hypochlorite hemostasis and placement of 3 to 4 mm Biodentine (Septodont, Saint-Maur-des-Fossés, France) plug. **E,** Radiograph after pulpotomy and temporary restoration placement. **F,** Clinical photograph 3 days after verified calcium silicate cements setting and reattachment of the fractured coronal segment after bonding. **G,** Radiograph of #8 with bonded crown segment reattachment showing immature open apex. **H,** Clinical photograph of full mouth banding after extraction of *(F)* and the mesiodens to permit normal eruption of tooth #9. **I,** Four-year clinical photograph with full orthodontic banding and complete eruption of #9 into the occlusal plane. Tooth #8 responded normally to cold sensibility testing at 1-, 2-, and 3-year recalls. **J.** Four-year periapical radiograph showing root-end closure of tooth #8 with normal periapical appearance. **K,** Five-year radiographic review of tooth #8 demonstrating complete radicular maturation. The incisor responded positive to cold testing. **L,** Clinical photograph at 5 years after finishing of a resin composite veneer created with a layering technique on tooth #8.

Set of photographs and radiographs, marked A through G, depicts the steps involved in recovery of coronal fracture in 8th tooth of the upper jaw. Radiograph G shows radiopaque patch on crown along with wide radiolucent root canal.

H) Close-up view of mouth shows one tooth missing at the center in the upper jaw. Upper teeth have braces.

I) close-up view shows braces on both upper and lower teeth.

J) Radiograph shows three upper teeth along with braces. The root apices of two teeth are joined the surrounding tissue has less bone density.

K) Radiograph shows upper teeth without braces.

L) Close-up view of upper jaw shows healthy teeth.

Source: (Courtesy Dr. Marga H. Ree with restoration by Dr. Caroline Werkhoven, Amsterdam, Netherlands.)

The amputation site is covered with a NaOCl soaked cotton pellet until hemorrhage ceases. If the formation of a blood coagulum is prevented, the same pulpal repair mechanisms associated with DPC are expected.[102,191] If the remaining pulp is healthy, bleeding can be stopped within 5 to 10 minutes. If hemostasis does not succeed within this time, it indicates that the

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pulp is irreversibly inflamed, and a complete pulpotomy (the removal of the entire coronal pulp) may be considered as a last resort to maintain vitality.[225] A CH suspension or CSC is applied to the newly exposed pulp surface and covered with a cement (for the CH suspension) or a thin layer of flowable composite, compomer, or RMGI (for the CSC).[92] A current study examining MTA versus CH pulpotomy has shown superior performance by MTA in mature permanent teeth compared with a hard-setting CH (Dycal). Since the capping material is used in larger quantities in the partial pulpotomy than in the DPC, the risk of tooth staining is considered higher when using a bismuth oxide containing CSC.[225] Partial pulpotomy and DPC are similar procedures, differing only in the amount of vital pulp tissue remaining after treatment. Partial pulpotomy is the preferred option in elective treatment procedures for teeth diagnosed with anatomic anomalies, such as dens evaginatus and invaginatus.

Complete pulpotomy

Complete pulpotomy, or complete *pulp amputation*, is a more intrusive procedure defined by the AAE as "the removal of the coronal portion of the vital pulp as a means of preserving the vitality of the remaining radicular portion: may be performed as emergency procedure for temporary relief of symptoms or therapeutic measure, as in the instance of a Cvek pulpotomy."[14] The entire coronal pulp is removed and the vital pulp tissue capped at the entrance level of the root canal orifice. After successful hemostasis, it is treated analogously to DPC (Fig. 24.5).[225]

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FIG. 24.5 A 10-year-old female was referred with a mandibular right first molar pulp exposure generated during caries excavation and temporized by the general dentist. **A,** Radiograph showing open apices with no evident apical pathosis. The tooth was diagnosed with reversible pulpitis. The patient received a new resin composite buildup to provide better isolation before a complete pulpotomy was performed. **B,** Clinical photograph of access opening through the buildup showing a sterile cotton pellet on the pulpal floor saturated with 5% sodium hypochlorite (NaOCl) for hemostasis. **C,** Photograph of radicular pulp in orifices after complete pulpotomy and 5% NaOCl hemostasis **D,** View through access opening after 5 mm thickness white mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) placement onto radicular pulp tissue and pulpal floor. **E,** Postoperative radiograph showing white MTA with wet cotton pellet and provisional restoration. One month later, MTA setting was verified and a permanent composite restoration provided. **F,** Radiographic review at 6 years shows apical maturation and thickening of the root walls. The patient remained asymptomatic and the tooth responded positively to electric pulp testing.

Set of six radiographs and photographs, marked A through F, shows the restoration of cavity in right first molar of the lower jaw. The final radiograph

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shows tooth with radiopaque patch in place of the cavity.

Source: (Reprinted courtesy Dr. Marga H. Ree, Purmerend, Netherlands, with permission.)

The assessment, selection, and amount of tissue removal are dependent on observer experience and completed on a case-by-case basis using magnification. Generally, after necrotic coronal tissue is completely removed, the inflamed pulp can be partially or completely amputated to the pulp floor or cervical area (pulpotomy) in the case of molars and some premolars.[144,147] The remaining tissue requires hemostasis before the placement of a CSC. The AAPD guidelines state, "A pulpotomy is performed in a tooth with extensive caries but without evidence of radicular pathology when caries removal results in a carious or mechanical pulp exposure."[13] However, the guideline requiring an absence of apical pathosis before pulp exposure for pulpotomy procedures may be outdated and require modification. Growing evidence from contemporary studies shows resolution of radicular lesions after pulpotomy treatments using CSCs in adult teeth diagnosed with irreversible pulpitis.[20,22,23,25,242,361–363]

Complete pulpotomy has traditionally been recommended for primary and immature permanent teeth for which short-term outcomes are generally favorable. However, the treatment of mature permanent teeth can be traced to Roman times around 200 BC.[415] Since the introduction of CSCs, there has been a renewed interest in pulpotomy as an alternative option to conventional orthograde root canal treatment, particularly in mature adult permanent teeth. Ongoing clinical investigations strongly support the use of CSCs for pulpotomy procedures in these teeth and show acceptable outcomes in pain reduction with continued normal function.[21–23,25,211,242,363,364,386]

Materials for vital pulp therapy

For successful pulp capping outcomes, the removal of noxious stimuli, control of any infection, and the biocompatibility of the capping material are important prerequisites.[55,75] It has been shown that rat pulp, in the absence of microorganisms, has the regenerative power to seal an exposed pulp with mineralized tissue even without medication or the use of restorative

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materials.[210,306] The presence or absence of microorganisms is the determining factor in the healing of pulp tissue.[210] The tissue reactions that follow capping (for instance, collagen synthesis and secretion) are generally similar to connective tissue injuries.[334,353] While excavating a deep caries lesion or during trauma, dentin or pulp tissue areas can be directly exposed, which are particularly permeable due to the structure of dentin. Closer to the pulp chamber, dentinal tubules occur more frequently per square millimeter and have larger diameters than further from the pulp.[336]

Since microorganisms, bacterial toxins (lipopolysaccharides), and many restorative materials can damage the exposed pulp tissue or diffuse via the dentinal tubules, causing irritation, both the pulp and the surrounding dentin should therefore be covered with the indicated capping material. Alternatively, pulp capping materials must create an artificial barrier between the vital pulp and the oral cavity to prevent the ingress of microorganisms. Moreover, the capping material should have antimicrobial properties without being toxic to the pulp.[108] In addition to the disinfection and sealing of dentin, another requirement of the capping material is hard tissue regeneration by pulp cell induction and thus preservation of pulpal vitality.[128,273,334] Mineralized tissue regeneration can occur in healthy pulps beneath a variety of capping materials.[15] The healing of pulp wounds is therefore not associated with a special drug or wound dressing.[44] However, medications to treat an exposed pulp should promote the innate capacity of pulpal cells to form hard tissue.[334]

Aqueous calcium hydroxide suspensions

CH has long been considered the universal standard material for vital pulp therapy. Although the material demonstrates many advantageous properties, long-term study outcomes in vital pulp therapy have been inconsistent.[30,40,42,196] Desirable characteristics of CH include an initial high alkaline pH, which is responsible for stimulating fibroblasts and enzyme systems. It neutralizes the low pH of acids, shows antibacterial properties, and promotes pulp tissue defense mechanisms and repair. The drawbacks of CH include weak marginal adaptation to dentin, degradation and dissolution over time, and resorption in primary teeth. Histologically, CH demonstrates

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cytotoxicity in cell cultures and has been shown to induce pulp cell apoptosis.[15,159,335]

Tunnel defects have also been demonstrated in hard tissue barriers generated over pulp wounds associated with both CH and CSCs.[10,15,38,98,99,159,162,275,334] However, the primary difference between the two agents is that CH products are absorbable over time and dimensionally unstable. The slow disintegration of the CH after mineralized tissue formation can allow microleakage, permitting a slow ingress of microorganisms through defects. This can induce subsequent pulpal degeneration and lead to potential dystrophic calcification and pulp necrosis. Over extended periods, this problematic outcome can complicate any necessary nonsurgical root canal treatment at a later date.[274]

Clinical retrospective investigations have shown variable success rates over 2- to 10-year recall periods for direct CH pulp capping in humans.[30,40,42,196] Two current studies have examined the efficacy of CH as a DPC agent. One examined the survival of 248 pulp-capped teeth that were diagnosed either as having normal pulps or exhibiting spontaneous pain; the researchers found an overall survival rate of 76.3% with an average recall period of 6.1 years.[110] Outcomes were less favorable for teeth showing spontaneous pain, in older compared to younger patients, and in teeth restored with glass ionomer cements (GIC). The probability of pulps becoming nonvital after CH pulp capping was greater within the first 5 years of treatment.

The second study observed 1075 teeth directly pulp capped with a CH-based agent; these teeth had either healthy pulps or showed signs of reversible pulpitis.[288] Inclusion criteria limited pulp chamber roof exposures to no larger than 2 mm in diameter. Successful outcomes were 80.1% after 1 year and 68% after 5 years; this diminished to 58.7% after a 9-year observation period.[395] The results demonstrate increasing failure rates over time, attributable to absorption of the material under permanent restorations proximal to hard tissue bridges with tunnel defects. Another investigation has confirmed decreasing success rates with CH pulp capping with extended recall periods.[258] CH clearly has many favorable characteristics, but it can no longer be considered the preferred agent in vital pulp therapy (Fig. 24.6).[134,193,258,260,286]

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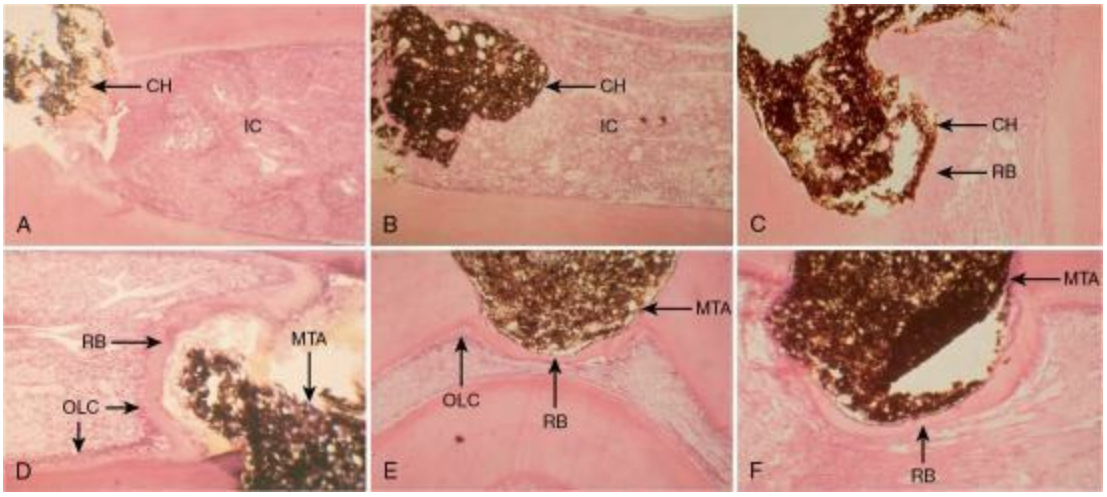

FIG. 24.6 Reparative bridge formation compared in dog pulps using mineral trioxide aggregate *(MTA)* and calcium hydroxide *(CH)*. **A,** Two-week pulp response to CH showing inflammatory cells *(IC).* **B,** Tissue specimen showing lack of bridge formation and disorganized tissue proximal to CH. **C,** An 8-week specimen with partial reparative bridge *(RB)* formation subjacent to CH. **D,** Two-week pulpal response to MTA showing notable barrier formation and layer of organized odontoblast-like cells *(OLC).* **E,** Pulp tissue section demonstrating complete calcific bridge formation proximal to MTA at 4 weeks. **F,** Sample section pulp capped with MTA at 8 weeks showing organized hard tissue formation with no inflammatory cell infiltrate.

Six stained micrographs are marked A through F.

A) It shows calcium hydroxide, CH, entering into inflammatory cell, IC.

B) Calcium hydroxide enters in inflammatory cells.

C) Reparative bridge, RB, is formed between calcium hydroxide pocket and inflammatory cells.

D) Reparative bridge is followed by odontoblast-like cells and has mineral trioxide aggregated, MTA, on the left.

E) A light patch is developed between MTA and RB.

Source: (Loma Linda University, Loma Linda, CA.)

Hard setting calcium hydroxide cements

In contrast to aqueous CH suspensions, other CH combinations such as cements (calcium salicylate ester cements) as well as liners or putties are less suitable for pulp capping due to a lower release of hydroxyl ions. In these

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preparations, the resulting pH is lower and the antimicrobial effect is significantly weaker.[139] In addition, hardening calcium salicylate ester cements (e.g., Dycal; Life, KerrHawe, Bioggio, Switzerland) are characterized by long-term disintegration.[38] They also fail to provide permanent support for the final restoration.[99] New mineralized tissue formation below CH-salicylate ester cements can be slow to generate and be less uniform in shape. Hard tissue regeneration may therefore be weaker using these CH preparations. In addition, inflammation is more common than with CH suspensions.[334] Some additives that harden the CH preparations may even have a toxic effect on the pulp.[99,334] For direct capping or pulpotomy, hard setting CH cements based on calcium salicylate esters cannot be recommended.

Light-curing liners and cements

To compensate for the disadvantages of proven CH preparations, light-curing liners and cements with CH additives were developed (e.g., Ultrablend Plus, Ultradent, South Jordan; Calcimol LC, VOCO, Cuxhaven, Germany). These materials do not have sufficient mechanical strength despite their composite content, and specific CH effects are minimal.[359] Moreover, the pH of these products is significantly lower compared to other CH products.[359] Nonetheless, antibacterial effects can be detected in vitro, although this has not been confirmed by other studies.[309,402] The cytotoxicity of these products, which is due to the composite component, has been clearly demonstrated.[184,309,310] Therefore according to the manufacturer's instructions, the direct contact of Calcimol LC to pulp cells should be avoided.

Another light-curing product is TheraCal LC (Bisco, Schaumburg, IL). The spectrum of indications according to the manufacturer corresponds to that of MTA and other CSCs. Unlike all other CSC products, where a cement powder must be mixed with a liquid (water), TheraCal LC consists of 45% Portland cement CEM III and 45% composite resin and is therefore photopolymerizable, significantly simplifying its clinical use.[154,155] However, it is known that all the monomers in TheraCal LC are cytotoxic in vitro.[180,202,207] After 1 day of direct contact with TheraCal LC, the fibroblast

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cell turnover rate decreased by 31.5% and after 1 week to 45.9%.[184] After 72 hours of contact with TheraCal LC there was a marked decrease in the percentage survival of the cells.[310] For tissue healing (hard tissue formation after pulp capping), it is crucial that there is no decrease but an increase in the cell turnover rate. In contrast to TheraCal LC, other calcium silicate products and CH have been shown to induce pulpal cell proliferation.[112,113]

Similar to other light-curing liners using CH, TheraCal LC exhibits a significant cell-damaging effect when in direct contact with human dental pulp stem cells.[58,184,309,310] It is also unclear how much Portland cement can be released from the resin matrix when the material is polymerized. After curing, TheraCal LC shows a heterogeneous structure with a high proportion of large, unhydrogenated particles, since the resin additives do not provide enough moisture to allow hydration. The hydration of TheraCal LC (the reaction of cement with water) is incomplete because of limited moisture diffusion within the material. Thus, no CH and only a small amount of calcium ions are released.[70] Light-curing liners and cements with added CH or MTA are collectively cytotoxic.[309,310] It is currently inadvisable to use photopolymerizable CH or calcium silicate-containing materials for indirect or DPC.

Composite resins, dentin adhesives, and resin-modified glass ionomer cements

Adhesive systems were introduced in the early 1980s as potential agents for DPC of cariously and mechanically exposed pulps.[200,253,404] Dentin adhesives, hydrophilic resins, and RMGI cements initially showed favorable outcomes in preliminary pulp capping investigations with nonhuman primates, based on standards set by the International Organization for Standardization.[96,367–369] However, the transitional use of these materials in human subjects did not demonstrate the corresponding biocompatibility or consistent reparative bridge formation.[5,119,159,183,197,271] Investigations that have examined responses to resin-based materials in human teeth have demonstrated unfavorable histologic reactions when the material is placed directly or in close proximity to pulp tissue.[5,124,172,272,315] Histological material typically demonstrates inflammatory cell infiltrates consistent with

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pulp cell cytotoxicity, subclinical adhesive failures at the pulp interface, and a profound absence of biocompatibility.[5,119,272,296,315,349]

The application of dentin adhesive to a thin layer of dentin (0.5 mm) leads to an expansion of the blood vessels and chronic inflammation of the pulp tissue.[183] Nonpolymerized or partially polymerized dentin adhesives cause apoptosis in various pulp cells, such as macrophages and undifferentiated pulp cells.[246] Besides monomers from dentin adhesives, identical cytotoxic components can also be released from composites, which then penetrate through dentinal tubules to pulp tissue causing damage.[59] Also, deep cavities (without pulp exposure) are problematic because the residual dentin thickness over the pulp can be thin, and tubular fluid can prevent complete polymerization. In addition, complete polymerization can be prevented by an excessively thick composite layer that is insufficiently penetrated by curing lights.[272] Both factors lead to an increased proportion of residual-free monomers, which are then released from the composite resin and diffuse into the pulp tissue. In clinical studies the use of dentin adhesives in deep cavities results in relatively frequent pulp necrosis.[337]

Unpolymerized, polymerized, and aged dentin adhesives release cytotoxic components that are responsible for inferior performance in human and animal experiments.[5,94,112] In addition, monomers from restorative materials (dentin adhesives/composites) may interact with the immune system of the pulp, weakening its defense against invading microorganisms.[44,268] Direct contact with dentin adhesives or composite resins is thought to reduce pulpal defense, either due to cytotoxicity or specific changes in the immune response.[207,267,268] Adhesive components thus inhibit the proliferation of immunocompetent cells and cause chemical immunosuppression, which promotes the development of pathological changes in the pulp.[44,207] Dentin adhesives and composites are essentially not biocompatible and should not be used for indirect or DPC.[9,94] However, hydrophilic resins and RMGI cements provide excellent seals when they are combined with light-cured composites as permanent restorations and placed directly over pulp capping materials such as MTA and other CSCs.[27,131,288]

Mineral trioxide aggregate

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MTA was introduced as a pulp capping agent by Torabinejad and associates in the mid-1990s.[141] Most preliminary experimental and current clinical data in vital pulp therapy are based on the proprietary material ProRoot MTA (Tulsa/Dentsply, Tulsa, OK). The cement consists of a hydraulic calcium silicate powder containing oxide compounds, including calcium oxide, ferric oxide, silicon oxide, sodium and potassium oxides, magnesium oxide, and aluminum oxide.[71] The material exhibits favorable physicochemical characteristics that stimulate reparative dentinogenesis by recruitment and activation of hard tissue–forming cells, contributing to matrix formation and mineralization.[298] Soluble cytokines and growth factors that mediate wound repair of the dentin-pulp complex are nested in the extracellular matrix. MTA stimulates reparative hard tissue formation by sequestering these growth factors and cytokines embedded in the surrounding dentin.[222,223,375,385]

CH and calcium silicate hydrate, the principal by-products formed during hydration of mixed MTA, contribute to a sustained alkaline pH.[68,143] The setting properties of the hydroscopic silicate cements are not affected by the presence of tissue fluids or blood.[377] They are therefore also referred to as "hydraulic" cements, as they are hard setting both in contact with air and water.[47] During the setting process, the gradual release of calcium ions encourages reparative barrier formation by promoting signaling molecules, such as VEGF, macrophage colony-stimulating factor, TGF-β, and interleukins IL-β and IL-1α.[251,300]

MTA demonstrates superior marginal adaptation to dentin compared to CH-based agents and forms a dense bond to dentin. Mineral components from the cement penetrate into the tubules, producing adhesion to dentin comparable to GICs.[29,216] MTA also forms an adherent interfacial layer during mineral nucleation at the dentin surface that appears similar in composition to hydroxyapatite when examined with x-ray diffraction, energy-dispersive x-ray analysis, and scanning electron microscopy (SEM).[298,329,379] Similar to CH, MTA induces an inflammatory cascade that results from calcium ion release and the creation of an alkaline environment, producing tissue necrosis. Both MTA and CH have been shown to stimulate and increase the Höhl cell mitosis index in rodents.[114,115]

MTA activates the migration of progenitor cells (fibroblasts) from the central pulp to the injury site and promotes their proliferation and

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differentiation into odontoblast-like cells without inducing pulp cell apoptosis.[298] MTA also stimulates in vitro the production of messenger RNA and increases protein expression of the mineralized matrix genes and cellular markers crucial for mineralization after matrix formation.[298] In a human study, odontoblast-like cells and the formation of a dentin or dentin-like tissue could not been proven after DPC with MTA (RetroMTA; BioMTA, Daejeon, Korea). Hence the new calcified hard tissue was not "regular dentin" and did not seem to be the product of genuine odontoblast differentiation. This observation is identical to results after DPC with CH. Hard tissue formation after DPC appears to be a dystrophic intrapulpal mineralization in response to the capping procedure.[111]

Gray MTA has been shown to enhance cell proliferation and survival of cultured human dental pulp stromal cells.[300] The biocompatibility of set MTA upregulates the expression of transcription factors, angiogenic factors, and gene products, such as dentin sialoprotein, osteocalcin, and alkaline phosphatase.[299] Odontoblast signaling proteins are essential in the differentiation of progenitor cells into cells responsible for repair and hard tissue deposition.[299,300] After MTA pulp capping, both sialoprotein and osteopontin have been observed in the mineralized hard tissue matrix at the exposure site during the process of reparative hard tissue formation.[227]

Dental pulp cells differentiate into the odontoblastic cell line in the presence of the signaling molecules, such as TGF-β, heme oxygenase-1 enzyme, and bone morphogenetic proteins BMP-2, BMP-4, and BMP-7.[171] MTA most likely upregulates fibroblast secretion of BMP-2 and TGF-β1 and therefore stimulates and promotes mineralization and hard tissue regeneration.[117,170,171,178,340,365,406] MTA induces a time-dependent environment that is proinflammatory and promotes wound regeneration through upregulation of cytokines.[318] Immunohistochemical analyses show that cytokines, including myeloperoxidase, inducible nitric oxide synthase, VEGF, nuclear factor-kappa B, activating protein-1, and cyclooxygenase-2, show increased expression in the presence of MTA. Cytokine upregulation is responsible for inducing biomineralization by producing apatite-like clusters on collagen fibrils at the MTA-dentin interface. MTA does not affect the generation of reactive oxygen species, thereby positively influencing cell survival. MTA also has been shown to improve the secretion of IL-1β, IL-6,

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and IL-8.[67,81,91] However, an inhibitory effect on dental pulp cells has been demonstrated in the presence of MTA, which may be attributed to the release of aluminum ions.[267] Overall, the data indicate that MTA promotes a biocompatible, noncytotoxic, antibacterial environment and surface morphology that is favorable for reparative calcific bridge formation. MTA stimulates the release of the dentin matrix components necessary for hard tissue repair and regeneration in mechanically exposed healthy and partially inflamed pulps (Figs. 24.7–24.9).[6,141,170,266,286,317,374]

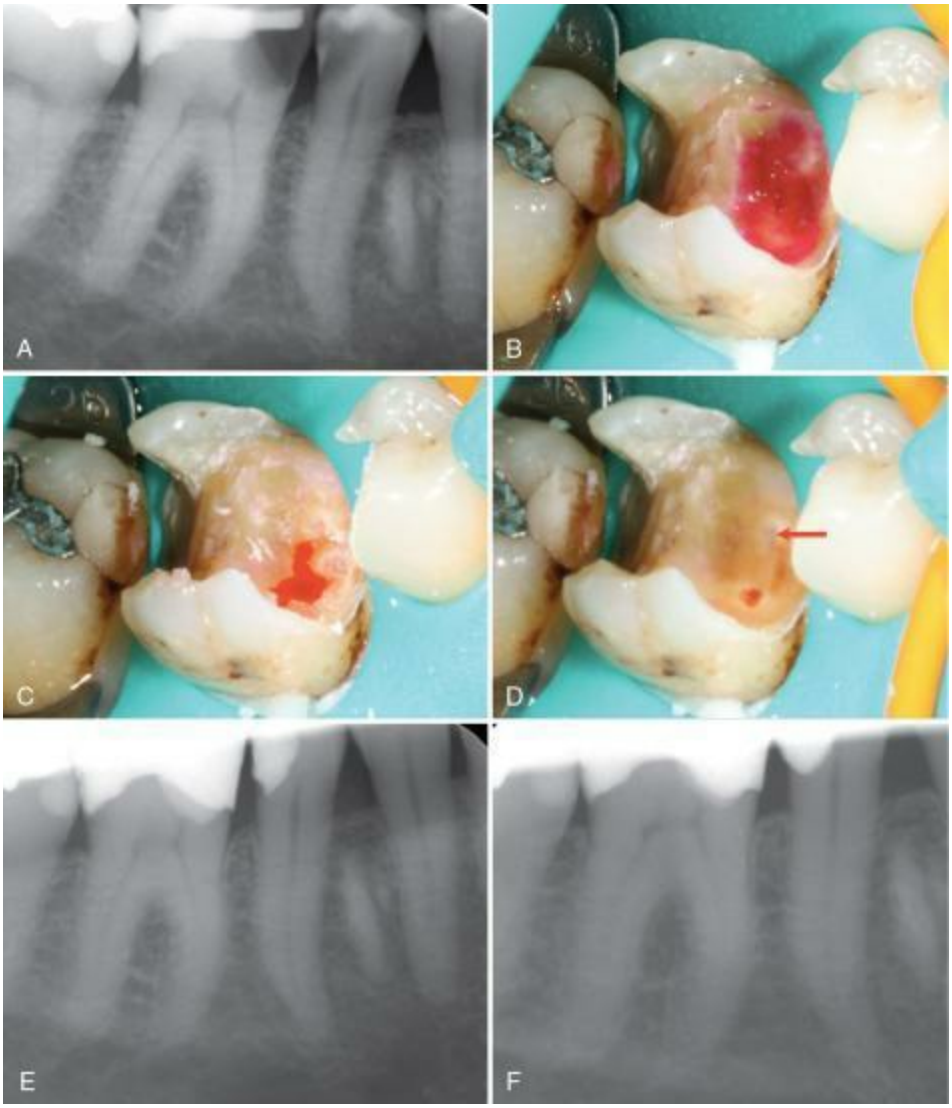

FIG. 24.7 A 50-year-old male patient with deep recurrent caries associated with a mandibular right first molar (#30). The patient had no history of pain and responded normally to cold testing. **A,** Periapical

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radiograph showing distal caries in the mandibular right second premolar (#29) and large carious lesion below mesial aspect of composite restoration in the molar. Note retained deciduous root tip mesial totooth #29 and normal periapical appearance of #30. **B,** Pulpal hemorrhaging evident after complete caries removal with hand excavators. **C** and **D,** Clinical photographs after bleeding was controlled using cotton pellets soaked in buffered 0.5% sodium hypochlorite (NaOCl). Note presence of reparative dentin protecting mesiolingual pulp horn *(red arrow)*. A 2 mm–thick layer of white mineral trioxide aggregate (Dentsply, Tulsa Dental, Tulsa, OK) placed directly over the exposure and surrounding dentin and covered with a thin wet cotton pellet. The tooth was provisionalized for 1 week and restored permanently with composite resin. **E,** One-year radiographic follow-up; tooth responded normally to cold sensibility testing. **F,** Control radiograph at 3 years. Patient was asymptomatic and exhibited normal pulp testing response.

Set of radiographs and photographs marked A to F shows the recovery of carries in the crown of 30th tooth.

Source: (Courtesy Dr. Rita Kundzina, Tromsø, Norway.)

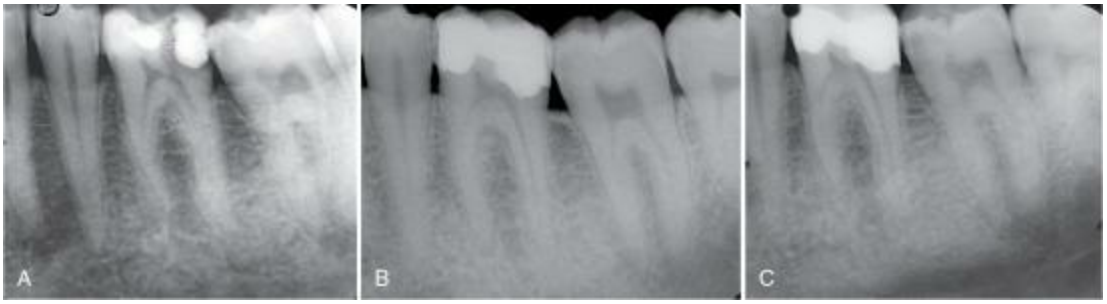

FIG. 24.8 Symptomatic mandibular left first molar in a 32-year-old male patient with temporary restoration placed over deep caries on the distal aspect and referred for root canal treatment. Sensibility testing elicited a normal pulpal response. **A,** Periapical film showing deep caries extending towards the distal pulp horn. **B,** Radiograph of direct mineral trioxide aggregate (Dentsply, Tulsa Dental, Tulsa, OK) pulp capping after 5.25% sodium hypochlorite hemostasis and placement of a three-surface bonded composite restoration. **C,** Nine-year 6-month radiographic control. The tooth was positive to cold testing and without symptoms. Note presence of mesial carious lesion in the second molar.

Set of three radiographs marked A through C shows gradual changes in radiolucent patch on crown in first molar of the lower jaw.

Source: (© Dr. George Bogen.)

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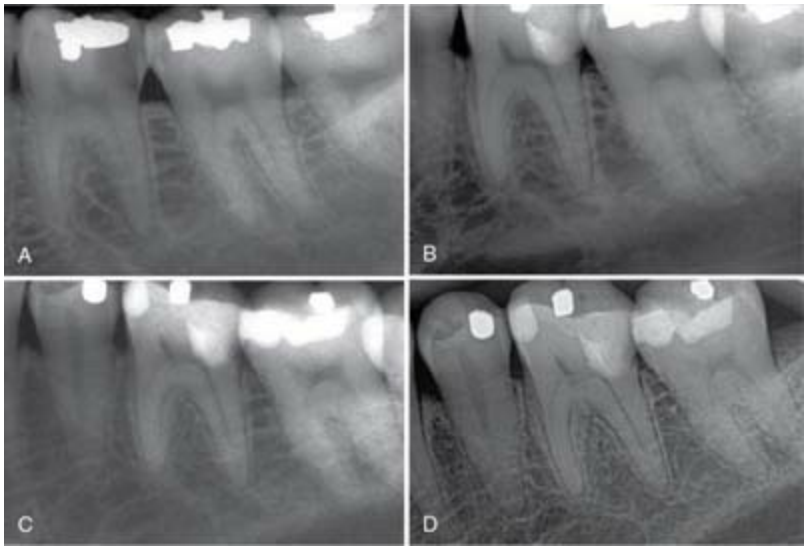

FIG. 24.9 **A,** Periapical radiograph of deep caries in a partially symptomatic mandibular left first molar in a 29-year-old female patient. **B,** Direct pulp capping completed with white mineral trioxide aggregate (MTA); the final bonded restoration was placed during the second visit after MTA curing. **C,** Radiographic recall at 7 years. Tooth was asymptomatic with normal response to CO_2 ice. **D,** Thirteen-year 10-month recall radiograph showing normal periapical appearance. The molar responded positive to cold testing and demonstrated normal mobility and periodontal probings.

Set of four radiographs, marked A through D, shows the radiolucent patch at the crown of tooth changed to hard density tissue in the D radiograph.

Source: (© Dr. George Bogen.)

One disadvantage of MTA is that it can lead to discoloration of the hard tooth tissue. This may be problematic, particularly in anterior teeth during trauma management.[279] It is due to heavy metals contained in MTA, such as bismuth oxide used for radiopacity or iron.[45,123,347] The discoloration is mainly induced by the oxidation of these metals after contact with NaOCl or the uptake of blood components.[69,234,347] Other CSCs contain less or minute levels of heavy metals and their potential to discolor teeth is minimal. CSCs containing zirconium oxide or tantalum oxide, which are used as radiopacifiers, are particularly color-stable.[279]

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Calcium silicate cements

A variety of new CSCs have developed since the introduction of MTA.[116,153,232,302] Preliminary investigations have demonstrated physicochemical and bioinductive properties comparable to MTA, indicating promise for their application in vital pulp therapy.[18,57,127] Some of these tricalcium-based materials include BioAggregate (Innovative Bioceramix, Vancouver, BC, Canada), Biodentine (Septodont, Saint-Maur-des-Fossés, France), MTA-Angelus (Angelus, Londrina PR, Brazil), MTA Bio, and MTA Branco (Angelus).[160,237] Other formulations include EndocemMTA (Maruchi, Wonju-si, Gangwon-do, South Korea) and EndoSequence BC root repair material (Brasseler USA, Savannah, GA). Currently, there are more than 40 different CSC formulations from international manufacturers. Some other silicate cement products include EndoBinder, iRoot BP, MicroMega MTA, MTA Bio, MTA Plus, NeoMTA Plus, RetroMTA, Tech Biosealer Capping, TheraCal LC, CEM Cement (calcium-enriched mixture), Grey MTA Plus, MEDCEM MTA, EndoCem Zr, Channels MTA, DiaRoot Bioaggregate, and Harvard MTA.[302] Additional compounds are currently undergoing clinical investigations to establish their safety and efficacy.[219,278,302,378]

The main components of MTA and the new CSCs are tricalcium silicate and dicalcium silicate, the major components of Portland cement. Hydraulic tricalcium silicates promote reparative barrier formation by the upregulation of transcription factors after gaining immediate strength on hydration. The cements also encourage hydroxyapatite crystal formation on the cement surface when in contact with calcium- and phosphate-containing fluids.[155,179] Moreover, the release of CH from CSCs during hydration has a positive effect on cell regeneration. Osteoblasts, cementoblasts, periodontal ligament cells, and pulp cells are deposited directly on the CSC surface, as the material is recognized as “non-foreign,” which affirms the high biocompatibility of these cements.[378]

In addition to CH, silicon is released during CSC cement hardening. The exact function of silicon in the metabolic processes of hard tissue formation is unclear, but it is believed to play a role in the early stages of mineralization.[80] It is also known that silicon positively influences the rate of

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new hard tissue deposition when it is released from bioactive materials such as MTA.[304] In addition, silicon can remineralize demineralized dentin in vitro and this has also been demonstrated for CSCs.[28,304,327] It can be concluded that the release of silicon from calcium silicate-containing materials contributes to the induction of mineralized tissue formation.[179]

BioAggregate is a bioinductive tricalcium cement that can induce mineralization in osteoblast cells by increasing levels of osteocalcin, collagen type 1, and osteopontin gene expression.[412] Hydration of the cement results in the formation of calcium silicate hydrate and CH, showing high concentrations of silica and calcium phosphate.[166] This characteristic is consistent with materials used in vital pulp therapy to promote hard tissue formation. Investigations using x-ray diffraction show the material to have a composition similar to that of MTA, but BioAggregate contains tantalum rather than bismuth oxide for radiopacity.[303] It demonstrates remarkable biocompatibility compared to MTA, inducing cell differentiation in both human periodontal ligament and gingival fibroblasts.[361,405] Both fresh and set mixtures of MTA and BioAggregate have shown antimicrobial properties effective against *Enterococcus faecalis* in vitro when combined with equal amounts of human dentin powder.[414] The material also shows a greater resistance to dislodgement in an acidic environment compared to MTA, and higher fracture resistance when used as a filling material.[182,383]

Biodentine is a tricalcium silicate–based cement that also demonstrates exceptional bioactive properties.[2] Whereas MTA is more or less comparable to a refined Portland cement, Biodentine consists mainly of pure tricalcium silicate (about 80%) with calcium carbonate as filler (about 15%).[47,109] Zirconium oxide is added as radiopacifier (about 5%). In contrast to other CSCs and MTA, Biodentine does not contain dicalcium silicate or metal oxides except the radiopacifier.[72,341] The liquid consists of calcium chloride in an aqueous solution with an admixture of modified polycarboxylate. The powder is mixed in a triturator with the liquid in a capsule. Once mixed, Biodentine sets in about 15 minutes minimum, and hardens completely in 85 minutes.[217]

Biodentine does not induce genotoxic or cytotoxic effects when measured with the Ames mutagenicity test. It is considered a biocompatible dentin

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replacement material for use under restorative materials as a base.[232] During setting, Biodentine releases calcium ions forming CH that lead to an alkaline pH in the surrounding tissues, inhibiting the growth of microorganisms.[48,70] Furthermore, when Biodentine is applied onto pulp cells ex vivo, TGF-β1 is released.[231,250] In vitro Biodentine induces proliferation, migration, and differentiation of human dental pulp cells and induces cell proliferation in osteoblasts and periodontal ligament cells.[17,209,232,307]

These factors may explain hard tissue formation after DPC with Biodentine.[2] The material stimulates biomineralization and encourages hard tissue formation when used as a capping material.[343,413] Histological evaluation in humans revealed complete mineralized tissue formation 6 weeks after DPC with mild to absent pulp inflammatory reactions, comparable to MTA.[295,297] Biodentine forms needle-like crystals resembling apatite at the dentin interface.[120] Identically to MTA, Biodentine seals dentin by micromechanical retention with a shear bond strength comparable to GICs.[29,216] Different light-curing adhesive lining materials are able to bond to Biodentine and may prevent bacterial leakage.[2,333] After indirect capping with Biodentine, a reversible pulpitis can reproducibly heal.[182] Success rates of human DPC with Biodentine are between 82.6% and 86% after 1.5 to 2.3 years and thus in the range given for ProRoot MTA (Figs. 24.10 and 24.11).[181,193,206,226,244,248,260]

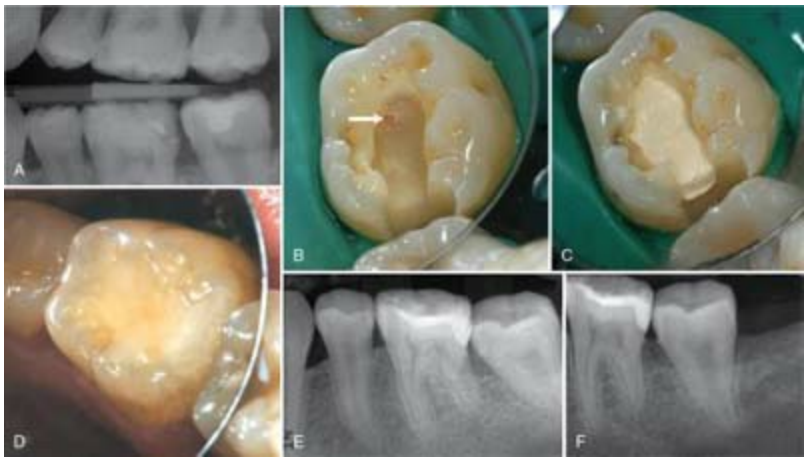

FIG. 24.10 **A,** Bitewing radiograph of quadrants 2 and 3 showing defective restoration in a mandibular left first molar (#19) of a 27-year-old female patient. **B,** Clinical photograph showing pulp exposure

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(white arrow) after cavity preparation and complete caries excavation. **C,** Pulp capping and restorative base completed under slight pressure using Biodentine (Septodont; St. Maur-des-Fossés, France). **D,** Clinical photograph of final two-surface composite restoration placed after a setting time of 15 minutes. The restoration was placed after acid etching and bonded with a two-bottle dentin adhesive system. **E,** One-year periapical radiograph showing a normal periapical region and Biodentine radiopacity similar to that of surrounding dentin. **F,** Three-year radiographic review shows no evident pathological changes apically. The tooth registered normal responses to sensibility testing at both recall periods.

Set of radiographs and photographs marked A through F shows the restoration of broken edge of crown in left first molar of lower jaw.

Source: (Reprinted with permission, Dr. Till Dammaschke and © Quintessence Deutschland.)

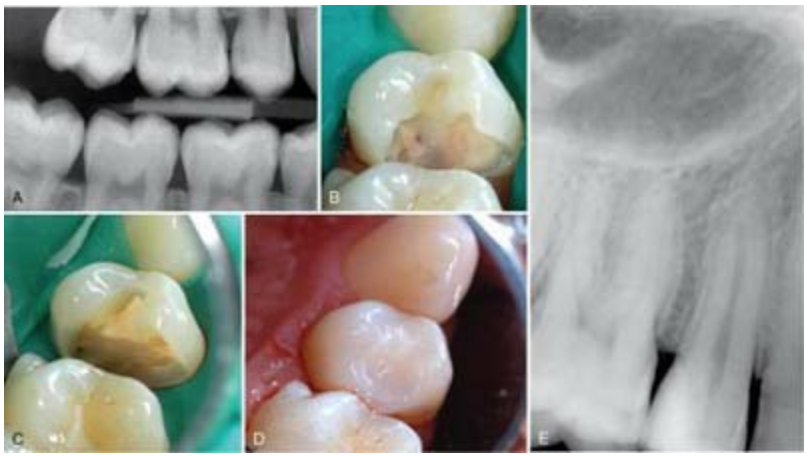

FIG. 24.11 **A,** Bitewing radiograph of quadrants 1 and 4 of an 18-year-old patient displaying a distal carious lesion in the maxillary right second premolar. The patient did not present at the scheduled appointment for restorative care. **B,** The patient returned 4 years later for treatment. Clinical photograph showing iatrogenic pulp exposure at two sites during complete caries excavation. **C,** Photograph of direct capping and base placement with Biodentine (Septodont; St. Maur-des-Fossés, France) applied to the cavity with cement pluggers using light pressure. **D,** Clinical view of cavity restored with bonded composite resin using a two-bottle dentin adhesive. **E,** Radiographic control recorded 4 years after direct capping showing normal periapical tissues. Tooth was asymptomatic with normal response to sensibility testing.

Set of radiographs and photographs marked A through E shows the recovery

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of cavity lesion in the second premolar.

Source: (Reprinted with permission, Dr. Till Dammaschke and © Septodont [St. Maur-des-Fossés, France].)

Another promising material for vital pulp therapy is MTA-Angelus, which has a basic formulation of 25% bismuth oxide and 75% Portland cement. The composition eliminates calcium sulfate, providing a short setting time of 15 minutes, preferable for pulp capping or pulpotomy procedures.[57] Variations in bismuth oxide and the presence of iron characterize the chemical composition of MTA-Angelus, and the crystalline structures formed on hydration are similar to gray and white ProRoot MTA.[355] These materials have been compared experimentally as pulp capping agents in intact, caries-free human teeth. Histomorphologic examination of the extracted teeth revealed similar inflammation and hard tissue formation responses.[4] MTA-Angelus also demonstrates antifungal properties and a lower compressive strength than ProRoot MTA.[41,212,219] Many CSC materials demonstrate acceptable antibacterial products; however, *E. faecalis* has shown resistance to some cements when measured with disc diffusion tests.[219]

EndoSequence BC root repair material shows low cytotoxicity, antibacterial activity against *Enterococcus faecalis*, and potential as a pulp capping material.[105,416] Another material, calcium-enriched mixture (CEM) cement, has also demonstrated excellent physical and biologic properties in vital pulp therapy investigations.[20–24,26,294] The advantages of these calcium silicate materials over the typically used CH products lie in the higher mechanical strength, lower solubility, and tighter sealing to dentine. Major disadvantages of CH are avoided when using CSCs: dissolution of the capping material as well as the mechanical instability and consequent lack of long-term protection against bacterial microleakage.[108] The new generation of CSCs appear promising when used as vital pulp therapeutic agents, and current investigations appear to support their future potential and expanded use in vital pulp therapy.

Calcium silicate cement applications in vital pulp therapy

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Direct pulp capping with calcium silicate cements

Controlled prospective investigations on DPC in humans using MTA and other CSCs against cariously exposed pulps are limited but expanding. Collectively, most studies are inconsistent with regard to case selection, treatment strategies, and clinical protocols.[268] The spectrum of outcomes reflects the absence of standardized guidelines for caries removal, hemostatic agents, choice and placement of the capping material, and single- versus two-visit delivery sequences. With the advent of quicker setting CSC formulations, indications for two-visit pulp capping are declining in favor of one-visit procedures. Moreover, although the concept of DPC in a carious field is gaining universal acceptance using MTA and other CSCs, clinical recommendations for avoiding and treating pulp exposures in permanent teeth as an accepted management strategy persist, despite contrary evidence.[129,316] Current international guidelines are variable because the research and technological advances within the field are moving exponentially without a universal consensus.

Human studies using CSCs other than MTA are showing the mutual biocompatibility of new bioceramic materials, indicating that conjointly, the cements exhibit similar properties and cellular responses when exposed to vital pulp tissues. Comparable outcomes are evident when pulp capped teeth are examined for mineralized tissue barrier formation, lack of inflammatory cells, and continued vitality in DPC.[31,62,181,185,206,211,214,219,243,301] Analysis has shown that initial pulp capping treatment is also cost-effective compared to root canal treatment in younger patients with pulp exposures due to caries.[63]

Current and past investigations comparing newer CSC formulations have shown clinical and radiographic success in permanent teeth treated with carious exposures ranging from 83.4% to 100% at 1 to 7.4 years.[31,62,181,185,214,243,244,301] In a larger study using Biodentine as a DPC agent, 245 teeth recalled with an average recall period of 2.3 years showed continued vitality in 86% of the cases. In one study where Biodentine was compared against MTA in a split mouth model or randomly allocated in different patients, outcomes at recall periods of 18 to 36 months were comparable for both materials when examined in permanent mature teeth with carious exposures.[214] Impressive results have also been demonstrated in

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several other contemporary studies.[3,6,238,258] An investigation examined 30 immature permanent teeth exhibiting wide-open apices directly pulp capped with MTA and restored temporarily with intermediate restorative material (IRM).[138] Definitive permanent composite restorations were placed 2 weeks later, after confirmation of pulp vitality. In these cariously exposed immature permanent teeth, the success rate at a 2-year review was 93%.

One notable factor revealed in a recent study with a broad sample size was that treatment outcomes were negatively influenced when restored with a GIC compared to all other restorative materials.[181] The outcome is a likely indicator that GIC should not be considered as a definitive restoration in DPC cases.[181] The outcomes of these current studies strongly suggest that MTA and introduction of new CSC capping agents show capable biocompatibility and sealing properties and can make the outcome of DPC techniques in mature permanent teeth with carious exposures increasingly reliable.

Another observational investigation examined DPC of carious exposures of mature and immature permanent teeth using slow-set MTA in a two-visit sequence.[56] Forty-nine teeth were examined in patients aged 7 to 45 years over a 1- to 9-year period, with an average 3.94-year observation time. The study protocol used detector dye-aided caries excavation using the DOM, 5.25% to 6% NaOCl hemostasis, bulk MTA placement on pulp exposures, and surrounding dentin, coupled with bonded composite restorations placed at a second visit to compensate for the delayed setting properties of ProRoot MTA. Based on subjective symptomology, cold testing, and radiographic evaluation, 97.96% of teeth showed continued vitality. All patients with immature apices showed continued root formation and apical closure within a 1- to 6-year period (Fig. 24.12).

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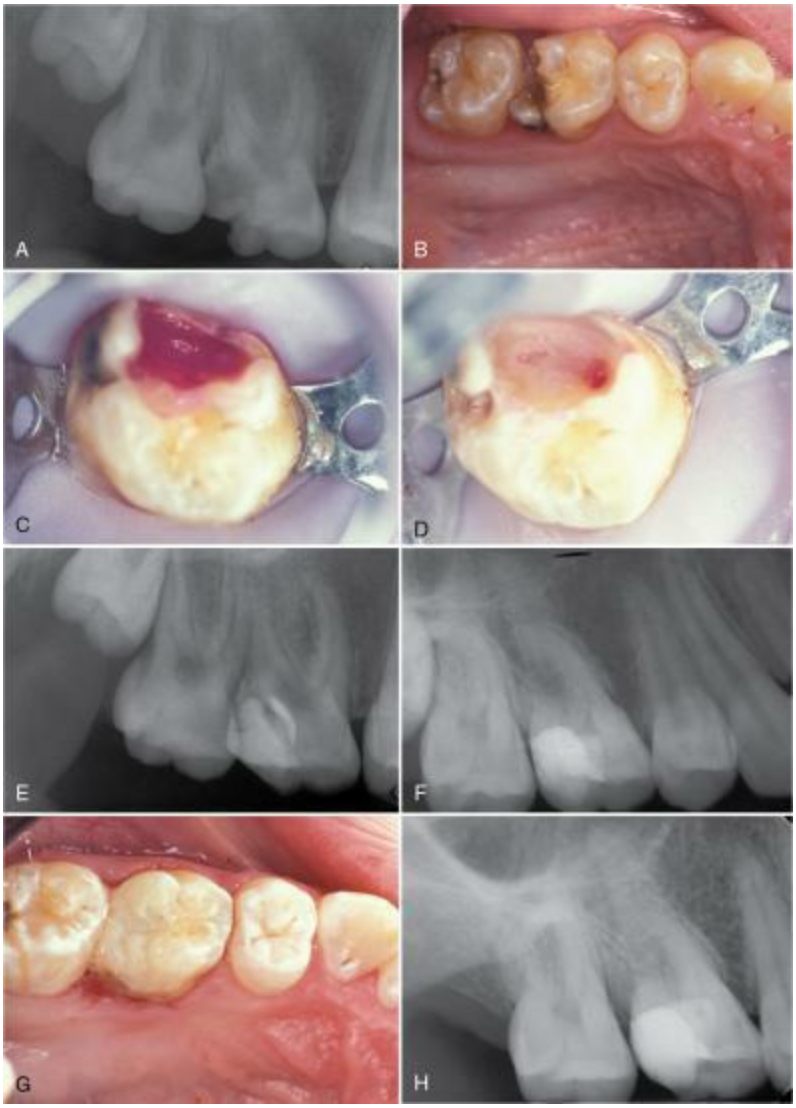

FIG. 24.12 A symptomatic 15-year-old patient presented for treatment with deep caries associated with the maxillary right first molar (#3). **A,** Preoperative radiograph showing deep caries but no evidence of apical pathosis. **B,** Clinical photograph of the carious molar. **C,** Caries removal using caries detector dye. **D,** Clinical view of pulp exposures after 5.25% sodium hypochlorite hemostasis. **E,** Periapical radiograph of direct mineral trioxide aggregate (MTA) pulp cap with wet cotton pellet and unbonded Photocore provisional. **F,** Permanent bonded composite placed 5 days after MTA direct pulp cap. **G,** Clinical photograph of final bonded composite restoration. **H,** Twenty-year 4-month recall radiograph without restorative intervention, patient was asymptomatic and responded normally to cold testing.

Set of radiographs and photographs marked A through H shows the restoration of caries at the one side of crown in 3rd tooth of upper jaw.

Source: (Reprinted courtesy Dr. George Bogen with permission.)

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A related DPC investigation using a two-step protocol in permanent teeth using MTA with bonded restorations with slight variations in procedural execution registered similar outcomes. Sixty-four teeth recalled after 3.6 years showed an overall success rate of 91.3%. The success in patients older than 40 years of age was 80% and for those younger than 40 years was 100%.[248] In a corresponding study, 33 patients with DPC after carious exposures and provisionalized for 1 week (two-visit protocol) showed a 85% success rate at 3 years.[226] The same study also compared outcomes with patients that received direct CH capping and demonstrated a survival rate of 52% at 3 years. The majority of human studies that compare MTA/CSC direct capping in permanent teeth with CH suggest MTA/CSCs offer more predictable long-term benefits.[78,226,239] Moreover, larger scale pulp capping studies comparing MTA/CSCs with CH corroborate previous data that demonstrate superior results when MTA/CSCs are applied in a clinical setting (Figs. 24.13 and 24.14).[66,193,238,260]

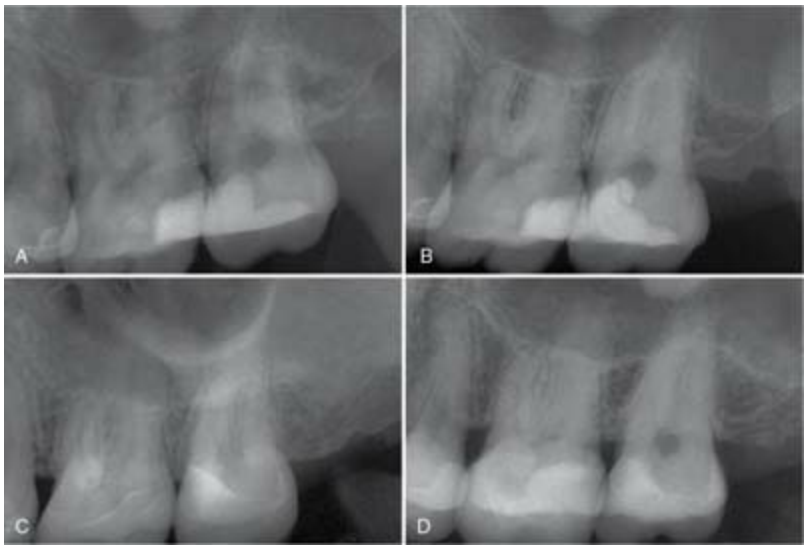

FIG. 24.13 A 23-year-old female patient presented with an IRM temporary restoration placed over a large pulp exposure after caries excavation of the maxillary left second molar (#15). The patient was referred for root canal treatment but had no history of pain and responded normally to sensibilty testing. **A,** Periapical radiograph showing temporary material in close proximity to mesial pulp horn. The tooth received a direct pulp cap with mineral trioxide aggregate (MTA) after further caries excavation and 5.25% sodium hypochlorite hemostasis. **B,** Radiograph showing direct MTA (Dentsply, Tulsa

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Dental, Tulsa, OK) pulp cap and bonded composite restoration placed at second visit. **C,** Two-year 5-month radiograph showing normal periapical structures and newly cemented porcelain onlay. **D,** Periapical radiographic review after 12 years and 8 months. The tooth responded positive to sensibility testing at both recall periods.

Set of four radiographs marked A through D shows two teeth with different extent of radiopaque material on the crown.

Source: (© Dr. George Bogen 2019.)

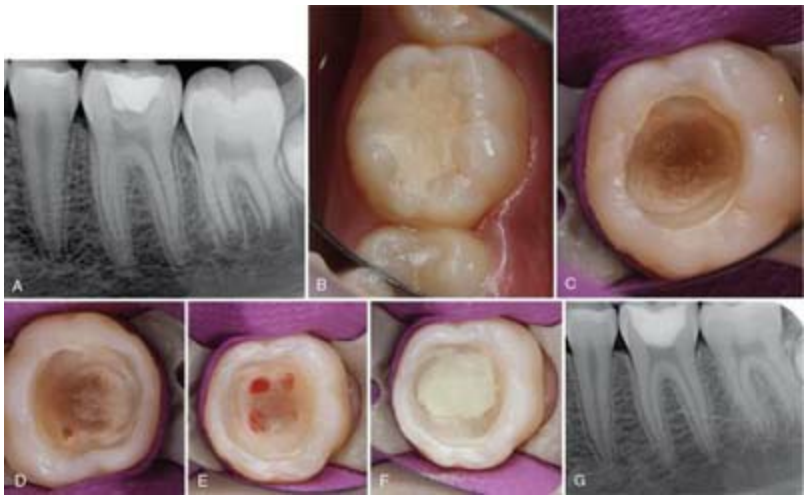

FIG. 24.14 A 12-year-old patient presented with a history of pain and cold sensitivity from a mandibular left first molar after having an occlusal composite restoration placed 16 weeks earlier. **A.** Periapical radiograph shows recurrent caries below base of the composite restoration. Note immature apices of the second molar. **B.** Clinical photograph of recently placed occlusal composite restoration. **C.** Photograph of initial caries removal showing remaining decay adjacent to pulpal roof. **D.** Photograph reveals three pulp horn exposures and presence of reparative dentin. **E.** Clinical view of three pulp horn exposures and hemostasis after application of 5% sodium hypochlorite. **F.** Photograph of bulk white mineral trioxide aggregate (MTA) (ProRoot MTA (Tulsa/Dentsply, Tulsa, OK) placement over exposures and surrounding dentin. The white MTA was covered with a flowable composite and the cavity restored with a bonded composite restoration using a two-bottle bonding system with enamel etching. **G,** One-year periapical radiograph showing normal apical structures. The tooth was asymptomatic and responded positively to vitality testing.

Set of radiographs and photographs marked A through G shows the restoration of caries above the pulp horn in left first molar of the lower jaw.

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Source: (Courtesy Dr. Miguel Marques, Amsterdam. Netherlands.)

The unique physicochemical properties of MTA and CSC also promote a superior environment for pulpal repair and bridge formation, compared to CH products.[193,238,260,286] MTA and CSC are hygroscopic cements that set in the presence of blood and serum, produce a gap-free interface with dentin, and generate a sustained alkaline pH; in addition, the surface morphology of the hardened cement allows for predictable bonding with current adhesion systems. Growth factors necessary for hard tissue formation are activated by CSCs through the gradual release of calcium ions during curing.[222] The small particle size and alkaline pH contribute to the entombment of remaining cariogenic bacteria at the dentin-MTA and CSC interface, impeding bacterial ingression and caries progression and discouraging continued pulpal injury (Fig. 24.15).[288,409,414]

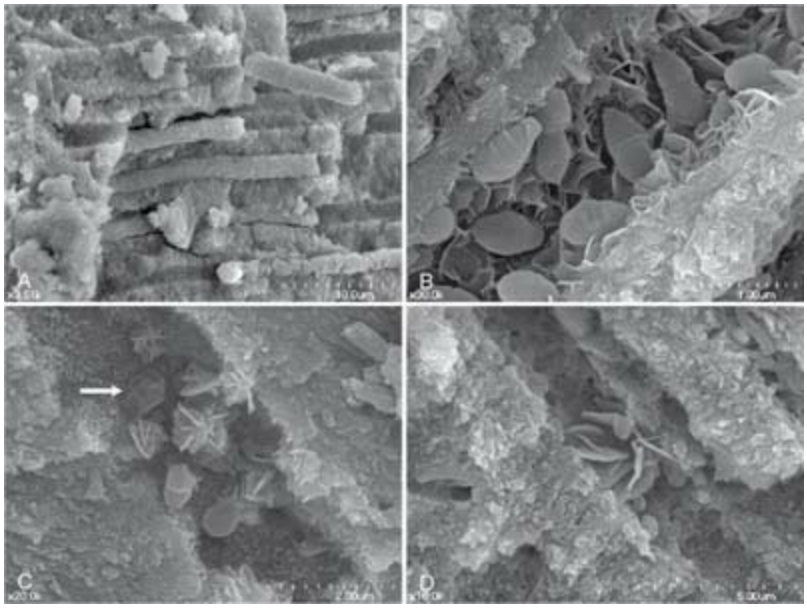

FIG. 24.15 Scanning electron micrographs of dentinal tubule obturation by calcium silicate cement (CSC) at the dentin/CSC interface after canals were seeded with *E. faecalis* in phosphate buffered saline (PBS). **A,** Tag formation in dentinal tubules at dentin interface in after application of OrthoMTA (BioMTA, Seoul, Korea) (×35,100). **B,** Four-week image showing *E. faecalis* entombed by growing crystalline structures within the dentinal tubule (×30,000). **C,** *White arrow* shows deformation and damaged cell membrane of *E. faecalis* within an aggregation of leaflet-like and needle-like crystals at 16 weeks (×20,000). **D,** Sixteen-week specimen showing complete

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blockage of the dentinal tubule lumen by CSC tight crystalline formation (×20,000).

Set of four scanning electron micrographs is marked A through D.

A) It has tubular structures placed on one above other.

B) It has oval structures in the wide canal with the porous background.

C) The oval structures have white thread-like structures on them.

D) The oval structures are clustered together in the narrow canal.

Source: (Reprinted from Yoo JS, Chang SW, Oh SR, et al: Bacterial entombment by intratubular mineralization following orthograde mineral trioxide aggregate obturation: a scanning electron microscopy study. *Int J Oral Sci* 6:227, 2014, with permission.)

Pulpotomy with calcium silicate cements

It has been recommended that pulpectomy be avoided in immature permanent teeth with vital canal tissue so as to protect the remaining radicular pulp tissue and thus encourage continued root development and apexogenesis.[140,335] Partial pulpotomy to treat direct pulp exposures in immature permanent teeth using CH products has been shown to be a reliable treatment option with proper case selection.[73,335] However, comparable success rates ranging from 83% to 100% have been demonstrated using CSCs for pulpotomies in both immature and mature permanent teeth.[23,25,39,242,313,361–363,398,399] Moreover, pulpotomies completed with MTA/CSCs do not show complications such as internal resorption, typically seen with CH, formocresol, and ferric sulfate pulpotomies (Fig. 24.16).[126,195,324]

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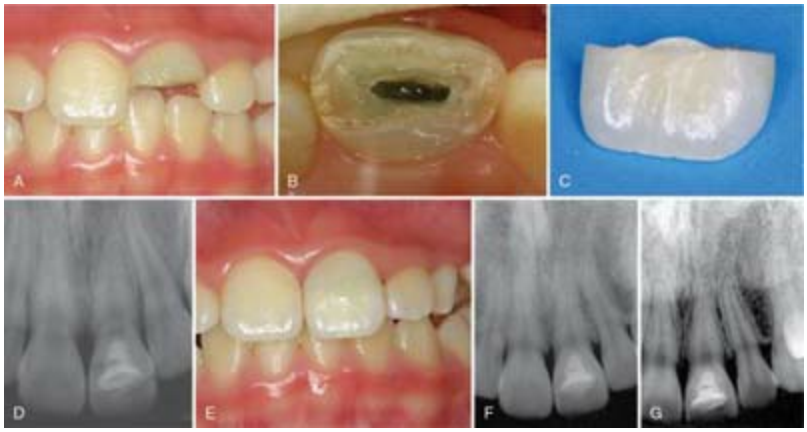

FIG. 24.16 Eleven-year-old female presented 3 hours after trauma with a horizontal crown fracture of the maxillary left central incisor. **A.** Clinical photograph showing midcoronal horizontal fracture. The pulp was exposed and exhibited normal vitality. **B,** Photograph of access cavity after Cvek pulpotomy and placement of gray mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK). **C,** Photograph of fractured incisal segment before bonding and reattachment. **D,** Six-month radiograph with bonded fractured segment showing initial reparative bridge formation with absence of apical pathosis. The incisor was asymptomatic and responded normally to cold sensibility testing. **E,** Clinical photograph of incisor at 6-month recall. **F,** Eight-year radiographic review. **G,** Eleven-year radiographic recall. Tooth was asymptomatic wth normal mobility, probings, and positive response to cold test.

Set of seven radiographs and photographs marked A through G shows the binding of detached segment in the central incisor of upper jaw.

Source: (Courtesy Dr. Winston Chee and Dr. Stefan Zweig, Los Angeles, CA.)

Adult pulpotomy using CSCs is a promising treatment strategy of considerable interest. More important is the inclusion of symptomatic teeth in clinical investigations that exhibit irreversible pulpitis and show apical radiolucencies consistent with advanced pulp inflammation or partial pulp necrosis.[23,25,39,242,313,361–363,398,399] The potential for resolution of apical disease in teeth with inflamed pulps displaying apical rarefaction after pulpotomy procedures has been previously documented.[66,208] One multicentered randomized controlled trial complemented by an earlier study evaluated randomly allocated MTA and CEM adult pulpotomies at 2 and 5 years. The clinical outcomes for both materials were ≥98%, showing equal effectiveness for both CSCs in permanent molars associated with irreversible

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pulpitis and apical periodontitis.[22,24] Another investigation by the same group examined pulpotomy outcomes for CEM measured against conventional orthograde root canal therapy in mature molars diagnosed with irreversible pulpitis. It demonstrated comparable outcomes in pain reduction and the resolution of apical periodontitis, with continued tooth function and survival after 5 years (Fig. 24.17).[23] Both studies showed postoperative pain reduction and the resolution of apical pathosis in the majority of cases. Other investigations have confirmed significant pain reduction in symptomatic permanent teeth with deep caries using CSC pulpotomy.[152,361] Concurrent studies corroborate findings that pulpotomy treatments performed in mature permanent teeth with irreversible pulpitis and advanced caries using CSCs are a viable future treatment alternative.[22,23,25,242,361–363]

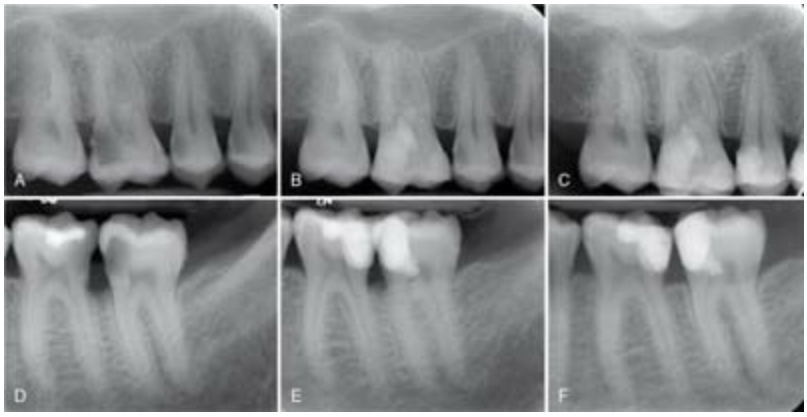

FIG. 24.17 Pulpotomy procedures completed for two patients clinically diagnosed with irreversible pulpitis using calcium enriched mixture (CEM) cement (BioniqueDent, Tehran, Iran). **A,** Preoperative radiograph from 27-year-old male patient with deep caries associated with maxillary right quadrant. The first molar was symptomatic with calcified tissue evident in the pulp chamber. **B,** Radiograph after complete pulpotomy using CEM cement restored with bonded composite. **C,** Five-year radiographic recall. The tooth was asymptomatic, firm, with normal probings. **D,** Preoperative radiograph showing deep caries in the mandibular left first (#19) and second (#18) molars in a 41-year-old male patient. Both teeth demonstrated lingering pain to cold testing and tooth #18 was sensitive to percussion. **E,** Postoperative radiograph showing completed miniature pulpotomy #19 and partial pulpotomy #18 after sodium hypochlorite hemostasis and application of CEM cement. Both molars were restored with bonded composites. Tooth #18 demonstrated resolution of symptoms after 24 hours. **F,** Six-year radiographic recall, patient remained asymptomatic

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and both teeth registered a positive response to sensibility testing.

Set of six radiographs marked A through F shows the fillings in radiolucent patches of crown in teeth 18 and 19.

Source: (Courtesy Dr. Saeed Asgary, Tehran, Iran.)

Indications for pulpotomy have recently transitioned from immature to mature teeth using CSCs, which may improve consumer acceptance and retention rates for patients seeking alternatives to conventional root canal therapy and protective ancillary care. Teeth that present with irreversible pulpitis and experience carious pulp exposures are normally treated with pulpectomy and orthograde root canal treatment coupled with restorative procedures designed to protect the tooth from fracture and microleakage. However, issues with cost effectiveness, holistic concerns, pain management, treatment time, and need for additional appointments when compared to orthograde root canal therapy are strong arguments in favor of this newer treatment option. Pulpotomy in permanent teeth using CSC materials can be recommended as a minimally invasive alternative to traditional root canal treatment.

Vital pulp therapy techniques

Diagnosis

A differential diagnosis based on clinical symptoms and radiographic findings is the aim in assessing pulp vitality. However, an accurate determination of the pulpal condition before treatment initiation is more challenging in younger patients.[356] Establishing a diagnosis of reversible versus irreversible pulpitis in immature teeth can be complicated by subjective symptoms and testing responses that may not accurately reflect pulp histopathology.[73] However, efforts should be directed toward the ultimate goal of pulpal preservation and continued apexogenesis in immature permanent teeth.[53,54] A diagnosis of irreversible pulpitis, based on signs and symptoms, along with clinical testing procedures, does not preclude vital pulp therapy options. Regardless of the treatment choice of pulp capping or partial or complete pulpotomy, preservation of the radicular pulp and apical

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papilla allows for root maturation in cases of trauma or deep caries.[146,147,169,342]

Diagnostic quality intraoral radiographs of the involved tooth must be taken to evaluate accurately the extent of root formation and periradicular or furcation changes associated with the periodontal ligament and supporting bone.[256] In young permanent teeth, the stage of root development directly influences the diagnosis and treatment options.[74] Because the faciolingual dimension of most immature roots is greater than the mesiodistal dimension, apical closure may be difficult to determine radiographically.[74,221] Teeth that demonstrate radiographic evidence of deep caries should not be planned for aggressive procedures, such as pulpectomy, without the benefit of thermal (cold) testing (see Fig. 24.1).

Before arriving at treatment decisions, the clinician should carefully assess all available information; the medical history, patient report, radiographic evidence, clinical evaluation, and vitality (cold) testing are recommended. Periodontal probing, mobility assessment, and the presence of any localized swelling or sinus tracts should be recorded during the evaluation. Radiographs, including bitewings and periapical views, should be evaluated for periapical and furcation pathosis, resorptive defects, and pulpal calcification resulting from trauma or previous restorations.

Patients with deep carious lesions often experience sensitivity to cold, heat, or sweet or acidic foods, and cold tests may evoke a short lingering response of 1 to 2 seconds. This may not be a definitive indicator that the pulp is irreversibly damaged. Determination of the pulpal condition with the aid of contemporary testing methods can be challenging, even for experienced clinicians, because of possible excessive responses to pulp percussion and palpation testing in children.[148,213,396] Clinical evidence indicates that cold testing with carbon dioxide ice is a more reliable prognosticator of pulp status in immature permanent teeth than electronic testing devices.[54] However, a diagnosis of irreversible pulpitis or pulp necrosis should be considered for teeth that generate pain on percussion.

Clinically, the difference between reversible and irreversible pulpitis is often determined on the basis of the duration and intensity of pain.[147] Unprovoked, spontaneous pain of long duration or unrelenting symptoms forcing sleep deprivation are consistent with irreversible pulp inflammation

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or an acute periapical abscess.[50] The difficulty in predictably assessing pulpal health and potential degrees of inflammation has been recently addressed in an attempt to improve current terminology that may not accurately reflect pulp conditions during the inflammatory process.[401] It has been proposed that the terms *initial*, *mild*, *moderate*, and *severe pulpitis* be used to replace current terminology for reversible and irreversible pulpitis, better reflecting conditions encountered in the clinical setting. The recategorization of stages of pulpal inflammation attempts to better direct minimally invasive treatment selections using CSCs and is designed to improve outcomes for pulp tissue preservation. However, these assessments can be more complicated than previously proposed, as diagnosed pulp conditions are not always consistent with clinical findings during treatment.[321,401]

Another consideration in a patient with displacement trauma is transient apical breakdown, which mimics periapical radiolucencies.[16] Teeth that experience luxation-type injuries can discolor and may not respond to cold testing for up to 4 months before they recover normal color and vitality. Also, biologically or pharmacokinetically immunosuppressed patients may not respond to conventional treatments because of abnormal function of related repair mechanisms.[245,389] Most clinical investigations clearly indicate that successful outcomes for vital pulp therapy decrease as the patient's age increases. Although aging of the pulp diminishes pulpal volume, vascularity, and host immune responses, functional repair mechanisms can still provide favorable treatment outcomes in older patients (Fig. 24.18).[1,270]

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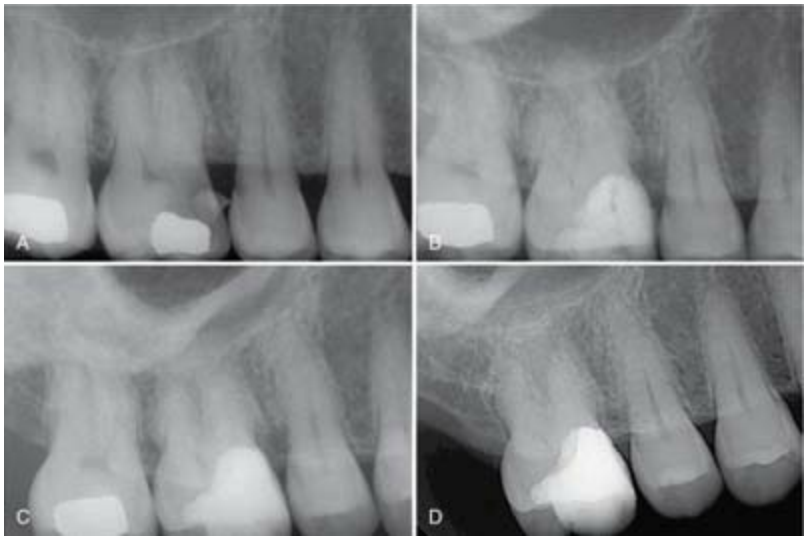

FIG. 24.18 A 51-year-old patient who presented with a deeply carious but asymptomatic maxillary right first molar. **A,** Preoperative radiograph reveals extensive mesial caries and an occlusal amalgam. **B,** Postoperative radiograph after 1.5 mm pulpal exposure, sodium hypochlorite hemostasis, mineral trioxide aggregate (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) direct pulp cap, wet cotton pellet placement, and Photocore provisionalization. **C,** Radiograph 1 week after pulp capping and placement of a permanent bonded composite restoration. The patient was asymptomatic and showed a positive response to cold testing. **D,** Six-year radiographic recall; cold testing revealed normal vitality.

Set of four radiographs marked A through D shows first molar with radiolucent patch on left side of crown restored after composite fillings.

Source: (© Dr. George Bogen.)

The initial pulpal diagnosis can be confirmed after visualization of the exposed pulp and assessed during tissue hemostasis. If no hemorrhaging is seen, this area of the tissue is most likely necrotic and must be removed with a high-speed round diamond bur until bleeding is evident (Fig. 24.19). After hemostasis with NaOCl, a large bulk of CSC can be placed directly against the remaining tissue. Alternatively, if hemorrhage control cannot be achieved after 5 to 10 minutes of direct contact with 3% to 6% NaOCl, the pulp is likely to be irreversibly involved, and a full pulpotomy or pulpectomy is recommended.

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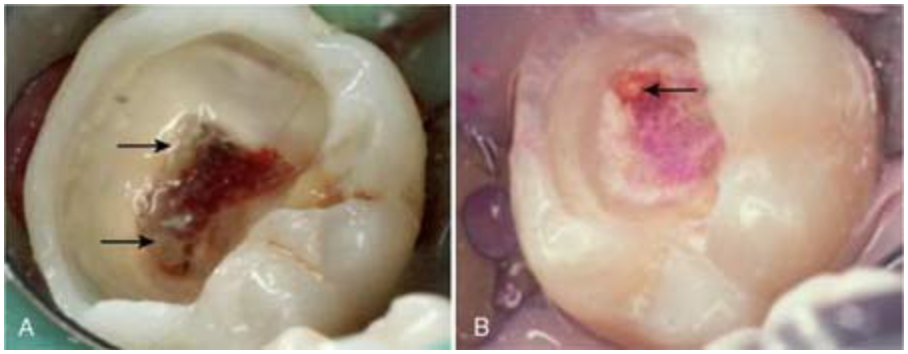

FIG. 24.19 Clinical examples of diseased pulp tissues after sodium hypochlorite hemostasis. **A,** Photograph of exposed pulp tissue of a mandibular right first molar in a 13-year-old patient. Note necrotic pulp tissue *(arrows)* that was subsequently removed with the remaining coronal pulp during a complete pulpotomy procedure. **B,** Clinical presentation of mandibular right molar in a 7-year-old patient after pulp exposure during caries excavation using a caries detector dye. Note the extruded seminecrotic, nonhemorrhagic tissue *(arrow)*. The tooth later underwent mineral trioxide aggregate partial pulpotomy and permanent restoration.

A) Close-up view shows wide cavity in the tooth with tissue residues and diminished bleeding.

B) Close-up view of stained tooth shows a lesion in the cavity.

Source: (© Dr. George Bogen.)

Although the size of the pulp exposure has no significant bearing on the final outcome, some clinicians falsely assume that larger exposures have an unfavorable prognosis.[252] Pulp sizes are underestimated on radiographs.[82–84] The size of the pulp exposures may also be overestimated, which could affect the decision-making process, leading clinicians to abandon more conservative vital pulp treatment options.[163] Pulp dimensions may also vary between various racial groups and genders.[83,346,392]

Teeth that have a history of trauma or previous restorations or that display pulpal calcification have a poorer prognosis than teeth showing only initial caries. In the selection of a specific vital pulp treatment, it is important to consider the remaining tooth structure and future restorative plan. In patients with uncontrolled caries or extensive loss of coronal structure in which full coverage is indicated, pulpotomy rather than pulp capping is recommended.[60,73,373]

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Caries removal

The main objective in caries removal is the identification and complete removal of infected tissue while preserving sound tooth structure; this contributes to pulpal protection and continued vitality. Caries removal is enhanced with the aid of a caries detector dye and optical magnification; however, it must be understood that dyes can cause excessive and unnecessary removal of healthy tooth structure.[37,255,408]

Caries removal has traditionally been completed somewhat subjectively using hand instruments and slow-speed burs. The procedure is performed using an explorer and tactile sense to differentiate soft from hard dentin to determine infected from noninfected dental tissue. However, this method has shortcomings because clinicians may leave decay at the dentinoenamel junction and unnecessarily remove dentin that has the potential to remineralize under a sealed restoration.[149] Furthermore, it has been found that the ability to remove caries varies among operators and during different time periods for the same operator.[149,150]

Investigators in the early 1970s used SEM to identify two different layers of carious dentin.[150] Teeth show two layers as the result of gram-positive bacteria releasing lactic acid as their main by-product. The outside carious layer subjacent to the enamel-dentin junction exhibited demineralized hydroxyapatite crystals that were dissolved by acidic bacterial by-products; this layer also featured unbound and altered collagen denatured by microbial proteolytic enzymes.[312] The second demineralized carious layer proximal to the pulp featured degraded hydroxyapatite crystals but contained collagen with intact intermolecular cross links unaffected by cariogenic acids.[149,150,331] If this deeper layer can be preserved during caries excavation, the remaining pulp tissue and odontoblasts will be subjected to less trauma, contributing to pulpal protection and survival. [282,326] This layer has a stronger capacity to remineralize when paired with bonded composite restorations to prevent bacterial microleakage.[269,282]

The two carious layers have been further classified into four zones when analyzed by atomic force microscopy and transverse digital microradiography.[312] Consistent with previous investigations, the four zones reinforce the concept that increasing levels of demineralization decrease the

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peritubular dentin rating and mechanical properties of dentin.[312]

Investigations in human, dog, and nonhuman primate models have demonstrated this regenerative characteristic in caries-affected dentin.[208,215,269,370,371] Several studies have questioned the efficacy of caries removal using a caries detector dye. Not all stainable dentin can be classified as infected, and the absence of staining does not eliminate the potential for residual cariogenic bacteria.[218,312] However, dyes allow the operator to visually inspect, with the aid of the DOM, infected dentin that may have been overlooked, particularly at the dentinoenamel junction, where it may compromise treatment outcome.[218,407] Although a compromise, it may be clinically preferable to inadvertently remove a small excess of dentin than to leave infected tissue.

Hemostatic agents

A wide range of hemostatic solutions and methods have been recommended to control a bleeding pulp exposure. These include various concentrations of NaOCl; 2% CHX; MTAD (Tulsa/Dentsply, Tulsa, OK); 30% hydrogen peroxide (Superoxol); ferric sulfate; disinfectants, such as Tubulicid (Global Dental Products, North Bellmore, NY); epinephrine; direct pressure with cotton pellets soaked in sterile water or saline; and the use of lasers.[8] NaOCl in concentrations of 1.5% to 5.25% is currently regarded as the most effective, safe, and inexpensive hemostatic solution for pulp capping and partial and complete pulpotomy procedures.[174,397] First used as a wound antiseptic during World War I and referred to as Dakin's solution, it was soon introduced for root canal irrigation.[100,104] NaOCl became a valuable hemostatic agent for direct pulp exposures in the late 1950s.[173,194,360] The antimicrobial solution provides hemostasis and disinfection of the dentin-pulp interface, chemical amputation of the blood clot and fibrin, biofilm removal, clearance of dentinal chips, and removal of damaged cells at the mechanical exposure site.[39,126,173,249] Concentrations of 1.5% to 5.25% in direct contact with pulp tissue do not appear to adversely alter pulp cell recruitment, cytodifferentiation, and hard tissue deposition.[121] Rinsing the exposed pulp tissue for 15 minutes with 5 mL NaOCl 5.25% (Clorox) leads to dissolution effects, but these effects are limited to the 3 to 5 upper cell

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layers without any changes in deeper pulp areas or the dentin. Perfused vital pulp tissue also buffers the chemical activity of NaOCl.[325]

An investigation into pulp-capped human third molars treated with either CH or a self-etching adhesive system examined pulps histologically at 30 and 60 days after the use of 2.5% NaOCl for hemostasis.[133] Histologic evidence demonstrated no impairment of the repair process after the use of NaOCl, although CH appeared to outperform the resin-based pulp capping agent. Current data clearly support the use of 1.25% to 5.25% NaOCl solutions in humans as a safe and appropriate hemostatic agent for DPC and pulpotomies.[5,34,121,133,173,192,252,384,388,397]

When pulp exposures occur in a carious field, the ability to attain hemostasis remains the most crucial factor in the success of vital pulp therapy. This was demonstrated in an innovative study that examined outcomes of teeth directly pulp capped with a hard setting CH after exposures were generated during caries excavation.[252] Caries removal during the investigation was aided by a caries detector dye with 10% NaOCl for hemostasis. The 2-year success rate was 81.8%. A statistical analysis of key factors revealed that preoperative thermal responses, percussion sensitivity, the diameter of the exposure, the age of the patient, and the tooth type and location had no significant influence on the outcome. The degree of bleeding and its control at the time of exposure constituted the most critical predictor of outcome. When hemostasis can be attained, pulpal repair and reparative dentin formation can proceed normally, in the absence of microbial challenges, when MTA and other CSCs are used as the pulp dressing.[87,210]

Sodium hypochlorite is an effective hemostatic agent and a valuable tool for assessing the difference between irreversibly and reversibly inflamed pulps as continued hemorrhaging after a 10-minute exposure strongly indicates irreversible pulpitis (Fig. 24.20).

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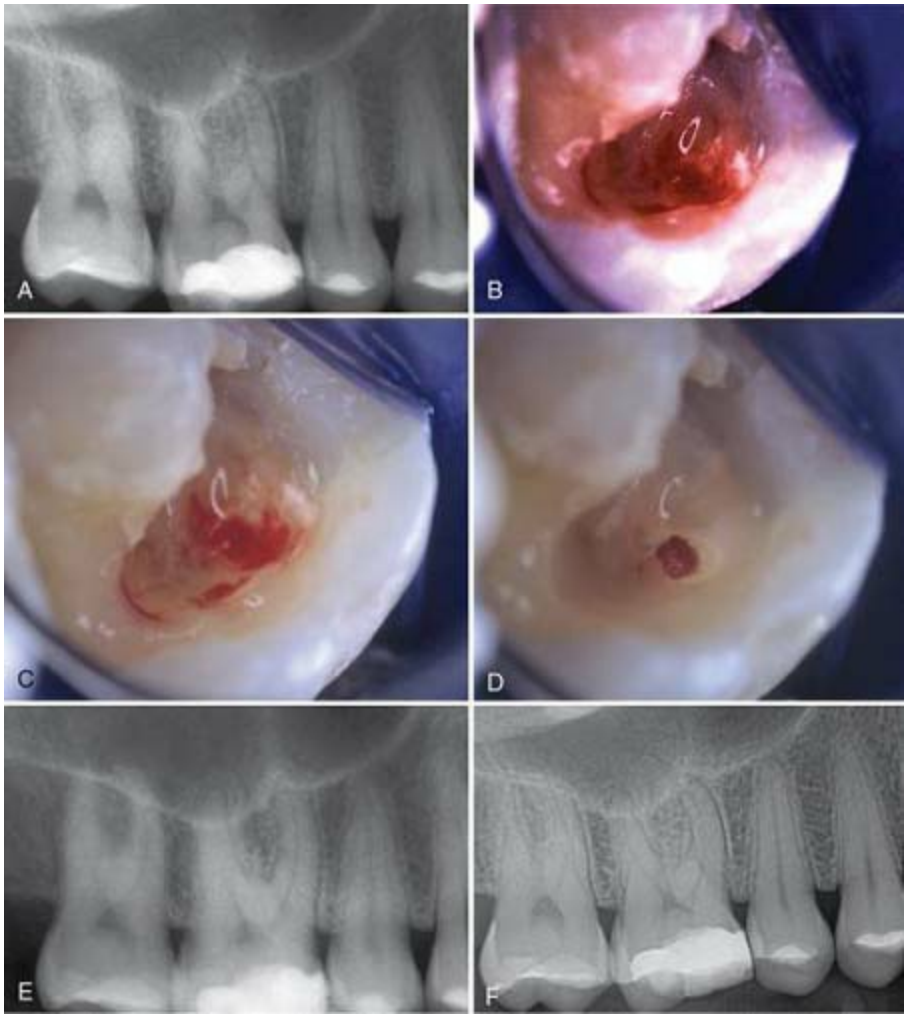

FIG. 24.20 Thirty-four-year-old female patient presented for root canal treatment with temporary restoration 1 week after caries excavation and pulp exposure of the maxillary right first molar (#3). The patient had mild discomfort and responded with a short lingering pain to cold testing. **A,** Periapical radiograph showing temporary restoration in tooth #3 with normal apical structures. **B,** Photograph of pulpal hemorrhage after temporary removal. **C,** Clinical photograph showing diminished bleeding after 5-minute exposure to 5.25% sodium hypochlorite. **D,** Photograph showing hemorrhage controlled in 10 minutes and 2 mm pulp exposure after continued caries excavation. The exposure and peripheral dentin received bulk OrthoMTA (BioMTA, Seoul, Korea) pulp capping and was covered with a thin wet cotton pellet and provisionalized with Photocore. The tooth was restored permanently with composite resin 1 week later. **E,** Two-year 6-month radiographic follow-up; tooth responded positive to sensibility testing. **F,** Control radiograph at 4 years. Patient exhibited normal pulp testing response and was asymptomatic with normal mobility and probings.

Set of radiographs and photographs marked A through F shows the changes in

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tooth number 3 during restoration.

Source: (© Dr. George Bogen.)

Treatment considerations

An important and often unrecognized aspect of DPC in a carious field is the potential survival of undetected bacteria in tubules adjacent to the exposure site even after caries removal. These microorganisms can compromise treatment even after meticulous excavation and disinfection. Unfortunately, traditional pulp capping protocols have directed clinicians to place pulp dressings only at the exposure site, without considering the adjacent dentin. It is therefore recommended that CSCs be placed over the exposure sites and onto the surrounding dentin to entomb residual microorganisms (see Fig. 24.15).[409,410]

The concept of biomineralization and bacterial entombment is particularly important when caries removal protocols are ineffective at completely removing undetected acidogenic microorganisms. During the CSC hydration process, calcium-deficient crystalline formation within the dentinal tubules produces bacterial entrapment, entombment, and neutralization.[409,414] This notable change in pulp capping strategy, based on CSC crystallization properties, can improve treatment outcomes in symptomatic and asymptomatic teeth featuring extensive caries. This includes cavities with multiple exposures after caries excavation or partial and complete pulptomies when indicated.[54] Together with an extensive bulk application of MTA/CSC to provide a cement thickness of 1.5 mm or greater, there is an increased likelihood of bacterial neutralization, minimizing further microbial challenges.

A disadvantage of CSCs is their long setting time of up to 228 minutes for ProRoot MTA and up to 85 minutes for Biodentine.[217] Thus according to the manufacturers' recommendation, CSCs should be allowed to set for a 15-minute minimum (e.g., Biodentine) or 2.5 hours (ProRoot MTA) before a permanent restoration can be placed. Nevertheless, in clinical use waiting even 15 minutes for set can lead to complications. To overcome these clinical drawbacks and to bypass the long setting time, it has been suggested to cover the MTA or CSC with an RMGI, compomer, or flowable composite resin.

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The final restoration is then placed allowing the cement to set beneath the interlayer.[53,75]

Recently it was shown that 3 minutes after mixing MTA or Biodentine, it may be covered with a thin layer of a self-etching, self-bonding flowable composite resin (Vertise flow), an RGMI, or composite resin in combination with a self-etching dentin adhesive. All these restorative materials achieved shear bond strengths to Biodentine or MTA that were similar to those after 15 minutes and 2 days. A longer waiting time after mixing did not increase the adhesion of the lining materials to MTA or Biodentine.[333] Furthermore, the results of previous studies indicated that when placing RMGI on these cements, the time span between the mixing of CSCs and the application of RMGI did not influence the setting reaction of the cement. The moisture of the CSC had no impact on the setting reaction or setting time of RMGI or on the MTA-RMGI structural interface.[35,131,287] A final permanent restoration can therefore be provided at the same appointment.

Treatment recommendations†

Pulp capping and pulpotomy

1. After profound local anesthesia is achieved, the clinical crown is disinfected with NaOCl or CHX after dental dam isolation. Optical magnification and illumination (ideally with a DOM) are highly recommended. The cavity outline is completed using high-speed burs under constant water cooling.
2. Caries excavation is completed using long shank #2-6 round burs in a slow-speed handpiece augmented by hand instrumentation. Removal of caries may be guided by a detector dye if a DOM is used.
3. The cavity and exposure site(s) are immersed with 1.5% to 5.25% NaOCl. Heavy bleeding may be controlled with a cotton pellet moistened with NaOCl under moderate pressure for 5 minutes. If no bleeding is present after the pulp exposure, the area must be examined for necrotic tissue. This is removed with a high-speed round diamond bur under water coolant until bleeding tissue is exposed. Uncontrolled hemorrhaging after 10 minutes can be

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managed with a full pulpotomy or pulpectomy after assessing the radicular tissue condition. It is CRITICAL that all necrotic tissue is removed (see Fig. 24.19).

4. Prepare MTA or CSC according to the manufacturers' instructions.
5. Using a suitable instrument (composite instrument, Glick #1 endodontic instrument, or MTA carrier gun), apply a large bulk of CSC over the exposure side, including most of the surrounding dentin at a thickness of 1.5 to 3 mm.
6. Remove excess moisture at the site with a dry cotton pellet and leave a clear periphery of dentin for adhesive bonding. A total etch and rinse technique with a phosphoric acid gel is not possible at this stage because the MTA or CSC can be washed away. Either the CSC must show a firm set or a flowable composite/RMGI material must been accurately placed and completely cover the cement.
7. Apply a small amount of a light-curing flowable compomer, an RMGI (or an equivalent light-cured resin–glass ionomer liner), a self-etching, self-bonding flowable composite resin or a flowable composite resin in combination with a self-etching dentin adhesive to cover the CSC and light-cure according to the material's instructions. NOTE: Placement of flowable resins can be more challenging in cases of DPC than indirect pulp capping and pulpotomy procedures.
8. Etch the remaining cavity walls with 34% to 37% phosphoric acid gel for 60 seconds, rinse thoroughly and apply a dentin adhesive (fourth generation, two-bottle systems are highly recommended). Avoid using self-etching dentin adhesives (single-bottle systems).[290] Cure according to manufacturers' instructions.
9. Place a composite resin material incrementally as a permanent, final restoration. Light-cure according to manufacturer's instructions.
10. Pulp vitality should be assessed at the next appointment, preferably at 7 to 10 days using a cold test. Radiographic assessment is only needed in case of negative sensibility testing or onset of pain during the first year. Assessments can be made yearly or every 2 years, if indicated.

The permanent restoration

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A definitive restoration should be provided during the pulp treatment appointment. Temporary restorations over MTA or CSC may lead to significantly lower success rates.[181,260] It has been reported that teeth which were permanently restored more than 2 days after DPC with MTA exhibited a diminished prognosis.[260] The negative impact of the time between DPC and placement of the permanent restoration was also shown for Biodentine and CH.[110,181] However, if the permanent restoration cannot be placed at the time of definitive pulp treatment due to management concerns, nonbonded translucent core materials such as Photocore (Kuraray Co. Ltd., Osaka, Japan) can provide acceptable protection for extended periods when used in conjunction with CSCs.[39]

The placement and quality of the permanent restoration can be crucial to the long-term maintenance of pulp vitality and may be more significant than the actual pulp treatment.[12,142,188,257,264] The aim of the final restoration is to complement the sealing ability of the pulp capping/pulpotomy material and defend the pulp from further microbial challenges. The selection of the restorative material, procedure execution, and adherence to proper restorative protocols can all contribute to minimizing potential restorative microleakage. Microleakage around restorations may be more detrimental to vital pulp tissue than unfilled cavity preparations directly exposed to the oral environment.[330] An important consideration in both mature and immature permanent teeth is the conservation of the remaining tooth structure, which ultimately contributes to favorable long-term retention and function.[93,122,199,351] The incorporation of adhesive restorative materials as definitive final restorations minimizes tooth reduction, encourages anatomic preservation, and thus provides better pulpal protection and repair potential.[149,172]

Advances in dental materials have increased treatment options in the restoration of teeth that have undergone vital pulp procedures. Permanent restorations for pulp capped or pulpotomized permanent teeth can include composite resins, bonded or unbonded amalgam restorations, and cuspal coverage restorations. However, the more conservative the restorative treatment, the greater the probability of pulp survival.[149] Vital pulp therapy must be considered an injury to an already challenged connective tissue, and every effort should be directed toward minimizing further injurious

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procedures.[1] Factors that affect the repair mechanisms in pulp tissue can include the age of the patient, the depth and size of the cavity preparation, and the choice of restorative material.[283]

Total etch and self-etching systems produce excellent bond strengths to enamel, dentin, and cured CSCs.[27,288] The most durable bond strengths generated with contemporary materials are achieved using selective etching of the enamel with 34% to 37% phosphoric acid, followed by two-step, two-bottle, self-etching adhesive systems.[79,291,387] Current advances in adhesive technology have also shown that beneath the resin-interfused hybrid layer, an acid-base–resistant zone is formed that enhances the resistance of normal dentin to recurrent caries. This reinforced layer forms when monomer penetration and polymerization occur, and it demonstrates an ability to restrict (inhibit) primary and secondary caries.[290,291] As a result of their simplifications and improved bonding strengths, modern adhesives complement vital pulp therapy. Bonding procedures must strictly follow manufacturers' recommendations and be paired with dental dam placement.[79]

Postoperative follow-up and recall

After treatment completion, the pulp status must be assessed periodically to ensure continued pulp vitality, normal function, and apical closure in immature teeth. Cold testing and radiographic evaluation most accurately assess continued pulp health and are excellent predicators for measuring survival rates. Recalls can address postoperative sensitivity, pulpal degeneration or necrosis, and indications for further endodontic and restorative care. Review also allows detection of emerging complications such as recurrent caries, poor hygiene, restoration failures, and cuspal fractures.

Patient compliance for recall in asymptomatic cases can be challenging. Because some parents do not practice regular preventive care and may not have a strong background in basic oral health care, recall compliance rates for children can be unpredictable.[205,311] Although recall has been traditionally based on a 6-month checkup and oral prophylaxis period, this has come into question.[265] Recalls can be tailored individually, based on patient need, symptomology, caries indices, periodontal status, and craniofacial

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development assessment in younger patients.[292,391]

A tentative diagnosis of tooth survivability can be made at the 3-month recall.[252] One study demonstrated that the prognosis for long-term pulp survival can be established at an observation period of 21 to 24 months.[252]

Radiographic evidence of root-end closure in immature adult teeth is a reliable prognostic marker of continued pulp vitality (Fig. 24.21).[36] Apexogenesis after DPC or pulpotomy procedures using CSCs should proceed normally in healthy patients at a progressive rate.[39,305,394,400] The observed root maturation should also follow a predictable pattern of tooth development that coincides with the contralateral teeth in the same patient when the teeth are compared radiographically and viewed chronologically.[36]

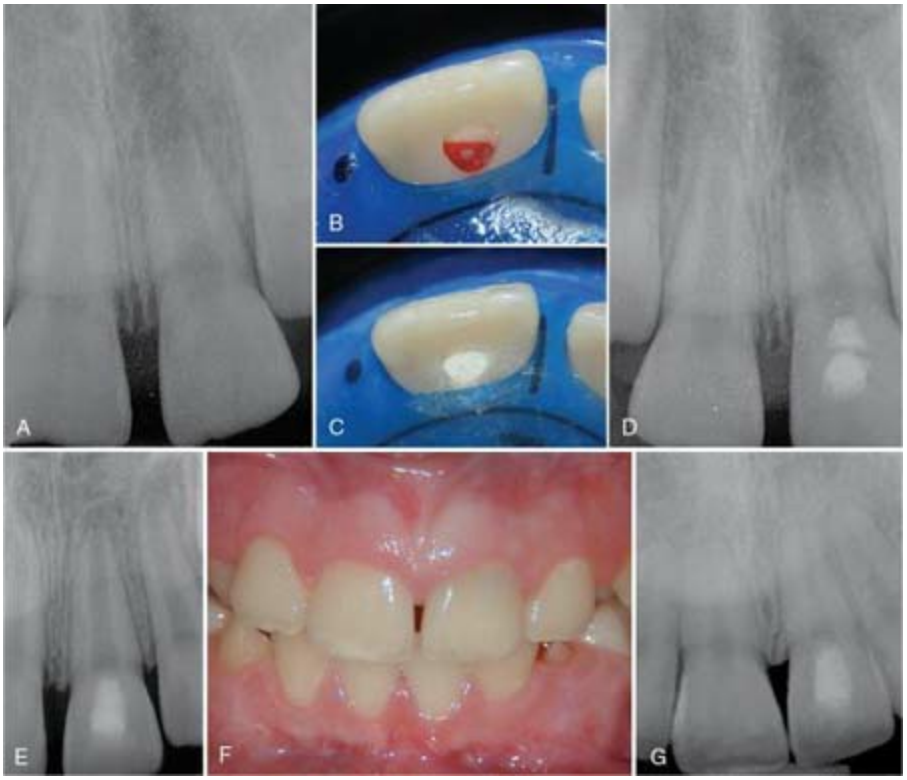

FIG. 24.21 An 8-year-old male presented following a traumatic injury to the maxillary left central incisor 2 months earlier. **A,** Periapical radiograph revealed an open apex with a developing radiolucency. The tooth was sensitive to palpation, percussion and negative to cold testing. The diagnosis was necrotic pulp and acute apical periodontitis. **B,** Clinical photograph after access cavity preparation, vital pulp tissue visible coronally. The diagnosis was changed to reversible pulpitis and a partial (Cvek) pulpotomy was performed. **C,** Photograph of white

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mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) applied over the pulp to a thickness of approximately 3 mm. **D,** Radiograph after MTA pulpotomy with provisional restoration. A definitive composite restoration was placed 1 week later. **E,** One-year radiographic recall showing advancing apical closure. **F,** Four-year clinical photograph reveals a slight grey discoloration from the MTA in the cervical area of the tooth. Parents declined option of internal bleaching. **G,** Radiographic review at 9 years reveals apical maturation and thickening of the root walls. The tooth responded positively to cold sensibility testing.

Set of radiographs and photographs marked A through G shows the recovery of wide radiolucent canal of left incisor in upper jaw.

Source: (Reprinted courtesy Dr. Marga H. Ree, Purmerend, Netherlands with permission.)

Observation of contralateral tooth development can be an invaluable method of measuring the success of vital pulp therapy when using CSCs. Alternatively by comparison, in traumatized teeth that exhibit necrotic pulps and periradicular pathosis, CSCs can be used as an apical plug to stimulate the apical papilla to promote barrier formation, which may require 5 to 20 months for completion (apexification).[7,90,344] If MTA or another CSC is substituted for CH in vital pulp therapy procedures or apexification, similar time periods for apical maturation or barrier formation can be anticipated (Fig. 24.22).[132,259 261]

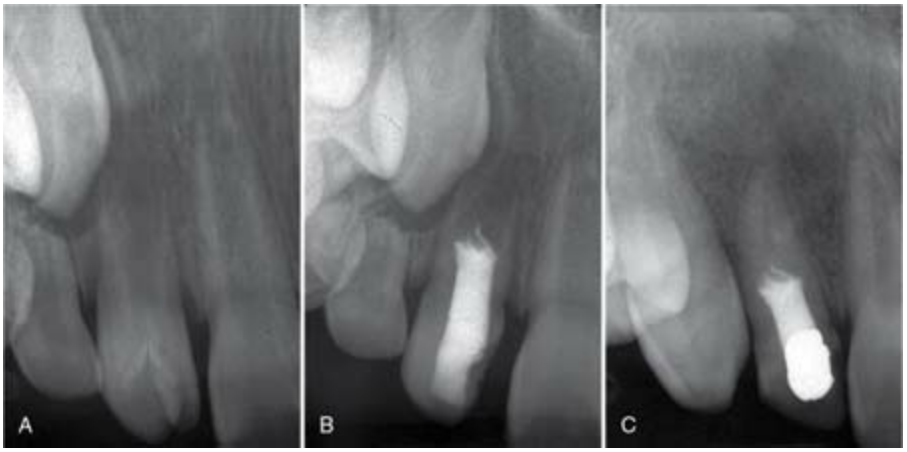

FIG. 24.22 **A,** Preoperative radiograph of a 7-year-old male patient exhibiting a dens evaginatus anatomical anomaly associated with the maxillary right lateral incisor. The parents had been advised to extract

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the tooth, which was symptomatic and nonresponsive to vitality tests. **B,** A periapical radiograph after cleaning the coronal portion of the canal and irrigation with 2.12% sodium hypochlorite. A coronal plug using ProRoot MTA (Tulsa/Dentsply, Tulsa, OK) was placed and sealed with a provisional restoration. **C,** Four-year radiographic review demonstrates complete root formation with maturation and apical root closure. The tooth was asymptomatic with normal mobility and probings.

Three radiographs are marked A through C.

A) Three teeth are shown with wide open root canals.

B) The central tooth with improper half-filled root canal. The root canal is wide.

C) The central tooth with closed root apex with half-filled root canal.

Source: (Courtesy Dr. Mahmoud Torabinejad.)

In the absence of microbial challenges, the human dental pulp demonstrates an exceptional regenerative capacity when treated with CSCs independent of patient age. Together with our advances in pulp biology and dental materials, changes in treatment protocols that encourage dental pulp preservation and survival will ultimately contribute to improved dental health for all patients requiring vital pulp therapy.

Acknowledgment

We wish to thank Prof. Dr. Leif K. Bakland and Prof. Dr. Sergio Kuttler for their contributions to this chapter.

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References

1. Abou-Rass M. *The stressed pulp condition: an endodontic-restorative diagnostic concept*, J Prosthet Dent 1982;48: 264.
2. About I. *Recent trends in tricalcium silicates for vital pulp therapy*, Curr Oral Health Rep 2018; https://doi.org/10.1007/s40496-018-0186-y.
3. Accorinte ML, Loguercio AD, Reis A, et al: *Response of human dental pulp capped with MTA and calcium hydroxide powder*, Oper Dent 2008;33: 488.
4. Accorinte MLR, Loguercio AD, Reis A, et al: *Evaluation of two mineral trioxide aggregate compounds as pulp-capping agents in human teeth*, Int Endod J 2009;42: 122.
5. Accorinte Mde L, Loguercio AD, Reis A, et al: *Adverse effects of human pulps after direct pulp capping with different components from a total-etch, three-step adhesive system*, Dent Mater 2005;21: 599.
6. Aeinehchi M, Eslami B, Ghanberiha M, et al: *Mineral trioxide aggregate (MTA) and calcium hydroxide as pulp-capping agents in human teeth: a preliminary report*, Int Endod J 2003;36: 225.
7. Aggarwal V, Miglani S, Singla M. *Conventional apexification and revascularization induced maturogenesis of two non-vital, immature teeth in same patient: 24 months follow up of a case*, J Conserv Dent 2012;68:.
8. Akashi G, Kato J, Hirai Y. *Pathological study of pulp treated with chemicals after Er:YAG laser preparation*, Photomed Laser Surg 2006;24: 698.
9. Akhlaghi N, Khademi A. *Outcomes of vital pulp therapy in permanent teeth with different medicaments based on*

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review of the literature, Dent Res J (Isfahan) 2015;12: 406.
10. Al-Hezaimi K, Salameh Z, Al-Fouzan K, et al: *Histomorphometric and micro-computed tomography analysis of pulpal response to three different pulp capping materials*, J Endod 2011;37: 507.
11. Alghaithy RA, Qualtrough AJ. *Pulp sensibility and vitality tests for diagnosing pulpal health in permanent teeth: a critical review*, Int Endod J 2017;50: 135.
12. Alptekin T, Ozer F, Unlu N, et al: *In vivo and in vitro evaluations of microleakage around Class I amalgam and composite restorations*, Oper Dent 2010;35: 641.
13. American Academy of Pediatric Dentistry, Clinical Affairs Committee, Pulp Therapy Subcommittee. *Guideline on pulp therapy for primary and immature permanent teeth: reference manual 2012-13*, Pediatr Dent 2012;34: 222.
14. American Association of Endodontists. Glossary of endodontic terms ed 8 2016; Chicago.
15. Andelin WE, Shabahang S, Wright K, et al: *Identification of hard tissue after experimental pulp capping using dentin sialoprotein (DSP) as a marker*, J Endod 2003;29: 646.
16. Andreasen F. *Transient apical breakdown and its relation to color and sensibility changes*, Endod Dent Traumatol 1986;2: 9.
17. Araújo LB, Cosme-Silva L, Fernandes AP, et al: *Effects of mineral trioxide aggregate, Biodentine™ and calcium hydroxide on viability, proliferation, migration and differentiation of stem cells from human exfoliated deciduous teeth*, J Appl Oral Sci 2018;26: e20160629.
18. Arruda RA, Cunha RS, Miguita KB, et al: *Sealing ability of mineral trioxide aggregate (MTA) combined with distilled water, chlorhexidine, and doxycycline*, J Oral Sci

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www.konkur.in

2012;54: 233.
19. Arun A, Mythri H, Chachapan D. *Pulp vitality tests - an overview on comparison of sensitivity and vitality*, Indian J Oral Sci 2015;6: 41.
20. Asgary S, Ahmadyar M., Vital pulp therapy using calcium-enriched mixture. *an evidence-based review*, J Conserv Dent 2013;16: 92.
21. Asgary S, Eghbal MJ. *Treatment outcomes of pulpotomy in permanent molars with irreversible pulpitis using biomaterials: a multi-center randomized controlled trial*, Acta Odontol Scand 2013;71: 130.
22. Asgary S, Eghbal MJ, Bagheban AA. *Long-term outcomes of pulpotomy in permanent teeth with irreversible pulpitis: a multi-center randomized controlled trial*, Am J Dent 2017;30: 151.
23. Asgary S, Eghbal MJ, Fazlyab M, et al: *Five-year results of vital pulp therapy in permanent molars with irreversible pulpitis: a non-inferiority multicenter randomized clinical trial*, Clin Oral Investig 2015;19: 335.
24. Asgary S, Eghbal MJ, Ghoddusi J, et al: *One-year results of vital pulp therapy in permanent molars with irreversible pulpitis: an ongoing multicenter, randomized, non-inferiority clinical trial*, Clin Oral Investig 2013;17: 431.
25. Asgary S, Hassanizadeh R, Torabzadeh H, et al: *Treatment outcomes of 4 vital pulp therapies in mature molars*, J Endod 2018;44: 529.
26. Asgary S, Moosavi SH, Yadegari Z, et al: *Cytotoxic effect of MTA and CEM cement in human gingival fibroblast cells: scanning electronic microscope evaluation*, N Y State Dent J 2012;78: 51.
27. Atabek D, Sillelioğlu H, Olmez A. *Bond strength of adhesive systems to mineral trioxide aggregate with*

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www.konkur.in

different time intervals, J Endod 2012;38: 1288.
28. Atmeh AR, Chong EZ, Richard G. *Calcium silicate cement-induced remineralization of totally demineralized dentine in comparison with glass ionomer cement: tetracycline labelling and two-photon fluorescence microscopy*, J Microsc 2015;257: 151.
29. Atmeh AR, Chong EZ, Richard G, et al: *Dentin-cement interfacial interaction: calcium silicates and polyalkenoates*, J Dent Res 2012;91: 454.
30. Auschill TM, Arweiler NB, Hellwig E, et al: *Success rate of direct pulp capping with calcium hydroxide*, Schweiz Monatsschr Zahnmed 2003;113: 946.
31. Awawdeh L, Al-Qudah A, Hamouri H, et al: *Outcomes of vital pulp therapy using mineral trioxide aggregate or biodentine: a prospective randomized clinical trial*, J Endod 2018;44: 1603.
32. Awawdeh L, Hemaidat K, Al-Omari W. *Higher maximal occlusal bite force in endodontically treated teeth versus vital contralateral counterparts*, J Endod 2017;43: 871.
33. Babbush CA Fehrenbach MJ Emmons M Mosby's dental dictionary ed 2 2008; Mosby Elsevier Publishing St. Louis.
34. Bal C, Alacam A, Tuzuner T, et al: *Effects of antiseptics on pulpal healing under calcium hydroxide pulp capping: a pilot study*, Eur J Dent 2011;5: 265.
35. Ballal S, Venkateshbabu N, Nandini S, et al: *An in vitro study to assess the setting and surface crazing of conventional glass ionomer cement when layered over partially set mineral trioxide aggregate*, J Endod 2008;34: 478.
36. Ballesio I, Marchetti E, Mummolo S, et al: *Radiographic appearance of apical closure in apexification: follow-up*

Telegram: @dental_k

www.konkur.in

after 7-13 years, Eur J Paediatr Dent 2006;7: 29.
37. Banerjee A, Kidd EA, Watson TF. *In vitro validation of carious dentin removed using different excavation criteria*, Am J Dent 2003;16: 228.
38. Barnes IM, Kidd EA. *Disappearing dycal*, Br Dent J 1979;147: 111.
39. Barrieshi-Nusair KM, Qudeimat MA. *A prospective clinical study of mineral trioxide aggregate for partial pulpotomy in cariously exposed permanent teeth*, J Endod 2006;32: 731.
40. Barthel CR, Rosenkranz B, Leuenberg A, et al: *Pulp capping of carious exposures treatment outcome after 5 and 10 years: a retrospective study*, J Endod 2000;26: 525.
41. Basturk FB, Nekoofar MH, Günday M, et al: *The effect of various mixing and placement techniques on the compressive strength of mineral trioxide aggregate*, J Endod 2013;39: 111.
42. Baume LJ, Holz J. *Long term clinical assessment of direct pulp capping*, Int Endod J 1981;31: 251.
43. Bergenholtz G. *Effect of bacterial products on inflammatory reactions in the dental pulp*, Scand J Dent Res 1977;85: 122.
44. Bergenholtz G. *Evidence for bacterial causation of adverse pulpal responses in resin-based dental restorations*, Crit Rev Oral Biol Med 2000;11: 467.
45. Berger T, Baratz AZ, Gutmann JL. *In vitro investigations into the etiology of mineral trioxide tooth staining*, J Conserv Dent 2014;17: 526.
46. Bernick S, Nedelman C. *Effect of aging on the human pulp*, J Endod 1975;1: 88.
47. Berzins DW. *Chemical properties of MTA*, Torabinejad M Mineral trioxide aggregate: properties and clinical

Telegram: @dental_k

www.konkur.in

applications 2014; Wiley Blackwell Publishing Ames.
48. Bhavana V, Chaitanya KP, Gandi P, et al: *Evaluation of antibacterial and antifungal activity of new calcium-based cement (Biodentine) compared to MTA and glass ionomer cement*, J Conserv Dent 2015;18: 44.
49. Bimstein E, Rotstein I. *Cvek pulpotomy - revisited*, Dent Traumatol 2016;32: 438.
50. Bjørndal L, Darvann T, Thylstrupt A. *A quantitative light microscopic study of the odontoblast and subodontoblastic reactions to active and arrested enamel caries without cavitation*, Caries Res 1998;32: 59.
51. Bjørndal L, Fransson H, Bruun G, et al: *Randomized clinical trials on deep carious lesions: 5-year follow-up*, J Dent Res 2017;96: 747.
52. Bjørndal L, Reit C, Bruun G, et al: *Treatment of deep caries lesions in adults: randomized clinical trials comparing stepwise vs direct complete excavation, and direct pulp capping vs partial pulpotomy*, Eur J Oral Sci 2010;118: 290.
53. Bogen B, Chandler N. *Vital pulp therapy*, Ingle J Bakand L Baumgartner J Ingle's endodontics 2008; BC Decker Publishing Hamilton.
54. Bogen G, Chandler NP. *Pulp preservation in immature permanent teeth*, Endod Topics 2012;23: 131.
55. Bogen G, Chandler NP. *Vital pulp therapy*, Rotstein I Ingle JI Ingle's endodontics ed 7 2019; PMPH U.S.A Raleigh.
56. Bogen G, Kim JS, Bakland LK. *Direct pulp capping with mineral trioxide aggregate: an observational study*, J Am Dent Assoc 2008;139: 305.
57. Bortoluzzi EA, Broon NJ, Bramante CM, et al: *Sealing ability of MTA and radiopaque Portland cement with or*

Telegram: @dental_k

www.konkur.in

without calcium chloride for root-end filling, J Endod 2006;32: 897.
58. Bortoluzzi EA, Niu LN, Palani CD, et al: *Cytotoxicity and osteogenic potential of silicate calcium cements as potential protective materials for pulpal revascularization*, Dent Mater 2015;31: 1510.
59. Bouillaguet S, Wataha J, Hanks CT, et al: *In vitro cytotoxcity and dentin permeability of HEMA (2-hydroxyethyl methacrylate)*, J Endod 1996;22: 244-248.
60. Brambilla E, García-Godoy F, Strohmenger L. *Principles of diagnosis and treatment of high-caries-risk subjects*, Dent Clin North Am 2000;44: 507.
61. Brännström M, Lind PO. *Pulpal response to early dental caries*, J Dent Res 1965;44: 1045.
62. Brizuela C, Ormeño A, Cabrera C, et al: *Direct pulp capping with calcium hydroxide, mineral trioxide aggregate, and biodentine in permanent young teeth with caries: a randomized clinical trial*, J Endod 2017;43: 1776.
63. Brodén J, Davidson T, Fransson H. *Cost-effectiveness of pulp capping and root canal treatment of young permanent teeth*, Acta Odontol Scand 2019;15: 1.
64. Burke FJ, Lucarotti PS. *Ten year survival of bridges placed in the general dental services in England and Wales*, J Dent. 2012;40: 886.
65. Byers MR, Taylor PE, Khayat BG, et al: *Effects of injury and inflammation on pulpal and periapical nerves*, J Endod 1990;16: 78.
66. Calişkan MK. *Pulpotomy of carious vital teeth with periapical involvement*, Int Endod J 1995;28: 172.
67. Camargo SE, Camargo CH, Hiller KA, et al: *Cytotoxicity and genotoxicity of pulp capping materials in two cell*

Telegram: @dental_k

www.konkur.in

lines, Int Endod J 2009;42: 227.
68. Camilleri J. *Characterization of hydration products of mineral trioxide aggregate*, Int Endod J 2008;41: 408.
69. Camilleri J. *Color stability of white mineral trioxide aggregate in contact with hypochlorite solution*, J Endod 2014;40: 436.
70. Camilleri J, Laurent P, About I. *Hydration of Biodentine, Theracal LC, and a prototype tricalcium silicate-based dentin replacement material after pulp capping in entire tooth cultures*, J Endod 2014;40: 1846.
71. Camilleri J, Montesin FE, Brady K, et al: *The constitution of mineral trioxide aggregate*, Dent Mater 2005;21: 297.
72. Camilleri J, Sorrentino F, Damidot D. *Investigation of the hydration and bioactivity of radiopacified tricalcium silicate cement, Biodentine and MTA Angelus*, Dent Mater 2013;29: 580.
73. Camp J. *Pediatric endodontic treatment*, Cohen S Burns RC Pathways of the pulp ed 7 1998; Mosby St. Louis.
74. Camp JH, Fuks AB. *Pediatric endodontics: endodontic treatment for the primary and young permanent dentition*, Cohen S Hargreaves K Pathways of the pulp ed 9 2006; Mosby/Elsevier St. Louis.
75. Cao Y, Bogen G, Lim J, et al: *Bioceramic materials and the changing concepts in vital pulp therapy*, J Calif Dent Assoc 2016;44: 278.
76. Caplan DJ, Cai J, Yin G, et al: *Root canal filled versus non-root canal filled teeth: a retrospective comparison of survival times*, J Public Health Dent 2005;65: 90.
77. Caplan DJ, Kolker J, Rivera EM, et al: *Relationship between number of proximal contacts and survival of root treated teeth*, Int Endod J 2002;35: 193.
78. Caplan DJ, Weintraub JA. *Factors related to loss of root*

Telegram: @dental_k

www.konkur.in

canal filled teeth, J Public Health Dent 1997;57: 31.
79. Cardoso MV, De Almeida Neves A, et al: *Current aspects on bonding effectiveness and stability in adhesive dentistry*, Aust Dent J Suppl 1, 2011;56: 31.
80. Carlisle EM. *Silicon: a possible factor in bone calcification*, Science 1970;176: 279.
81. Cavalcanti BN, Rode Sde M, Franca CM, et al: *Pulp capping materials exert an effect of the secretion of IL-1b and IL-8 by migrating human neutrophils*, Braz Oral Res 2011;25: 13.
82. Chandler NP, Ng BP, Monteith BD. *Radiographic recognition and distribution of approximal carious lesions in New Zealand undergraduate dental students*, N Z Dent J 2005;101: 106.
83. Chandler NP, Pitt Ford TR, Monteith BD. *Coronal pulp size in molars: a study of bitewing radiographs*, Int Endod J 2003;36: 757.
84. Chandler NP, Pitt Ford TR, Monteith BD. *Pulp size in molars: underestimation on radiographs*, J Oral Rehabil 2004;31: 764.
85. Charadram N, Austin C, Trimby P, et al: *Structural analysis of reactionary dentin formed in response to polymicrobial invasion*, J Struct Biol 2013;181(3): 207-222.
86. Charadram N, Farahani RM, Harty D, et al: *Regulation of reactionary dentin formation by odontoblasts in response to polymicrobial invasion of dentin matrix*, Bone 2012;50: 265.
87. Cho SY, Seo DG, Lee SJ, et al: *Prognostic factors for clinical outcomes according to time after direct pulp capping*, J Endod 2013;39: 327.
88. Christensen GJ. *Pulp capping*, J Am Dent Assoc

Telegram: @dental_k

www.konkur.in

1998;129: 1297.
89. Chueh LH, Chiang CP. *Histology of irreversible pulpitis premolars treated with mineral trioxide aggregate pulpotomy*, Oper Dent 2010;35: 370.
90. Chueh LH, Huang GT. *Immature teeth with periradicular periodontitis or abscess undergoing apexogenesis: a paradigm shift*, J Endod 2006;32: 1205.
91. Ciasca M, Aminoshariae A, Jin G, et al: *A comparison of the cytotoxicity and proinflammatory cytokine production of EndoSequence root repair material and ProRoot mineral trioxide aggregate in human osteoblast cell culture using reverse-transcriptase polymerase chain reaction*, J Endod 2012;38: 486.
92. Cohenca N, Paranjpe A, Berg J. *Vital pulp therapy*, Dent Clin North Am 2013;57: 59.
93. Convery Y LP. *Conserving the immature first permanent molar*, J Ir Dent Assoc 1968;14: 76.
94. Costa CAS, Hebling J, Hanks CT. *Current status of pulp capping with dentin adhesive systems: a review*, Dent Mater 2000;16: 188.
95. Couve E, Osorio R, Schmachtenberg O. *The amazing odontoblast: activity, autophagy, and aging*, J Dent Res 2013;92: 765.
96. Cox CF, Hafez AA, Akimoto N, et al: *Biocompatibility of primer, adhesive and resin composite systems on non-exposed and exposed pulps of non-human primate teeth*, Am J Dent 1998;11: S55.
97. Cox CF, Keall CL, Keall HJ, et al: *Biocompatibility of surface-sealed dental materials against exposed pulps*, J Prosthet Dent 1987;57: 1.
98. Cox CF, Sübay RK, Ostro E, et al: *Tunnel defects in dentinal bridges: their formation following direct pulp*

Telegram: @dental_k

www.konkur.in

capping, Oper Dent 1996;21: 4.
99. Cox CF, Suzuki S. *Re-evaluating pulp protection: calcium hydroxide liners vs cohesive hybridization*, J Am Dent Assoc 1994;125: 823.
100. Crane AB. A practicable root canal technique 1920; Lea & Febiger Philadelphia.
101. Cvek M. *A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated root fractures*, J Endod 1978;4: 232.
102. Cvek M, Lundberg M. *Histological appearance of pulps after exposure by a crown fracture, partial pulpotomy, and clinical diagnosis of healing*, J Endod 1983;9: 8.
103. de Blanco LP. *Treatment of crown fractures with pulp exposure*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82: 564.
104. Dakin HD. *On the use of certain antiseptic substances in treatment of infected wounds*, Br Med J 1915;2: 318.
105. Damas BA, Wheater MA, Bringas JS, et al: *Cytotoxicity comparison of mineral trioxide aggregates and EndoSequence bioceramic root repair materials*, J Endod 2011;37: 372.
106. Dammaschke T. *The formation of reparative dentine and Höhl cells in the dental pulp*, ENDO (Lond Engl) 2010;4: 255.
107. Dammaschke T. *Die indirekte Űberkappung der Pulpa*, Quintessenz 2016;67: 1309.
108. Dammaschke T, Camp JH, Bogen G. *MTA in vital pulp therapy*, Torabinejad M Mineral trioxide aggregate – properties and clinical applications 2014; Wiley Blackwell Publishing Ames.
109. Dammaschke T, Gerth HUV, Züchner H, et al: *Chemical and physical surface and bulk material characterization of*

Telegram: @dental_k

www.konkur.in

white ProRoot MTA and two Portland cements, Dent Mater 2005;21: 731.

110. Dammaschke T, Leidinger J, Schäfer E. *Long-term evaluation of direct pulp capping: treatment outcomes over an average period of 6.1 years*, Clin Oral Investig 2010;14: 559.
111. Dammaschke T, Nowicka A, Lipski M, et al: *Histological evaluation of hard tissue formation after direct pulp capping with a fast-setting mineral trioxide aggregate (RetroMTA) in humans*, Clin Oral Investig 2019; doi:1007/s00784-019-02876-2. [Epub ahead of print].
112. Dammaschke T, Stratmann U, Fischer RJ, et al: *A histological investigation of direct pulp capping in rodents with dentin adhesives and calcium hydroxide*, Quintessence Int 2010;41: e62.
113. Dammaschke T, Stratmann U, Fischer RJ, et al: *Proliferation of rat molar pulp cells after direct pulp capping with dentine adhesive and calcium hydroxide*, Clin Oral Investig 2011;15: 577.
114. Dammaschke T, Stratmann U, Wolff P, et al: *Direct pulp capping with mineral trioxide aggregate: an immunohistologic comparison with calcium hydroxide in rodents*, J Endod 2010;36: 814.
115. Dammaschke T, Wolff P, Saqheri D, et al: *Mineral trioxide aggregate for direct pulp capping: an histologic comparison with calcium hydroxide in rat molars*, Quintessence Int 2010;41: 20.
116. Darvell BW, Wu RC. *"MTA"—an hydraulic silicate cement: review update and setting reaction*, Dent Mater 2011;27: 407.
117. D'Antò V, Di Caprio MP, Ametrano G, et al: *Effect of mineral trioxide aggregate on mesenchymal stem cells*, J

Telegram: @dental_k

www.konkur.in

Endod 2010;36: 1839.
118. De Backer H, Van Maele G, Decock V, et al: *Long-term survival of complete crowns, fixed dental prostheses, and cantilever prostheses with post and cores on root-canal treated teeth*, Int J Prosthodont 2007;20: 229.
119. De Mendonça AA, De Oliveira CF, Hebling J, et al: *Influence of thicknesses of smear layer on the transdentinal cytotoxicity and bond strength of a resin-modified glass-ionomer cement*, Braz Dent J 2012;23: 379.
120. Déjou J, Raskin A, Colombani J, et al: *Physical, chemical and mechanical behavior of a new material for direct posterior fillings*, Eur Cell Mater Suppl 4, 2005;10: 22.
121. Demir T, Cehreli ZC. *Clinical and radiographic evaluation of adhesive pulp capping in primary molars following hemostasis with 1.25% sodium hypochlorite: 2-year results*, Am J Dent 2007;20: 182.
122. Dennison JB, Hamilton JC. *Treatment decisions and conservation of tooth structure*, Dent Clin North Am 2005;49: 825.
123. Dettwiler CA, Walter M, Zaugg LK, et al: *In vitro assessment of the tooth staining potential of endodontic materials in a bovine tooth model*, Dent Traumatol 2016;32: 480.
124. Do Nascimento ABL, Fontana UF, Teixeira HM, et al: *Biocompatibility of a resin-modified glass-ionomer cement applied as pulp capping in human teeth*, Am J Dent 2000;13: 28.
125. Domine L, Holz J. *[The aging of the human pulp-dentin organ]*, Schweiz Monatsschr Zahnmed 1991;101: 725.
126. Doyle TL, Casas MJ, Kenny DJ, et al: *Mineral trioxide aggregate produces superior outcomes in vital primary molar pulpotomy*, Pediatr Dent 2010;32: 41.

Telegram: @dental_k

www.konkur.in

127. Dreger LA, Felippe WT, Reyes-Carmona JF, et al: *Mineral trioxide aggregate and Portland cement promote biomineralization in vivo*, J Endod 2012;38: 324.
128. Duda S, Dammaschke T. *Maßnahmen zur Vitalerhaltung der Pulpa. Gibt es Alternativen zum Kalziumhydroxid bei der direkten Űberkappung?*, Quintessenz 2008;59: 1327.
129. Duncan HF, Galler KM, Tomson PL, et al: *European Society of Endodontology position statement: Management of deep caries and the exposed pulp*, Int Endod J 2019; doi:10.1111/iej.13080.
130. Eghbal MJ, Asgary S, Baglue RA, et al: *MTA pulpotomy of human permanent molars with irreversible pulpitis*, Aust Endod J 2009;35: 4.
131. Eid AA, Komabayashi T, Watanabe E, et al: *Characterization of the mineral trioxide aggregate-resin modified glass ionomer cement interface in different setting conditions*, J Endod 2012;38: 1126.
132. El- Meligy OA, Avery DR. *Comparison of mineral trioxide aggregate and calcium hydroxide as pulpotomy agents in young permanent teeth (apexogenesis)*, Pediatr Dent 2006;28: 399.
133. Elias RV, Demarco FF, Tarquinio SB, et al: *Pulp responses to the application of a self-etching adhesive in human pulps after controlling bleeding with sodium hypochlorite*, Quintessence Int 2007;38: 67.
134. Eskandarizadeh A, Shahpasandzadeh MH, Shahpasandzadeh M, et al: *A comparative study on dental pulp response to calcium hydroxide, white and grey mineral trioxide aggregate as pulp capping agents*, J Conserv Dent 2011;14: 351.
135. European Society of Endodontology. *Quality guidelines for endodontic treatment: consensus report of the*

Telegram: @dental_k

www.konkur.in

European Society of Endodontology, Int Endod J 2006;39: 921.

136. Farges JC, Alliot-Licht B, Renard E, et al: *Dental pulp defence and repair mechanisms in dental caries*, Mediators Inflamm 2015;2015: 230251- doi:10.1155/2015/230251.
137. Farges JC, Carrouel F, Keller JF, et al: *Cytokine production by human odontoblast-like cells upon Toll-like receptor-2 engagement*, Immunobiology 2011;216: 513.
138. Farsi N, Alamoudi N, Balto K, et al: *Clinical assessment of mineral trioxide aggregate (MTA) as direct pulp capping in young permanent teeth*, Pediatr Dent 2006;31: 72.
139. Fisher FJ, McCabe JF. *Calcium hydroxide base materials: an investigation into the relationship between chemical structure and antibacterial properties*, Br Dent J 1978;144: 341.
140. Fong CD, Davis MJ. *Partial pulpotomy for immature permanent teeth, its present and future*, Pediatr Dent 2002;24: 29.
141. Ford TR, Torabinejad M, Abedi HR, et al: *Using mineral trioxide aggregate as a pulp-capping material*, J Am Dent Assoc 1996;127: 1491.
142. Forss H, Widström E. *The post-amalgam era: a selection of materials and their longevity in primary and young permanent dentitions*, Int J Paediatr Dent 2003;13: 158.
143. Fridland M, Rosado R. *MTA solubility: a long term study*, J Endod 2005;31: 376.
144. Fuks AB. *Pulp therapy for the primary and young permanent dentitions*, Dent Clin North Am 2000;44: 571.
145. Fuks AB, Cosack A, Klein H, et al: *Partial pulpotomy as a treatment alternative for exposed pulps in crown-fractured permanent incisors*, Endod Dent Traumatol

Telegram: @dental_k

www.konkur.in

1987;3: 100.
146. Fuks AB, Gavra S, Chosack A. *Long-term follow up of traumatized incisors treated by partial pulpotomy*, Pediatr Dent 1993;15: 334.
147. Fuks AB, Heling I. *Pulp therapy for the young permanent dentition*, Pinkham JR Casamassimo PS Fields HW Pediatric dentistry: infancy through adolescence ed 24 2005; Saunders/Elsevier St. Louis.
148. Fulling HJ, Andreasen JO. *Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing procedures*, Scand J Dent Res 1976;84: 286.
149. Fusayama T. A simple pain-free adhesive restorative system by minimal reduction and total etching 1993; Ishiyaku Euro America Publishing St. Louis.
150. Fusayama T, Kurosaki N. *Structure and removal of carious dentin*, Int Dent J 1972;22: 401.
151. Fuss Z, Lustig J, Katz A, et al: *An evaluation of endodontically treated vertical root fractured teeth: impact of operative procedures*, J Endod 2001;27: 46.
152. Galani M, Tewari S, Sangwan P, et al: *Comparative evaluation of postoperative pain and success rate after pulpotomy and root canal treatment in cariously exposed mature permanent molars: a randomized controlled trial*, J Endod 2017;43: 1953.
153. Gandolfi MG, Ciapetti G, Taddei P, et al: *Apatite formation on bioactive calcium-silicate cements for dentistry affects surface topography and human marrow stromal cells proliferation*, Dent Mater 2010;26: 974.
154. Gandolfi MG, Siboni F, Prati C. *Chemical-physical properties of TheraCal, a novel light-curable MTA-like material for pulp capping*, Int Endod J 2012;45: 571.

Telegram: @dental_k

www.konkur.in

155. Gandolfi MG, van Lunduyt K, Taddei P, et al: *Environmental scanning electron microscopy connected with energy dispersive X-ray analysis and Raman techniques to study ProRoot mineral trioxide aggregate and calcium silicate cements in wet conditions and in real time*, J Endod 2010;36: 851.
156. Goldberg M. *Reactionary and reparative dentin-like structures*, Goldberg M The dental pulp – biology, pathology, and regenerative therapies 2014; Springer-Verlag Berlin.
157. Goldberg M. *The pulp reaction beneath the carious lesion*, Goldberg M Understanding dental caries 2016; Springer-Verlag Berlin.
158. Goldberg M, Farges JC, Lacerda-Pinheiro S, et al: *Inflammatory and immunological aspects of dental pulp repair*, Pharmacol Res 2008;58: 137.
159. Goldberg M, Lasfargues JJ, Legrand JM. *Clinical testing of dental materials—histological considerations*, Dent J 1994;22: S25.
160. Gonçalves JL, Viapiana R, Miranda CE, et al: *Evaluation of physico-chemical properties of Portland cements and MTA*, Braz Oral Res 2010;24: 277.
161. Goodis HE, Kahn A, Simon S. *Aging and the pulp*, Hargreaves K Goodis HE Tay F Seltzer and Bender's dental pulp ed 2 2012; Quintessence Publishing Hanover Park.
162. Goracci G, Mori G. *Scanning electron microscopic evaluation of resin-dentin and calcium hydroxide dentin-interface with resin composite restorations*, Quintessence Int 1996;27: 129.
163. Gracia TB. *Accuracy of size estimations by dentists of simulated pulp exposures and cavity preparations*. MDS

Telegram: @dental_k

www.konkur.in

(endodontics) research report 2006; University of Otago Dunedin, New Zealand.

164. Graham L, Cooper PR, Cassidy N, et al: *The effect of calcium hydroxide on solubilisation of bioactive dentine matrix components*, Biomaterials 2006;27: 2865.

165. Granath LE, Hagman G. *Experimental pulpotomy in human bicuspids with reference to cutting technique*, Acta Odontol Scand 1971;29: 155.

166. Grech L, Mallia B, Camilleri J. *Characterization of set intermediate restorative material, Biodentine, Bioaggregate and a prototype calcium silicate cement for use as root-end filling materials*, Int Endod J 2012;46: 632.

167. Grötz KA, Duschner H, Reichert TE, et al: *Histotomography of the odontoblast processes at the dentine-enamel junction of permanent healthy human teeth in the confocal laser scanning microscope*, Clin Oral Investig 1998;2: 21.

168. Gudkina J, Mindere A, Locane G, et al: *Review of the success of pulp exposure treatment of cariously and traumatically exposed pulps in immature permanent incisors and molars*, Stomatologija 2012;14: 71.

169. Gutmann JL, Heaton JF. *Management of the open (immature) apex. Part 1. Vital teeth*, Int Endod J 1981;14: 166.

170. Guven EP, Yalvac ME, Sahin F, et al: *Effect of calcium hydroxide–containing cement, mineral trioxide aggregate, and enamel matrix derivative on proliferation and differentiation of human tooth germ stem cells*, J Endod 2011;37: 650.

171. Guven G, Cehreli ZC, Ural A, et al: *Effect of mineral trioxide aggregate cements on transforming growth factor*

Telegram: @dental_k

www.konkur.in

beta-1 and bone morphogenetic protein production by human fibroblasts in vitro, J Endod 2007;33: 447.

172. Gwinnett J, Tay FR, Pang KM, et al: *Quantitative contribution of collagen network in dentin hybridization*, Am J Dent 1996;9: 140.
173. Hafez AA, Cox CF, Tarim B, et al: *An in vivo evaluation of hemorrhage control using sodium hypochlorite and direct capping with a one- or two-component adhesive system in exposed nonhuman primate pulps*, Quintessence Int 2002;33: 261.
174. Haghgoo R, Abbasi F. *A histopathological comparison of pulpotomy with sodium hypochlorite and formocresol*, Iran Endod J 2012;7: 60.
175. Hahn CL, Liewehr FR. *Innate immune responses of the dental pulp to caries*, J Endod 2007;33: 643.
176. Hahn CL, Liewehr FR. *Relationships between caries bacteria, host responses, and clinical signs and symptoms of pulpitis*, J Endod 2007;33: 213.
177. Hahn CL, Liewehr FR. *Update on the adaptive immune responses of the dental pulp*, J Endod 2007;33: 773.
178. Ham KA, Witherspoon DE, Gutmann JL, et al: *Preliminary evaluation of BMP-2 expression and histological characteristics during apexification with calcium hydroxide and mineral trioxide aggregate*, J Endod 2005;31: 275.
179. Han L, Okiji T. *Uptake of calcium and silicon released from calcium silicate-based endodontic materials into root canal dentine*, Int Endod J 2011;44: 1081.
180. Hanks CT, Strawn SE, Wataha JC, et al: *Cytotoxic effects of resin components on cultured mammalian fibroblasts*, J Dent Res 1991;70: 1450.
181. Harms CS, Schäfer E, Dammaschke T. *Clinical*

Telegram: @dental_k

www.konkur.in

evaluation of direct pulp capping using a calcium silicate cement - treatment outcomes over an average period of 2.3 years, Clin Oral Investig 2018; doi:10.1007/s00784-018-2767-5.

182. Hashem D, Mannocci F, Patel S, et al: *Clinical and radiographic assessment of the efficacy of calcium silicate indirect pulp capping: a randomized controlled clinical trial*, J Dent Res 2015;94: 562.
183. Hebling J, Giro EMA, DeSouza Costa CA. *Biocompatibility of an adhesive system applied to exposed human dental pulp*, J Endod 1999;25: 676.
184. Hebling J, Lessa FC, Nogueira I, et al: *Cytotoxicity of resin-based light-cured liners*, Am J Dent 2009;22: 137.
185. Hegde S, Sowmya B, Mathew S, et al: *Clinical evaluation of mineral trioxide aggregate and biodentine as direct pulp capping agents in carious teeth*, J Conserv Dent 2017;20: 91.
186. Helfer AR, Melnick S, Schilder H. *Determination of the moisture content of vital and pulpless teeth*, Oral Surg Oral Med Oral Pathol 1972;34: 661.
187. Henderson B, Wilson M. *Cytokine induction by bacteria: beyond lipopolysaccharide*, Cytokine 1996;8: 269.
188. Henderson HZ, Setcos JC. *The sealed composite resin restoration*, ASDC J Dent Child 1985;52: 300.
189. Hermann B. *Ein weiterer Beitrag zur Frage der Pulpenbehandlung*, Zahnärztl Rundsch 1928;37: 1327.
190. Hessle CC, Andersson B, Wold AE. *Gram-positive and gram-negative bacteria elicit different patterns of pro-inflammatory cytokines in human monocytes*, Cytokine 2005;30: 311.
191. Higashi T, Higashi T, Okamoto H. *Characteristics and effects of calcified degenerative zones on the formation of*

Telegram: @dental_k

www.konkur.in

hard tissue barriers in amputated canine dental pulp, J Endod 1996;22: 168.

192. Hilton TJ. *Keys to clinical success with pulp capping: a review of the literature*, Oper Dent 2008;34: 615.
193. Hilton TJ, Ferracane JL, Mancl L, Northwest Practice-based Research Collaborative in Evidence-based Dentistry (NWP). *Comparison of CaOH with MTA for direct pulp capping: a PBRN randomized clinical trial*, J Dent Res 2013;92: 16S.
194. Hirota K. *A study on the partial pulp removal (pulpotomy) using four different tissue solvents*, J Jpn Stomatol Soc 1959;26: 1588.
195. Holan G, Eidelman E, Fuks A. *Long-term evaluation of pulpotomy in primary molars using mineral trioxide aggregate or formocresol*, Pediatr Dent 2005;27: 129.
196. Hørsted P, Søndergaard B, Thylstrup A, et al: *A retrospective study of direct pulp capping with calcium hydroxide compounds*, Endod Dent Traumatol 1985;1: 29.
197. Hörsted-Bindslev P, Vilkinis V, Sidlauskas A. *Direct capping of human pulps with a dentin bonding system or with calcium hydroxide cement*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96: 591.
198. Hørsted-Bindslev P, Bergenholtz G. *Vital pulp therapies*, Bergenholtz G Hørsted-Bindslev P Erik-Reit C Textbook of endodontology 2003; Blackwell Munksgaard Publishing Oxford.
199. Hosoda H, Fusayama T. *A tooth substance saving restorative technique*, Int Dent J 1984;34: 1.
200. Inokoshi S, Iwaku M, Fusayama T. *Pulpal response to a new adhesive resin material*, J Dent Res 1982;61: 1014.
201. Ishizaka R, Hayashi Y, Iohara K, et al: *Stimulation of angiogenesis, neurogenesis and regeneration by side*

Telegram: @dental_k

www.konkur.in

population cells from dental pulp, Biomaterials 2013;34: 1888.

202. Issa Y, Watts DC, Brunton PA, et al: *Resin composite monomers alter MTT and LDH activity of human gingival fibroblasts in vitro*, Dent Mater 2004;20: 12.
203. Izumi T, Kobayashi I, Okamura K, et al: *Immunohistochemical study on the immunocompetent cells of the pulp in human non-carious and carious teeth*, Arch Oral Biol 1995;40: 609.
204. Jameson MW, Hood JAA, Tidmarsh BG. *The effects of dehydration and rehydration on some mechanical properties of human dentine*, J Biomech 1993;26: 1055.
205. Jamieson WJ, Vargas K. *Recall rates and caries experience of patients undergoing general anesthesia for dental treatment*, Pediatr Dent 2007;29: 253.
206. Jang Y, Song M, Yoo IS, et al: *A randomized controlled study of the use of ProRoot mineral trioxide aggregate and endocem as direct pulp capping materials: 3-months versus 1-year outcome*, J Endod 2015;41: 1201.
207. Jontell M, Hanks CT, Bratel J, et al: *Effects of unpolymerized resin components on the function of accessory cells derived from the rat incisor pulp*, J Dent Res 1995;74: 1162.
208. Jordan RE, Suzuki M. *Conservative treatment of deep carious lesions*, J Can Dent Assoc 1971;37: 337.
209. Jung S, Mielert J, Kleinheinz J, et al: *Human oral cells' response to different endodontic restorative materials: an in vitro study*, Head Face Med 2014;10: 55-doi:10.1186/s13005-014-0055-4.
210. Kakehashi S, Stanley HR, Fitzgerald RT. *The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats*, Oral Surg Oral Med Oral

Telegram: @dental_k

www.konkur.in

Pathol 1965;20: 340.
211. Kang CM, Sun Y, Song JS, et al: *A randomized controlled trial of various MTA materials for partial pulpotomy in permanent teeth*, J Dent 2017;60: 8.
212. Kangarlou A, Sofiabadi S, Asgary S, et al: *Assessment of antifungal activity of ProRoot mineral trioxide aggregate and mineral trioxide aggregate–Angelus*, Dent Res J (Isfahan) 2012;9: 256.
213. Karibe H, Ohide Y, Kohno H, et al: *Study on thermal pulp testing of immature permanent teeth*, Shigaku 1989;77: 1006.
214. Katge FA, Patil DP. *Comparative analysis of 2 calcium silicate-based cements (biodentine and mineral trioxide aggregate) as direct pulp-capping agent in young permanent molars. A split mouth study*, J Endod 2017;43: 507.
215. Kato S, Fusayama T. *Recalcification of artificially decalcified dentin in vivo*, J Dent Res 1970;49: 1060.
216. Kaup M, Dammann CH, Schäfer E, et al: *Shear bond strength of Biodentine, ProRoot MTA, glass ionomer cement and composite resin on human dentine ex vivo*, Head Face Med 2015;11: 14.
217. Kaup M, Schäfer E, Dammaschke T. *An in vitro study of different material properties of Biodentine compared to ProRoot MTA*, Head Face Med 2015;11: 16.
218. Kidd EA, Ricketts DN, Beighton D. *Criteria for caries removal at the enamel-dentine junction: a clinical and microbiological study*, Br Dent J 1996;180: 287.
219. Kim RJ, Kim MO, Lee KS, et al: *An in vitro evaluation of the antibacterial properties of three mineral trioxide aggregate (MTA) against five oral bacteria*, Arch Oral Biol 2015;60: 1497.

Telegram: @dental_k

www.konkur.in

220. Kim S, Heyeraas KJ, Haug SR. *Structure and function of the dentin-pulp complex*, Ingle JI Bakand LK Baumgartner JC Ingle's endodontics ed 6 2008; Decker Hamilton, Ontario.
221. Kim YJ, Chandler NP. *Determination of working length for teeth with wide or immature apices: a review*, Int Endod J 2013;46: 483.
222. Koh ET, Pitt Ford TR, Torabinejad M, et al: *Mineral trioxide aggregate stimulates cytokine production in human osteoblasts*, J Bone Min Res 1995;10S: S406.
223. Koh ET, Torabinejad M, Pitt Ford TR, et al: *Mineral trioxide aggregate stimulates a biological response in human osteoblasts*, J Biomed Mater Res 1997;37: 432.
224. Krastl G, Allgayer N, Lenherr P, et al: *Tooth discoloration induced by endodontic materials: a literature review*, Dent Traumatol 2013;29: 2.
225. Krastl G, Weiger R. *Vital pulp therapy after trauma*, Endod Pract Today 2014;8: 293.
226. Kundzina R, Stangvaltaite L, Eriksen HM, et al: *Capping carious exposures in adults: a randomized controlled trial investigating mineral trioxide aggregate versus calcium hydroxide*, Int Endod J 2017;50: 924.
227. Kuratate M, Yoshiba K, Shigetani Y, et al: *Immunohistochemical analysis of nestin, osteopontin, and proliferating cells in the reparative process of exposed dental pulp capped with mineral trioxide aggregate*, J Endod 2008;34: 970.
228. Kuttler Y. *Classification of dentin into primary, secondary and tertiary*, Oral Surg Oral Med Oral Pathol 1959;12: 996.
229. Langeland K. *Management of the inflamed pulp associated with deep carious lesion*, J Endod 1981;7: 169.

Telegram: @dental_k

www.konkur.in

230. Lasfargues JJ, Machtou P. *Collège National des Enseignants en Odontologie Conservatrice et Endodontie (CNEOC). Dictionnaire Francophone des termes d'Odontologie Conservatrice*, Endodontie & Odontologie restauratrice 2ème édition 2010; Espace id.
231. Laurent P, Camps J, About I. *Biodentine™ induces TGF-b 1 release from human pulp cells and early dental pulp mineralization*, Int Endod J 2012;45: 439.
232. Laurent P, Camps J, De Méo M, et al: *Induction of specific cell responses to a Ca(3)SiO(5)–based posterior restorative material*, Dent Mater J 2008;24: 1486.
233. Lee YH, Kim GE, Cho HJ, et al: *Aging of in vitro pulp illustrates change of inflammation and dentinogenesis*, J Endod 2013;39: 340.
234. Lenherr P, Allgayer N, Weiger R, et al: *Tooth discoloration induced by endodontic materials: a laboratory study*, Int Endod J 2012;45: 942.
235. Lertchirakarn V, Palamara JE, Messer HH. *Patterns of vertical root fracture: factors affecting stress distribution in the root canal*, J Endod 2003;29: 523.
236. Lesot H, Bègue-Kirn C, Kubler MD, et al: *Experimental induction of odontoblast differentiation and stimulation during reparative processes*, Cells Mater 1993;3: 201.
237. Lessa FC, Aranha AM, Hebling J, et al: *Cytotoxic effects of white MTA and MTA-Bio cements on odontoblast-like cells (MDPC-23)*, Braz Dent J 2010;21: 24.
238. Leye Benoist F, Gaye Ndiaye F, Kane AW, et al: *Evaluation of mineral trioxide aggregate (MTA) versus calcium hydroxide cement (Dycal) in the formation of a dentine bridge: a randomised controlled trial*, Int Dent J 2012;62: 33.
239. Li Z, Cao L, Fan M, et al: *Direct pulp capping with*

Telegram: @dental_k

www.konkur.in

calcium hydroxide or mineral trioxide aggregate: a meta-analysis, J Endod 2015;41: 1412.

240. Lin LM, Rosenberg PA. *Repair and regeneration in endodontics*, Int Endod J 2011;44: 889.
241. Linn J, Messer HH. *Effect of restorative procedures on the strength of endodontically treated molars*, J Endod 1994;20: 479.
242. Linsuwanont P, Wimonsutthikul K, Pothimoke U, et al: *Treatment outcomes of mineral trioxide aggregate pulpotomy in vital permanent teeth with carious pulp exposure: the retrospective study*, J Endod 2017;43: 225.
243. Linu S, Lekshmi MS, Varunkumar VS, et al: *Treatment outcome following direct pulp capping using bioceramic materials in mature permanent teeth with carious exposure: a pilot retrospective study*, J Endod 2017;43: 1635.
244. Lipski M, Nowicka A, Kot K, et al: *Factors affecting the outcomes of direct pulp capping using Biodentine*, Clin Oral Investig 2018;22: 2021.
245. Mahmoud SH, Mel A, Zaher AR, et al: *Influence of selective immunosuppressive drugs on the healing of exposed dogs' dental pulp capped with mineral trioxide aggregate*, J Endod 2010;36: 95.
246. Mantellini MG, Botero TM, Yaman P, et al: *Adhesive resin induces apoptosis and cell-cycle arrest of pulp cells*, J Dent Res 2003;82: 592.
247. Marchi JJ, De Araujo FB, Fröner AM, et al: *Indirect pulp capping in the primary dentition: a 4 year follow-up study*, J Clin Pediatr Dent 2006;31: 68.
248. Marques MS, Wesselink PR, Shemesh H. *Outcome of direct pulp capping with mineral trioxide aggregate*, J Endod 2015;41: 1026.

Telegram: @dental_k

www.konkur.in

249. Mass E, Zilberman U. *Long-term radiologic pulp evaluation after partial pulpotomy in young permanent molars*, Quintessence Int 2011;42: 547.
250. Mathieu S, Jeanneau C, Sheibat-Othman N, et al: *Usefulness of controlled release of growth factors in investigating the early events of dentin-pulp regeneration*, J Endod 2013;39: 228.
251. Matsumoto S, Hayashi M, Suzuki Y, et al: *Calcium ions released from mineral trioxide aggregate convert the differentiation pathway of C2C12 cells into osteoblast lineage*, J Endod 2013;39: 68.
252. Matsuo T, Nakanishi T, Shimizu H, et al: *A clinical study of direct pulp capping applied to carious-exposed pulps*, J Endod 1996;22: 551.
253. Matsuura T, Katsumata T, Matsuura T, et al: *Histopathological study of pulpal irritation of dental adhesive resin. Part 1. Panavia EX*, Nihon Hotetsu Shika Gakkai Zasshi 1987;31: 104.
254. Matysiak M, Dubois JP, Ducastelle T, et al: *Morphometric analysis of human pulp myelinated fibers during aging*, J Biol Buccale 1986;14: 69.
255. McComb D. *Caries-detector dyes: how accurate and useful are they?*, J Can Dent Assoc 2000;66: 195.
256. McDonald RE, Avery DR, Dean JA. *Treatment of deep caries, vital pulp exposure, and pulpless teeth*, Dean JA Avery DR McDonald RE McDonald and Avery's dentistry of the child and adolescent ed 9 2011; Mosby/Elsevier St. Louis.
257. Memarpour M, Mesbahi M, Shafiei F. *Three-and-a- half-year clinical evaluation of posterior composite resin in children*, J Dent Child 2010;77: 92.
258. Mente J, Geletneky B, Ohle M, et al: *Mineral trioxide*

Telegram: @dental_k

www.konkur.in

aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcome, J Endod 2010;36: 806.

259. Mente J, Hage N, Pfefferie T, et al: *Mineral trioxide aggregate apical plugs in teeth with open apical foramina: a retrospective analysis of treatment outcome*, J Endod 2009;35: 1354.
260. Mente J, Hufnagel S, Leo M, et al: *Treatment of mineral trioxide aggregate or calcium hydroxide direct pulp capping: long-term results*, J Endod 2014;40: 1746.
261. Mente J, Leo M, Panagidis D, et al: *Treatment outcome of mineral trioxide aggregate in open apex teeth*, J Endod 2013;39: 20.
262. Mentink AG, Meeuwissen R, Käyser AF, et al: *Survival rate and failure characteristics of the all metal post and core restoration*, J Oral Rehabil 1993;20: 455.
263. Merdad K, Sonbul H, Bukhary S, et al: *Caries susceptibility of endodontically versus nonendodontically treated teeth*, J Endod 2011;37: 139.
264. Mertz-Fairhurst EJ, Call-Smith KM, Shuster GS, et al: *Clinical performance of sealed composite restorations placed over caries compared with sealed and unsealed amalgam restorations*, J Am Dent Assoc 1987;115: 689.
265. Mettes D. *Insufficient evidence to support or refute the need for 6-monthly dental check-ups: What is the optimal recall frequency between dental checks?*, Evid Based Dent 2005;6: 62.
266. Min KS, Park HJ, Lee SK, et al: *Effect of mineral trioxide aggregate on dentin bridge formation and expression of dentin sialoprotein and heme oxygenase-1 in human pulp*, J Endod 2008;34: 666.
267. Minamikawa H, Yamada M, Deyama Y, et al: *Effect of*

Telegram: @dental_k

www.konkur.in

N-acetylcysteine on rat dental pulp cells cultured on mineral trioxide aggregate, J Endod 2011;37: 637.

268. Miyashita H, Worthington HV, Qualtrough A, et al: *Pulp management for caries in adults: maintaining pulp vitality*, Cochrane Database Syst Rev 2007;18: CD004484.
269. Miyauchi H, Iwaku M, Fusayama T. *Physiological recalcification of carious dentin*, Bull Tokyo Med Dent Univ 1978;25: 169.
270. Mjör IA. *Pulp-dentin biology in restorative dentistry. Part 5: Clinical management and tissue changes associated with wear and trauma*, Quintessence Int 2001;32: 771.
271. Mjör IA. *Pulp-dentin biology in restorative dentistry. Part 7: The exposed pulp*, Quintessence Int 2002;33: 113.
272. Modena KC, Casas-Apayco LC, Atta MT, et al: *Cytotoxicity and biocompatibility of direct and indirect pulp capping materials*, J Appl Oral Sci 2009;17: 544.
273. Moghaddame-Jafari S, Mantellini MG, Botero TM, et al: *Effect of ProRoot MTA on pulp cell apoptosis and proliferation in vitro*, J Endod 2005;31: 387.
274. Mohammadi Z, Dummer PMH. *Properties and applications of calcium hydroxide in endodontics and dental traumatology*, Int Endod J 2011;44: 697.
275. Moretti AB, Sakai VT, Oliveira TM, et al: *The effectiveness of mineral trioxide aggregate, calcium hydroxide and formocresol for pulpotomies in primary teeth*, Int Endod J 2008;41: 547.
276. Morse DR. *Age-related changes of the dental pulp complex and their relationship to systemic aging*, Oral Surg Oral Med Oral Pathol 1991;72: 721.
277. Morse DR, Esposito JV, Schoor RS. *A radiographic study of aging changes of the dental pulp and dentin in*

Telegram: @dental_k

www.konkur.in

normal teeth, Quintessence Int 1993;24: 329.
278. Mozayeni MA, Milani AS, Marvasti LA, et al: *Cytotoxicity of calcium enriched mixture cement compared with mineral trioxide aggregate and intermediate restorative material*, Aust Dent J 2012;38: 70.
279. Możyńska J, Metlerski M, Lipski M, et al: *Tooth discoloration induced by different calcium silicate-based cements. A systematic review of in vitro studies*, J Endod 2017;43: 1593.
280. Mudie AS, Holland GR. *Local opioids in the inflamed dental pulp*, J Endod 2006;32: 319.
281. Murray PE, About I, Lumley PJ, et al: *Cavity remaining dentin thickness and pulpal activity*, Am J Dent 2002;15: 41.
282. Murray PE, Hafez AA, Smith AJ, et al: *Histomorphometric analysis of odontoblast-like cell numbers and dentine bridge secretory activity following pulp exposure*, Int Endod J 2003;36: 106.
283. Murray PE, Smith AJ. *Saving pulps: a biological basis—an overview*, Prim Dent Care 2002;9: 21.
284. Murray PE, Smith AJ, Windsor LJ, et al: *Remaining dentine thickness and human pulp responses*, Int Endod J 2003;36: 33.
285. Murray PE, Stanley HR, Matthews JB, et al: *Age-related odontometric changes of human teeth*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93: 474.
286. Nair PNR, Duncan HF, Pitt Ford TR, et al: *Histological, ultrastructural and quantitative investigations on the response of healthy human pulps to experimental pulp capping with mineral trioxide aggregate: a randomized controlled trial*, Int Endod J 2008;41: 128.
287. Nandini S, Ballal S, Kandaswamy D. *Influence of glass-*

Telegram: @dental_k

www.konkur.in

ionomer cement on the interface and setting reaction of mineral trioxide aggregate when used as a furcal repair material using laser Raman spectroscopic analysis, J Endod 2007;33: 167.

288. Neelakantan P, Grotra D, Subbarao CV, et al: *The shear bond strength of resin-based composite to white mineral trioxide aggregate*, J Am Dent Assoc 2012;143: e40.
289. Ng YL, Mann V, Gulabivala K. *A prospective study of the factors affecting outcomes of non-surgical root canal treatment. Part 2. Tooth survival*, Int Endod J 2011;44: 610.
290. Nikaido T, Nurrohman H, Takagaki T, et al: *Nanoleakage in hybrid layer and acid-base resistant zone at the adhesive/dentin interface*, Microsc Microanal 2015;21: 1271.
291. Nikaido T, Weerasinghe DD, Waidyasekera K, et al: *Assessment of the nanostructure of acid-base resistant zone by the application of all-in-one adhesive systems: super dentin formation*, Biomed Mater Eng 2009;19: 163.
292. Nikiforuk G. *Optimal recall intervals in child dental care*, J Can Dent Assoc 1997;63: 618.
293. Nissan R, Segal H, Pashley D, et al: *Ability of bacterial endotoxin to diffuse through human dentin*, J Endod 1995;21: 62.
294. Nosrat A, Seifi A, Asgary S. *Pulpotomy in caries-exposed immature permanent molars using calcium-enriched mixture cement or mineral trioxide aggregate: a randomized clinical trial*, Int J Paediatr Dent 2013;23: 56.
295. Nowicka A, Lipski M, Parafiniuk M, et al: *Response of human dental pulp capped with Biodentine and mineral trioxide aggregate*, J Endod 2013;39: 743.
296. Nowicka A, Parafiniuk M, Lipski M, et al: *Pulpo-dentin*

Telegram: @dental_k

www.konkur.in

complex response after direct capping with self-etch adhesive systems, Folia Histochem Cytobiol 2012;50: 565.

297. Nowicka A, Wilk G, Lipski M, et al: *Tomographic evaluation of reparative dentin formation after direct pulp capping with Ca(OH) 2, MTA, Biodentine, and dentin bonding system in human teeth*, J Endod 2015;41: 1234.
298. Okiji T, Yoshiba K. *Reparative dentinogenesis induced by mineral trioxide aggregate: a review from the biological and physicochemical points of view*, Int J Dent 2009;464: 280.
299. Paranjpe A, Smoot T, Zhang H, et al: *Direct contact with mineral trioxide aggregate activates and differentiates human dental pulp cells*, J Endod 2011;37: 1691.
300. Paranjpe A, Zhang H, Johnson JD. *Effects of mineral trioxide aggregate on human pulp cells after pulp-capping procedures*, J Endod 2010;36: 1042.
301. Parinyaprom N, Nirunsittirat A, Chuveera P, et al: *Outcomes of direct pulp capping by using either ProRoot Mineral Trioxide Aggregate or Biodentine in permanent teeth with carious pulp exposure in 6- to 18-year-old patients: a randomized controlled trial*, J Endod 2018;44: 341.
302. Parirokh M, Torabinejad M, Dummer PMH. *Mineral trioxide aggregate and other bioactive endodontic cements: an updated overview - part I: vital pulp therapy*, Int Endod J 2018;51: 177.
303. Park JW, Hong SH, Kim JH, et al: *X-ray diffraction analysis of white ProRoot MTA and Diadent BioAggregate*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109: 155.
304. Patel N, Best SM, Bonfield W, et al: *A comparative study on the in vivo behavior of hydroxyapatite and silicon*

Telegram: @dental_k

www.konkur.in

substituted hydroxyapatite granules, J Mater Sci Mater Med 2002;13: 1199.

305. Patel R, Cohenca N. *Maturogenesis of a cariously exposed immature permanent tooth using MTA for direct pulp capping: a case report*, Dent Traumatol 2006;22: 328.
306. Paterson RC, Watts A. *Further studies on the exposed germ-free dental pulp*, Int Endod J 1987;20: 112.
307. Pedano MS, Li X, Li S, et al: *Freshly mixed and setting calcium-silicate cements stimulate human dental pulp cells*, Dent Mater 2018;34: 797.
308. Pfaff P. Abhandlung von den Zähnen des menschlichen Körpers und deren Krankheiten 1756; Haude und Spener Berlin.
309. Poggio C, Arciola CR, Beltrami R, et al: *Cytocompatibility and antibacterial properties of capping materials*, Sci World J 2014;2014: 181945.
310. Poggio C, Ceci M, Dagna A, et al: *In vitro cytotoxicity evaluation of different pulp capping materials: a comparative study*, Arh Hig Rada Toksikol 2015;66: 181.
311. Primosch RE, Balsewich CM, Thomas CW. *Outcomes assessment an intervention strategy to improve parental compliance to follow-up evaluations after treatment of early childhood caries using general anesthesia in a medicaid population*, ASDC J Dent Child 2001;68: 102.
312. Pugach MK, Strother J, Darling CL, et al: *Dentin caries zones: mineral, structure, and properties*, J Dent Res 2009;88: 71.
313. Qudeimat MA, Barrieshi-Nusair KM, Owais AI. *Calcium hydroxide vs mineral trioxide aggregates for partial pulpotomy of permanent molars with deep caries*, Eur Arch Paediatr Dent 2007;8: 99.

Telegram: @dental_k

www.konkur.in

314. Randow K, Glantz PO. *On cantilever loading of vital and non-vital teeth: an experimental clinical study*, Acta Odontol Scand 1986;44: 271.
315. Ranly DM, Garcia-Godoy F. *Current and potential pulp therapies for primary and young permanent teeth*, Dent J 2000;28: 153.
316. Rebel HH. *Űber die Ausheilung der freigelegten Pulpa*, Dtsch Zahnheilkd 1922;55: 3.
317. Reyes-Carmona JF, Santos AR, Figueiredo CP, et al: *In vivo host interactions with mineral trioxide aggregate and calcium hydroxide: inflammatory molecular signaling assessment*, J Endod 2011;37: 1225.
318. Reyes-Carmona JF, Santos AS, Figueiredo CP, et al: *Host-mineral trioxide aggregate inflammatory molecular signaling and biomineralization ability*, J Endod 2010;36: 1347.
319. Ricucci D, Loghin S, Lin LM, et al: *Is hard tissue formation in the dental pulp after the death of the primary odontoblasts a regenerative or a reparative process?*, J Dent 2014;42: 1156.
320. Ricucci D, Loghin S, Niu LN, et al: *Changes in the radicular pulp-dentine complex in healthy intact teeth and in response to deep caries or restorations: a histological and histobacteriological study*, J Dent 2018;73: 76.
321. Ricucci D, Loghin S, Siqueira JF, et al: *Correlation between clinical and histologic pulp diagnoses*, J Endod 2014;40: 1932.
322. Ricucci D, Siqueira JF Jr. *Vital pulp therapy*, Ricucci D Siqueira JF Jr Endodontology – an integrated biological and clinical view 2013; Quintessence Publishing London.
323. Rodd HD, Boissonade FM. *Comparative immunohistochemical analysis of the peptidergic*

Telegram: @dental_k

www.konkur.in

innervation of human primary and permanent tooth pulp, Arch Oral Biol 2002;47: 375.

324. Rölling I, Hasselgren G, Tronstad L. *Morphologic and enzyme histochemical observations on the pulp of human primary molars 3 to 5 years after formocresol treatment*, Oral Surg Oral Med Oral Pathol 1976;42: 518.
325. Rosenfeld EF, James GA, Burch BS. *Vital pulp tissue response to sodium hypochlorite*, J Endod 1978;4: 140.
326. Roth KK, Müller M, Ahrens G. *Staining of carious dentin with Kariesdetektor*, Dtsch Zahnärztl Z 1989;44: 460.
327. Saito T, Toyooka H, Ito S, et al: *In vitro study of remineralization of dentin: effects of ions on mineral induction by decalcified dentin matrix*, Caries Res 2003;37: 445.
328. Salvi GE, Siegrist Guldener BE, Amstad T, et al: *Clinical evaluation of root filled teeth restored with or without post-and-core systems in a specialist practice setting*, Int Endod J 2007;40: 209.
329. Sarkar NK, Caicedo R, Ritwik P, et al: *Physiochemical basis of the biologic properties of mineral trioxide aggregate*, J Endod 2005;31: 97.
330. Sasafuchi Y, Otsuki M, Inokoshi S, et al: *The effects on pulp tissue of microleakage in resin composite restorations*, J Med Dent Sci 1999;46: 155.
331. Sato Y, Fusayama T. *Removal of dentin guided by Fuchsin staining*, J Dent Res 1976;55: 678.
332. Schäfer E, Hickel R, Geurtsen W, et al: *Offizielles Endodontologisches Lexikon - mit einem Anhang für Materialien und Instrumente - der Deutschen Gesellschaft für Zahnerhaltung*, Endodontie 2000;9: 129.
333. Schmidt A, Schäfer E, Dammaschke T. *Shear bond strength of lining materials to calcium silicate cements at*

Telegram: @dental_k

www.konkur.in

different time intervals, J Adhes Dent 2017;19: 129.
334. Schröder U. *Effect of calcium hydroxide–containing pulp capping agents on pulp cell migration, proliferation, and differentiation*, J Dent Res 1985;66: 1166.
335. Schröder U. *Pedodontic endodontics*, Koch G Poulsen S Pediatric dentistry: a clinical approach 2001; Munksgaard Copenhagen.
336. Schroeder HE. Orale Strukturbiologie. 3 1987; Thieme Verlag Aufl. Stuttgart.
337. Schuurs AH, Gruythuysen RJ, Wesselink PR. *Pulp capping with adhesive resin-based composite vs. calcium hydroxide: a review*, Endod Dent Traumatol 2000;16: 240.
338. Schwendicke F. *Contemporary concepts in carious tissue removal: a review*, J Esthet Restor Dent 2017;29: 403.
339. Schwendicke F, Stolpe M. *Direct pulp capping after a carious exposure versus root canal treatment: a cost-effectiveness analysis*, J Endod 2014;40: 1764.
340. Seo MS, Hwang KG, Lee J, et al: *The effect of mineral trioxide aggregate on odontogenic differentiation in dental pulp stem cells*, J Endod 2013;39: 242.
341. Setbon HM, Devaux J, Iserentant A, et al: *Influence of composition on setting kinetics of new injectable and/or fast setting tricalcium silicate cements*, Dent Mater 2014;30: 1291.
342. Shabahang S, Torabinejad M. *Treatment of teeth with open apices using mineral trioxide aggregate*, Pract Periodontics Aesthet Dent 2000;12: 315.
343. Shayegan A, Jurysta C, Atash R, et al: *Biodentine used as a pulp-capping agent in primary pig teeth*, Pediatr Dent 2012;34: 202.
344. Sheehy EC, Roberts GJ. *Use of calcium hydroxide for apical barrier formation and healing in non-vital*

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immature permanent teeth: a review, Br Dent J 1997;183: 241.

345. Shiba H, Nakanishi K, Rashid F, et al: *Proliferative ability and alkaline phosphatase activity with in vivo cellular aging in human pulp cells*, J Endod 2003;29: 9.
346. Shields ED, Altschuller B, Choi EY, et al: *Odontometric variation among American black, European, and Mongoloid populations*, J Craniofac Genet Dev Biol 1990;10: 7.
347. Shokouhinejad N, Nekoofar MH, Pirmoazen S, et al: *Evaluation and comparison of occurrence of tooth discoloration after the application of various calcium silicate-based cements: an ex vivo study*, J Endod 2016;42: 140.
348. Sigal MJ, Pitaru S, Aubin JE, et al: *A combined scanning electron microscopy and immunofluorescence study demonstrating that the odontoblast process extends to the dentinoenamel junction in human teeth*, Anat Rec 1984;210: 453.
349. Silva GA, Gava E, Lanza LD, et al: *Subclinical failures of direct pulp capping of human teeth by using a dentin bonding system*, J Endod 2013;39: 182.
350. Simon S, Smith AJ, Berdal A, et al: *The MAP kinase pathway is involved in odontoblast stimulation via p38 phosphorylation*, J Endod 2010;36: 256.
351. Simonsen RJ. *Conservation of tooth structure in restorative dentistry*, Quintessence Int 1985;16: 15.
352. Sloan AJ, Perry H, Matthews JB, Smith AJ. *Transforming growth factor-beta isoform expression in mature human healthy and carious molar teeth*, Histochem J 2000;32: 247.
353. Smith AJ. *Formation and repair of dentin in the adult*,

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www.konkur.in

Hargreaves KM Goodis HE Tay FR Seltzer and Bender's dental pulp ed 2 2012; Quintessence Publishing Chicago.

354. Smith AJ, Cassidy N, Perry H, et al: *Reactionary dentinogenesis*, Int J Dev Biol 1995;39: 273.
355. Song JS, Mante FK, Romanow WJ, et al: *Chemical analysis of powder and set forms of Portland cement, gray ProRoot MTA, white ProRoot MTA, and gray MTA-Angelus*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102: 809.
356. Souza RA, Gomes SC, Dantas Jda C, et al: *Importance of the diagnosis in the pulpotomy of immature permanent teeth*, Braz Dent J 2007;18: 244.
357. Staehle HJ, Pioch T. *Zur alkalisierenden Wirkung von kalziumhaltigen Präparaten*, Dtsch Zahnärztl Z 1988;43: 308.
358. Stanley HR. *Pulp capping—conserving the dental pulp: Can it be done? Is it worth it?*, Oral Surg Oral Med Oral Pathol 1989;68: 628.
359. Subramaniam P, Konde S, Prashanth P. *An in vitro evaluation of pH variations in calcium hydroxide liners*, J Indian Soc Pedod Prev Dent 2006;24: 144.
360. Sudo C. *A study on partial pulp removal (pulpotomy) using NaOCl (sodium hypochlorite)*, J Jpn Stomatol Soc 1959;26: 1012.
361. Taha NA, Abdeukhader SZ. *Outcome of full pulpotomy using Biodentine in adult patients with symptoms indicative of irreversible pulpitis*, Int Endod J 2018;51: 819.
362. Taha NA, Abdulkhader SZ. *Full pulpotomy with biodentine in symptomatic young permanent teeth with carious exposure*, J Endod 2018;44: 932.
363. Taha NA, Ahmad MB, Ghanim A. *Assessment of mineral*

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www.konkur.in

trioxide aggregate pulpotomy in mature permanent teeth with carious exposures, Int Endod J 2017;50: 117.

364. Taha NA, Khazali MA. *Partial pulpotomy in mature permanent teeth with clinical signs indicative of irreversible pulpitis: a randomized clinical trial*, J Endod 2017;43: 1417.
365. Takita T, Hayashi M, Takeichi O, et al: *Effect of mineral trioxide aggregate on proliferation of cultured human dental pulp cells*, Int Endod J 2006;39: 415.
366. Tang W, Wu Y, Smales RJ. *Identifying and reducing risks for potential fractures in endodontically treated teeth*, J Endod 2010;36: 609.
367. Tarim B, Hafez AA, Cox CF. *Pulpal response to a resin-modified glass-ionomer material on nonexposed and exposed monkey pulps*, Quintessence Int 1998;29: 535.
368. Tarim B, Hafez AA, Suzuki SH, et al: *Biocompatibility of compomer restorative systems on nonexposed dental pulps of primate teeth*, Oper Dent 1997;22: 149.
369. Tarim B, Hafez AA, Suzuki SH, et al: *Biocompatibility of Optibond and XR-Bond adhesive systems in nonhuman primate teeth*, Int J Periodontics Restorative Dent 1998;18: 86.
370. Tatsumi T. *Physiological remineralization of artificially decalcified monkey dentin under adhesive composite resin restoration*, Kokubyo Gakkai Zasshi 1989;56: 47.
371. Tatsumi T, Inokoshi S, Yamada T, et al: *Remineralization of etched dentin*, J Prosthet Dent 1992;67: 617.
372. Ten Cate AR. *Dentin-pulp complex*, Oral histology ed 4 1994; Mosby St. Louis.
373. Tinanoff N, Douglass JM. *Clinical decision making for caries management in children*, Pediatr Dent 2002;24: 386.

Telegram: @dental_k

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374. Tomson PL, Grover LM, Lumley PJ, et al: *Dissolution of bio-active dentine matrix components by mineral trioxide aggregate*, Dent J 2007;35: 636.
375. Tomson PL, Lumley PJ, Alexander MY, et al: *Hepatocyte growth factor is sequestered in dentine matrix and promotes regeneration-associated events in dental pulp cells*, Cytokine 2013;61: 622.
376. Tønder KJ. *Vascular reactions in the dental pulp during inflammation*, Acta Odontol Scand 1983;41: 247.
377. Torabinejad M, Higa RK, McKendry DJ, et al: *Dye leakage of four root end filling materials: effects of blood contamination*, J Endod 1994;20: 159.
378. Torabinejad M, Parirokh M. *Mineral trioxide aggregate: a comprehensive literature review - Part II: leakage and biocompatibility investigations*, J Endod 2010;36: 190.
379. Torabinejad M, Smith PW, Kettering JD, et al: *Comparative investigation of marginal adaptation of mineral trioxide aggregate and other commonly used root-end filling materials*, J Endod 1995;21: 295.
380. Torbjörner A, Karlsson S, Odman PA. *Survival rate and failure characteristics for two post designs*, J Prosthet Dent 1995;73: 439.
381. Tranasi M, Sberna MT, Zizzari V, et al: *Microarray evaluation of age-related changes in human dental pulp*, J Endod 2009;35: 1211.
382. Tronstad L, Mjör IA. *Capping of the inflamed pulp*, Oral Surg Oral Med Oral Pathol 1972;34: 477.
383. Tuna EB, Dinçol ME, Gençay K, et al: *Fracture resistance of immature teeth filled with Bioaggregate, mineral trioxide aggregate and calcium hydroxide*, Dent Traumatol 2011;27: 174.
384. Tüzüner T, Alacam A, Altunbas DA, et al: *Clinical and*

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www.konkur.in

radiographic outcomes of direct pulp capping therapy in primary molar teeth following haemostasis with various antiseptics: a randomised controlled trial, Eur J Paediatr Dent 2012;13: 289.

385. Tziafas D, Pantelidou O, Alvanou A, et al: *The dentinogenic effect of mineral trioxide aggregate (MTA) in short-term capping experiments*, Int Endod J 2002;35: 245.
386. Uesrichai N, Nirunsittirat A, Chuveera P, et al: *Partial pulpotomy with two bioactive cements in permanent teeth of 6- to 18-year-old patients with signs and symptoms indicative of irreversible pulpitis: a noninferiority randomized controlled trial*, Int Endod J 2019; doi:10.1111/iej.13071. [Epub ahead of print].
387. Van Meerbeek B, Peumans M, Poitevin A, et al: *Relationship between bond-strength tests and clinical outcomes*, Dent Mater 2010;26: e100.
388. Vargas KG, Packham B, Lowman D. *Preliminary evaluation of sodium hypochlorite for pulpotomies in primary molars*, Pediatr Dent 2006;28: 511.
389. Wang CH, Chueh LH, Chen SC, et al: *Impact of diabetes mellitus, hypertension, and coronary artery disease on tooth extraction after nonsurgical endodontic treatment*, J Endod 2011;37: 1.
390. Wang G, Wang C, Qin M. *Pulp prognosis following conservative pulp treatment in teeth with complicated crown fractures - a retrospective study*, Dent Traumatol 2017;33: 255.
391. Wang NJ, Aspelund GØ. *Preventive care and recall intervals: targeting of services in child dental care in Norway*, Community Dent Health 2010;27: 5.
392. Wang Y, Zheng QH, Zhou XD, et al: *Evaluation of the root and canal morphology of mandibular first permanent*

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www.konkur.in

molars in a western Chinese population by cone-beam computed tomography, J Endod 2010;36: 1786.

393. Wegner PK, Freitag S, Kern M. *Survival rate of endodontically treated teeth with posts after prosthetic restoration*, J Endod 2006;32: 928.
394. Weisleder R, Benitez CR. *Maturogenesis: Is it a new concept?*, J Endod 2003;29: 776.
395. Willershausen B, Willershausen I, Ross A, et al: *Retrospective study on direct pulp capping with calcium hydroxide*, Quintessence Int 2011;42: 165.
396. Winters J, Cameron AC, Widmer RP. *Pulp therapy for primary and immature permanent teeth*, Cameron AC Widmer RP Handbook of pediatric dentistry ed 3 2008; Mosby/Elsevier Philadelphia.
397. Witherspoon DE. *Vital pulp therapy with new materials: new directions and treatment perspectives—permanent teeth*, J Endod 2008;34: S25.
398. Witherspoon DE, Regan JD, Small JC. *MTA pulpotomies: a long-term case series outcomes assessment of complicated crown fractures*, Tex Dent J 2017;134: 166.
399. Witherspoon DE, Small JC, Harris GZ. *Mineral trioxide aggregate pulpotomies: a case series outcomes assessment*, J Am Dent Assoc 2006;137: 610.
400. Witherspoon DE, Small JC, Regan JD, et al: *Retrospective analysis of open apex teeth obturated with mineral trioxide aggregate*, J Endod 2008;34: 1171.
401. Wolters WJ, Duncan HF, Tomson PL, et al: *Minimally invasive endodontics: a new diagnostic system for assessing pulpitis and subsequent treatment needs*, Int Endod J 2017;50: 825.
402. Yalcin M, Arsaln U, Dundar A. *Evaluation of*

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antibacterial effects of pulp capping agents with direct contact test method, Eur J Dent 2014;8: 95.

403. Yamada T, Nakamura K, Iwaku M, et al: *The extent of the odontoblast process in normal and carious human dentin*, J Dent Res 1983;62: 798.
404. Yamani T, Yamashita A, Takeshita N, et al: *Histopathological evaluation of the effects of a new dental adhesive resin on dog dental pulps*, J Jpn Prosthet Soc 1986;30: 671.
405. Yan P, Yuan Z, Jiang H, et al: *Effect of bioaggregate on differentiation of human periodontal ligament fibroblasts*, Int Endod J 2010;43: 1116.
406. Yasuda Y, Ogawa M, Arakawa T, et al: *The effect of mineral trioxide aggregate on the mineralization ability of rat dental pulp cells: an in vitro study*, J Endod 2008;34: 1057.
407. Yazici AR, Baseren M, Gokalp S. *The in vitro performance of laser fluorescence and caries-detector dye for detecting residual carious dentin during tooth preparation*, Quintessence Int 2005;36: 417.
408. Yip HK, Stevenson AG, Beeley JA. *The specificity of caries detector dyes in cavity preparation*, Br Dent J 1994;176: 417.
409. Yoo JS, Chang SW, Oh SR, et al: *Bacterial entombment by intratubular mineralization following orthograde mineral trioxide aggregate obturation: a scanning electron microscopy study*, Int J Oral Sci 2014;6: 227.
410. Yoo YJ, Baek SH, Kum KY, et al: *Dynamic intratubular biomineralization following root canal obturation with pozzolan-based mineral trioxide aggregate sealer cement*, Scanning 2016;38: 50.
411. Yoshimura A, Lien E, Ingalls RR, et al: *Cutting edge:*

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recognition of gram-positive bacterial cell wall components by the innate immune system occurs via toll-like receptor 2, J Immunol 1999;163: 1.

412. Yuan Z, Peng B, Jiang H, et al: *Effect of bioaggregate on mineral-associated gene expression in osteoblast cells*, J Endod 2010;36: 1145.
413. Zanini M, Sautier JM, Berdal A, et al: *Biodentine induces immortalized murine pulp cell differentiation into odontoblast-like cells and stimulates biomineralization*, J Endod 2012;38: 1220.
414. Zhang H, Pappen FG, Haapasalo M. *Dentin enhances the antibacterial affect of mineral trioxide aggregate*, J Endod 2009;35: 221.
415. Zias J, Numeroff K. *Operative dentistry in the second century BCE*, J Am Dent Assoc 1987;114: 665.
416. Zoufan K, Jiang J, Komabayashi T, et al: *Cytotoxicity evaluation of gutta flow and endo sequence BC sealers*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112: 657.

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[a]Adapted from Hahn and Liewehr.[175–177]

[†]Adapted from Bogen and Chandler.[55]

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25: Endo-perio

Gerald N. Glickman, Vincent J. Iacono

CHAPTER OUTLINE

The interrelationships between pulpal and periodontal disease primarily occur

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by way of the intimate anatomic and vascular connections between the pulp and the periodontium; these interrelationships have been traditionally demonstrated using radiographic, histologic, and clinical criteria. Pulpal and periodontal problems are responsible for more than 50% of tooth loss.[17] Diagnosis is often challenging because these diseases have been primarily studied as separate entities, and each primary disease may mimic clinical characteristics of the other disease. Some studies suggest that these two diseases may have etiologic influences on the progression of the other disorder. Pulp tissue succumbs to degeneration by way of a multitude of insults, such as caries, restorative procedures, chemical and thermal insults, trauma, and periodontal disease. When products from pulp degeneration reach the supporting periodontium, rapid inflammatory responses can ensue that are characterized by bone loss, tooth mobility, and sometimes sinus tract formation. If this occurs in the apical region, a periradicular lesion forms. If this occurs with crestal extension of the inflammation, a retrograde periodontitis or reverse pocket is formed. However, the lesion formed has little anatomic similarity to a periodontally induced defect.

The 2017 World Workshop, jointly held by the American Academy of Periodontology and European Federation of Periodontology, developed an evidence-based classification of periodontal and peri-implant diseases and conditions.[16] The classification of periodontitis has been clarified to include staging, extent, and grading.[62] The staging system indicates whether there is stage 1 (mild disease), stage 2 (moderate disease), stage 3 (severe disease with ≤ four missing teeth), and stage 4 (severe disease with ≥ five missing teeth and complex needs) disease.[62] The pathophysiology of the disease stages is similar and includes a dysbiosis of the plaque biofilm in response to the host response and energizing this response. Independent of the dysbiosis of the biofilm, the loss of connective tissue attachment and the associated bone resorption are attributed to the proinflammatory cytokines, prostaglandins, and matrixmetalloproteinases elicited by the host response.[81] The severity of the staging system also includes the extent of disease presentation from localized (less than 30% of teeth involved), generalized (greater than 30% of teeth involved) to a molar/incisor pattern (previously termed localized aggressive periodontitis).[81] In addition, the new classification system includes grading, which is an indication of the rate of

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disease progression from grade A (slow rate), grade B (moderate rate), to grade C (rapid rate).[62]

Regardless of the staging of periodontitis, endo-periodontal lesions occur at teeth with or without root damage (e.g., fracture, root perforation) and in patients with or without periodontitis.[37] Although there is a lack of evidence indicating a unique pathophysiology between a periodontal lesion and an endo-periodontal lesion, the combined lesion adversely affects the successful management of the involved teeth.[37,62] This chapter discusses the intercommunication between pulpal and periodontal tissues; the effects of pulpal disease on the periodontium; periodontal disease and its effects on the pulp; and the classification, differential diagnosis, and management and prognosis of endodontic and periodontal problems.

Intercommunication between pulpal and periodontal tissue

Several possible channels between the pulp and periodontium that lead to the interaction of the disease process in both tissues have been suggested. These include neural (i.e., reflex) pathways, lateral canals, dentinal tubules, palatogingival grooves, periodontal ligament, alveolar bone, apical foramina, and common vasculolymphatic drainage pathways. The most interconnected and evidenced relationship between the two tissues is by way of the vascular system, as illustrated anatomically by the presence of the apical foramen, lateral (i.e., accessory) canals, and dentinal tubules.[7,19,47,56,71,79] These communications, when they exist, may serve as paths for inflammatory reciprocity.[13,34,47,49,56,69]

The apical foramen is the most direct route of communication to the periodontium, but by no means is it the only location where pulpal and periodontal tissues communicate with each other. Lateral and accessory canals, mainly in the apical area and in the furcation of molars, also connect the dental pulp with the periodontal ligament. Table 25.1 lists the incidence of furcation canals. These have been suggested as a direct pathway between pulp and periodontium and typically contain connective tissue and vessels that connect the circulatory system of the pulp with that of the periodontium. Research has demonstrated that inflammation in the interradicular periodontal

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tissues may develop after the induction of pulpal inflammation.[73,74] Serial sectioning of 74 teeth revealed that 45% of accessory canals were present, primarily in the apical region.[69] More significantly, lateral accessory canals in eight teeth were located more coronally on the roots. Among them, connection of the accessory canals with periodontal pockets was microscopically demonstrable in five of the specimens. Gutmann[34] introduced safranin dye into 102 molar teeth that were placed in a vacuum chamber and found that 28% of the teeth had furcation canals, although only 10% of the total group showed canals on the lateral root surface.

Table 25.1

Incidence of Furcation Canals

Investigators	Techniques	Incidence
Rubach and Mitchell (1965)[69]	Sectioned teeth	45%
Lowman et al. (1973)[56]	Dissecting microscope	Maxillary molars: 59% Mandibular molars: 55%
Burch and Hulen (1974)[13]	Radiopaque dye	Accessory furcal canals: 76%
Vertucci and Williams (1974)[84]	Hematoxylin dye	46%
Kirkham (1975)[45]	Radiopaque dye	23%
Gutmann (1978)[34]	Safranin dye	Maxillary molars: 28.4% Mandibular molars: 27.4%

In addition to the apical foramen and lateral accessory canals, dentinal tubules have been suggested as another common pathway between the periodontium and pulpal tissue. Dentinal tubules contain cytoplasmic extensions or odontoblastic processes that extend from the odontoblasts at the pulpodentin interface to the dentinoenamel junction (DEJ) or the cementodentinal junction (CDJ). It has been reported that the pulp chamber can communicate with the external root surface by way of dentinal tubules, especially when the cementum is denuded.[34,88]

Palatogingival grooves are developmental anomalies of the maxillary incisor teeth; lateral incisors are more often affected than central incisors

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(4.4% vs. 0.28%, respectively).[88] These grooves usually begin in the central fossa, cross the cingulum, and extend apically for varying distances. Generally, the incidence of palatogingival grooves ranges from 1.9% to 8.5%.[27,88] In their study, Everett and Kramer[27] reported that 0.5% of the teeth examined had a palatogingival groove extension to the root apex, thus contributing to an endodontic pathologic condition. Bilateral buccal radicular grooves have also been reported on maxillary incisors.[43]

Perforation of the root creates a communication between the root canal system and the periodontal ligament. This may occur as a result of overinstrumentation during endodontic procedures, internal or external root resorption, or caries invading through the floor of the pulp chamber. The prognosis for teeth with root perforation is usually determined by the location of the perforation, the time left unsealed, the ability to seal the perforation, the chance of building new attachments, and the accessibility of the remaining root canals. Teeth that have perforations in the middle or apical third of the root have the greatest chance of healing. The closer the perforation is to the gingival sulcus, particularly into the coronal third of the root or the furcation region, the greater the likelihood of apical migration of the gingival epithelium in the initiation of a periodontal lesion.[4]

A vertical root fracture can produce a “halo” effect around the tooth radiographically (see also Chapter 2).[67] Deep periodontal pocketing and localized destruction of alveolar bone are often related to longstanding root fractures. The fractured root can mimic a radiographic profile of occlusal trauma, with localized loss of lamina dura, altered trabecular pattern, and a widened periodontal ligament. The fracture site provides a portal of entry for irritants from the root canal system to the surrounding periodontal ligament. Vertical root fractures have contributed to the progression of periodontal destruction in the presence of apparently successful endodontic tooth therapy and overall periodontal site stability.[68]

Influence of pulpal pathologic condition on the periodontium

Pulpal pathosis as a cause of periodontal disease has received much attention during the past decade. Pulpal degeneration results in necrotic debris,

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bacterial by-products, and other toxic irritants that can move toward the apical foramen, causing periodontal tissue destruction apically and potentially migrating toward the gingival margin. Investigators termed this *retrograde periodontitis* to differentiate the process from *marginal periodontitis*, in which the disease proceeds physically from the gingival margin toward the root apex. When pulpal disease progresses beyond the confines of the tooth, inflammation extends and affects the adjacent periodontal attachment apparatus.[77] This inflammatory process often results in dysfunction of the periodontal ligament and resorption of alveolar bone, cementum, and even dentin.

The endodontic infection has been regarded as a local modifying risk factor for periodontitis progression if left untreated.[24] It is believed that an unresolved periapical infection could sustain endodontic pathogen growth, and infectious products would egress into the periodontium by way of the apex and lateral or accessory canals, as well as encourage osteoclastic activity. These may aggravate periodontal pocket formation and bone loss and impair wound healing to accelerate further periodontal disease development and progression. In addition, the high concentrations of the medicaments used for root canal therapy (e.g., calcium hydroxide, corticosteroids, and antibiotics) can irritate the periodontal attachment apparatus.[10,11] The nature and extent of periodontal destruction depend on several factors, including the virulence of the irritating stimuli present in the root canal system (e.g., microbiota, medications, foreign body reactions), the duration of the disease, and host defense mechanisms.[17] The rare development of endodontic-periodontal lesions argues that periradicular pathoses in general have little effect on localized inflammatory bone resorption, given that few combined lesions are seen in cases of apical periodontitis.

The ability of the periodontium to regenerate lost attachment apparatus on pulpless teeth has been questioned, especially if these teeth contain a root canal filling and have been denuded of cementum. One investigator suggested that endodontically treated teeth may not respond as well as untreated teeth to periodontal procedures. He found 60% osseous regeneration of periodontal defects in teeth not treated endodontically, compared with 33% defect fill in endodontically treated teeth.[70] However, in

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a monkey study, others reported that all tissue of the periodontium had the potential for regeneration after periodontal surgery, regardless of the status of the pulp (vital, filled, medicated, or open).[21] Another study reported that pulpal status has little influence on initial cementogenesis and that substances leaching from certain root canal filling materials do not alter deposition of new cementum.[65]

Although endodontic infections have been highly correlated with deeper periodontal pockets and furcation involvement in mandibular molars, the causal relationship between the two pathoses has not yet been established.[41] It has been suggested that endodontic treatment should occur before treatment of furcation lesions (i.e., bone regeneration) to ensure successful results. Extensive evidence is lacking to prove this hypothesis, but there is general agreement that with the proper endodontic treatment, periodontal disease of pulpal origin should heal. The question of whether endodontic infections play a significant role in affecting the health of the periodontium remains to be further elucidated.[59]

Influence of periodontal inflammation on the pulp

Clinically, it is not uncommon to observe a stage 3 or 4 periodontitis spreading to the apical foramen, with associated pulp necrosis. It is also recognized that infection from a periodontal pocket may spread to the pulp through accessory canals, which occur most often in the furcation and closer to the apex of teeth.[60] Investigators proved that pulpitis and pulp necrosis can occur as a result of periodontal inflammation involving accessory and apical canals.[69] Bacterial products and toxins may also gain access to the pulp by way of exposed dentinal tubules. The pulpal reaction is influenced not only by the stages of periodontal disease, but also by the type of periodontal treatment, such as scaling, root planing, and administration of medication.[33] Inflammatory lesions of varying severity and necrotic pulp tissue are usually found in teeth with large canals or in cases in which periodontal breakdown has extended to the apex.[33] Whyman[87] stated that during periodontal therapy, the blood vessels supplying the pulp by way of accessory canals may be damaged. Another animal study found that 70% of root specimens examined

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showed no pathologic changes despite the fact that 30% to 40% of the periodontal attachment was lost.[8] The remaining 30% of roots displayed only small inflammatory cell infiltrates or the formation of reparative dentin, or both, in areas where pulp was adjacent to root exposed through periodontal destruction. These tissue changes were frequently associated with root surface resorption, which suggests that dentinal tubules must be uncovered before irritation can be transmitted.

These observations suggest that the presence of an intact cementum layer is important for the protection of the pulp from toxic elements produced by the plaque biofilm microbiota, so periodontal disease and periodontal treatments should be regarded as potential causes of pulpitis and pulpal necrosis. It has also been reported that the pulps of teeth with longstanding periodontal disease develop fibrosis and various forms of mineralization. Canals associated with periodontally involved teeth were reported to be narrower than canals of teeth that were not periodontally involved. This result is thought to be a reparative process rather than an inflammatory response.[7,51]

Although consensus supports the theory that a degenerating or an inflamed pulp can affect the periodontium, not all researchers agree about the effect of periodontal disease on the pulp. Specifically, inflammatory alterations and localized pulp necrosis have been observed adjacent to lateral canals in roots exposed by periodontal disease.[69,73,74] Additional studies have failed to confirm a direct correlation between periodontal disease and pulp tissue changes.[20,58,82] When pathologic changes occur in the pulp as a result of periodontal disease, the pulp usually does not degenerate so long as the main canal is not involved.[50] It seems plausible to assume that periodontal disease rarely jeopardizes the vital function of the pulp. Generally, if the blood supply through the apical foramen remains intact, the pulp is usually capable of withstanding physiologic insults induced by periodontal disease.

Theoretic pathways of osseous lesion formation

For the clinician, the close relationship between pulpal and periodontal disease is reasonably established on clinical and radiographic levels. Because interpretations vary as to which came first (the proverbial “chicken or egg”

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controversy), clinical data gathering in endodontic and periodontal problems is often complex, requiring a medical history review, pulp vitality testing, pocket and furcation probing, tooth mobility determinations, and critical examination of radiographs. When formulating a differential diagnosis, the clinician should first consider both the periodontal and pulpal status of the affected tooth. If an interrelationship in disease entities exists, appropriate treatment must be rendered to remove true causative factors and enhance the prognosis for tooth retention.[76] Fig. 25.1 shows interrelationships between pulpal and periodontal disease.

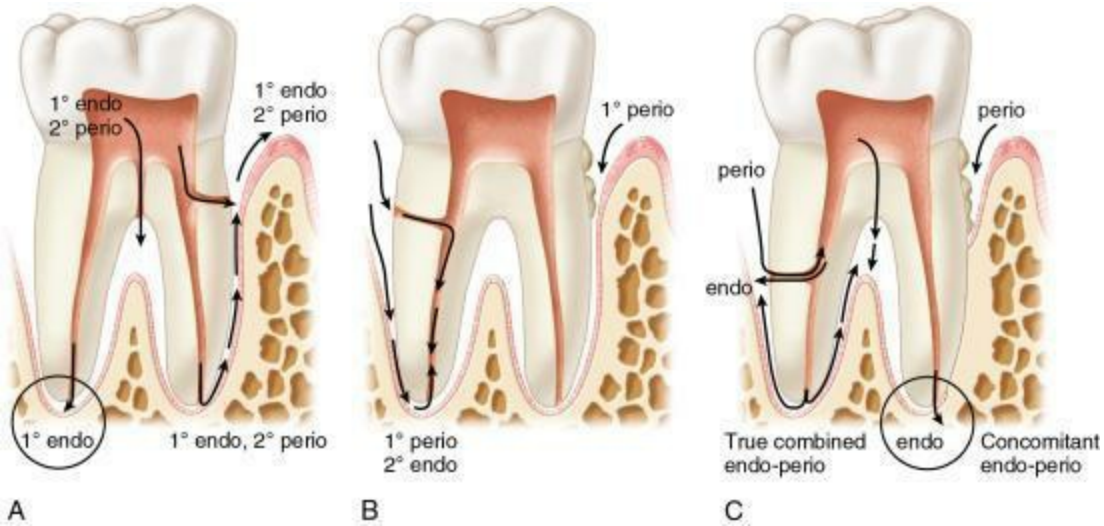

FIG. 25.1 Endodontic and periodontal pathways. **A,** Endodontic lesions. The pathway of inflammation is through the apical foramen, furcation canals, and lateral accessory canals to the periodontium. This results in a primary endodontic lesion, sometimes progressing to secondary periodontal involvement. **B,** Periodontal lesions. This is the progression of periodontitis by way of the lateral canal and apex to induce a secondary endodontic lesion. **C,** True combined endodontic and periodontal lesion and concomitant endodontic and periodontal lesions.

A) Pathway for primary endo and secondary perio from the center of the root below the pulp chamber leads to gum line on the right. A primary endo lesion is encircled at the tip of one root canal.

B) Pathway for primary perio and secondary endo from the root tip of right canal and above the mid root meets slightly above the root tip. A pathway of primary perio from the gum line on the right leads toward the root.

C) It shows true combined endo-perio and concomitant endo-perio. An

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endolesion is encircled at the right root tip.

Primary endodontic lesions

Disease processes of the dental pulp frequently involve inflammatory changes. Caries, restorative procedures, and traumatic injuries are the most common causes. Typically, endodontic lesions resorb bone apically and laterally and destroy the attachment apparatus adjacent to a nonvital tooth. Inflammatory processes in the periodontium that occur as a result of root canal infection not only may be localized at the apex, but also may appear along the lateral aspects of the root (Fig. 25.2) and in furcation areas of two- and three-rooted teeth (Fig. 25.3).

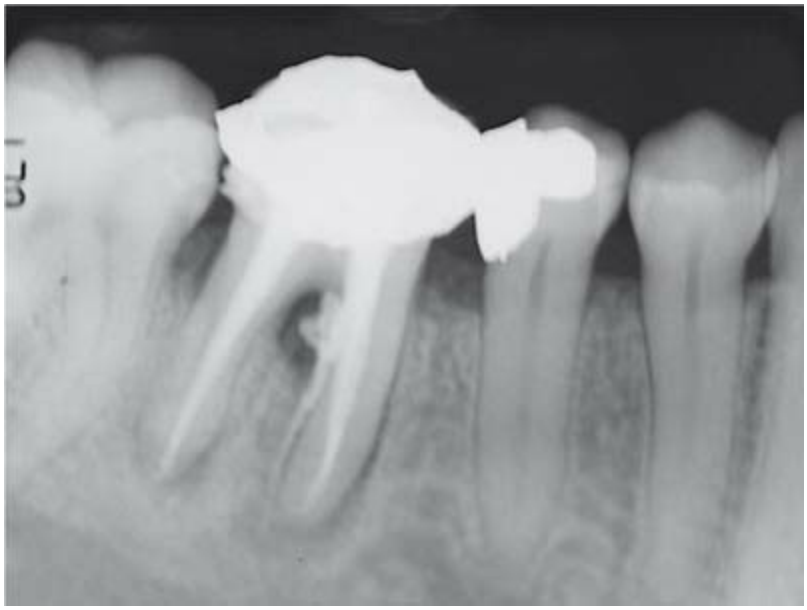

FIG. 25.2 Primary endodontic lesion. Mandibular molar showing endodontic filling material extending into the furcation and along the lateral root surface because of inadequate canal preparation. Furcation could be probed.

Radiograph of lower jaw shows molar with radiolucent patch at the center of beginning of root system. The canal on the left has less-bone density at the tip.

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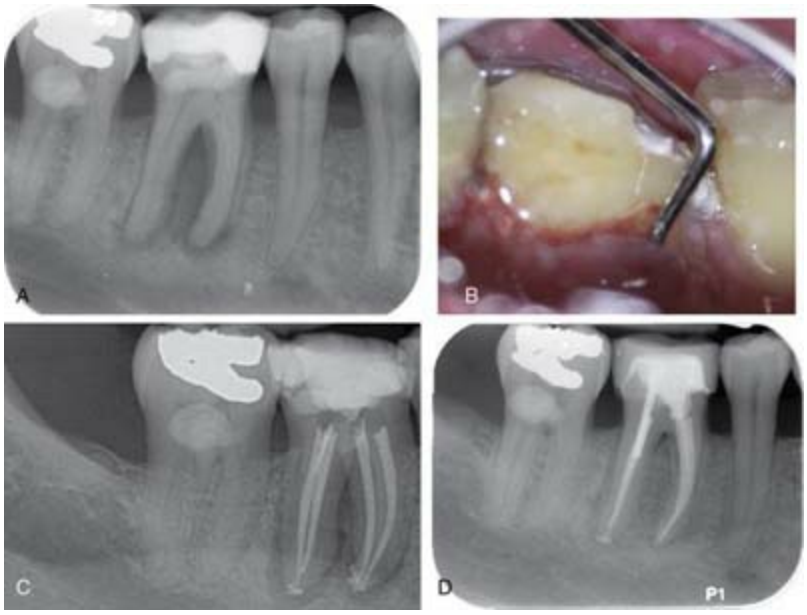

FIG. 25.3 Primary endodontic lesion on mandibular first molar. **A,** Mandibular first molar with deep restoration and pulpal necrosis; apical radiolucency on mesial and distal roots with furcation radiolucency. **B,** Probing of 12 mm with periodontal probe. **C,** X-ray image of completed root canal treatment. **D**, Eight months after root canal treatment, there is evidence of osseous healing at both the apex and furcation area.

Set of radiographs and photograph marked A through D shows the restoration of radiolucent patch in-between the canals and less-bone density in the pulp chamber of first molar in the lower jaw.

Source: (Courtesy Dr. Elham Shadmehr, San Francisco, CA.)

The emergence of these processes may be associated with clinical signs of inflammation: pain, tenderness to pressure and percussion, increased tooth mobility, and swelling of the marginal gingiva, simulating a periodontal abscess. The suppurative process may cause a sinus tract along the periodontal ligament space or through patent channels (including the apical foramen and lateral accessory canals). This usually results in a narrow opening of the sinus tract into the gingival sulcus and pocket that can be easily traced with a gutta-percha cone or a periodontal probe. Such a tract can readily be probed down to the tooth apex, where no increased probing depth would otherwise exist around the tooth. In multirooted teeth, a periodontal ligament sinus tract can drain off into the furcation area and resemble a grade III through-and-through furcation defect resulting from periodontal disease.

Clinically, endodontic testing procedures should reveal a necrotic pulp or,

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in multirooted teeth, at least an abnormal response, indicating that the pulp is degenerating. Because the primary lesion is an endodontic problem that has merely manifested itself through the periodontal ligament, complete resolution is usually anticipated after nonsurgical endodontic therapy, without any periodontal treatment.

Primary endodontic lesions with secondary periodontal involvement

When a lesion of endodontic origin is not treated, pathosis usually continues, leading to destruction of the periapical alveolar bone and progression into the interradicular area, causing breakdown of surrounding hard and soft tissues (Fig. 25.4). As drainage persists through the gingival sulcus, accumulation of plaque biofilm and calculus in the purulent pocket results in periodontal disease and further apical migration of the attachment. When this occurs, not only does the diagnosis become more difficult, but the prognosis and treatment may be altered. Diagnostically, these lesions have a necrotic root canal and plaque biofilm or calculus accumulation, demonstrable by a probe and radiograph. Radiographs may show generalized periodontal disease with angular defects at the initial site of the endodontic involvement.

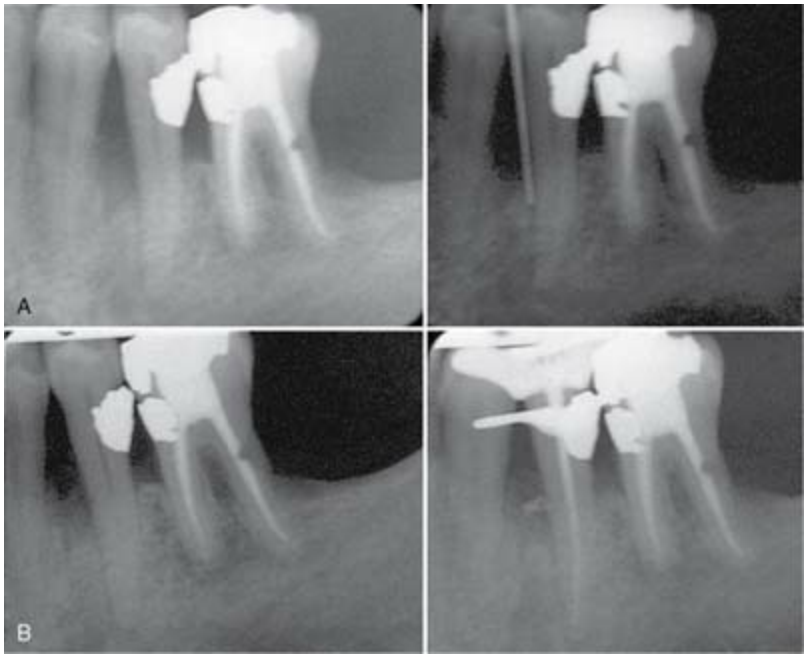

FIG. 25.4 Primary endodontic lesion with secondary periodontal

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involvement. **A,** The mesial aspect of the mandibular second premolar had deep periodontal pocketing (a periodontal probe in place demonstrated 6 mm of probing depth), even after periodontal therapy. **B,** Pulp tests were performed on the premolar; the tooth was nonresponsive. After endodontic treatment, the periodontal pocket resolved (filling of a lateral accessory canal after obturation is demonstrated).

Four radiographs are shown as follows:

A: Three teeth are shown the central tooth has a large radiolucent patch on the right of root.

Radiograph on top-right: A tube is inserted into the radiolucent patch.

B radiograph: The tissue at the root tip of tooth has somewhat high density as compared to first two radiographs.

Radiograph at bottom-right: The radiolucent patch has high density as compared to first three radiographs.

Resolution of the primary endodontic and secondary periodontal lesion relies on treatment of both conditions. When only endodontic therapy is provided, only part of the lesion can be expected to heal. If endodontic therapy is adequate, the prognosis depends on the severity of periodontal involvement and the efficacy of periodontal therapy.

Primary periodontal lesions

Periodontal disease has a progressive nature. It begins in the sulcus and migrates to the apex as deposits of plaque biofilm and calculus produce inflammation, causing loss of surrounding alveolar bone and supporting periodontal soft tissues. This leads to a loss of clinical attachment and the formation of a periodontal abscess during the acute phase of destruction.[75] The progression of periodontal disease to the formation of osseous defects and subsequent radiographic appearance along lateral aspects of roots and in furcation areas is well known. These defects may or may not be in association with trauma from occlusion, which can often be the cause of an isolated periodontal problem. Osseous lesions of periodontal origin are usually associated with tooth mobility, and the affected teeth respond positively to pulp testing. In addition, careful periodontal examination usually reveals broad-based pocket formation and an accumulation of plaque biofilm

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and calculus. The bony lesion is usually more widespread and generalized than are lesions of endodontic origin (Fig. 25.5).

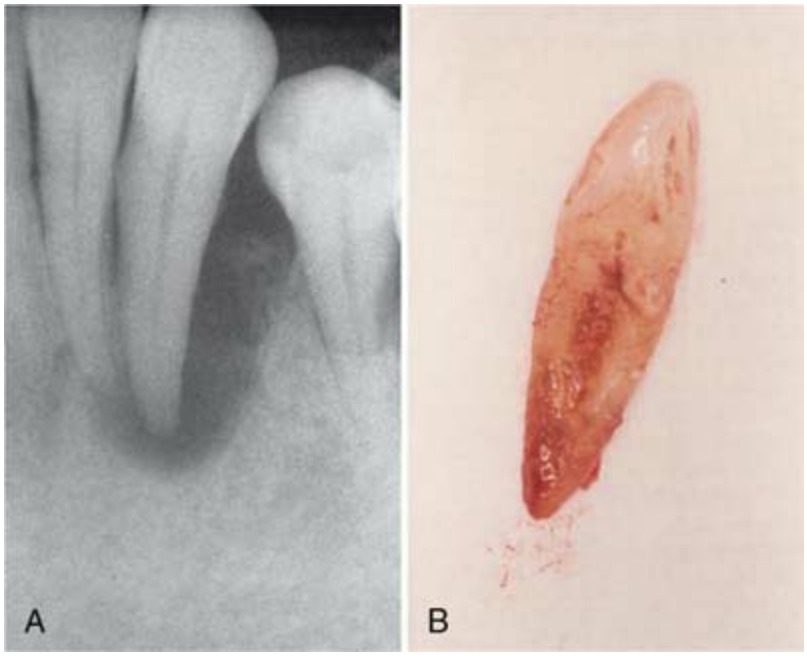

FIG. 25.5 Primary periodontal lesion. **A,** Mandibular cuspid exhibits extensive periodontal destruction; the tooth responded normally to pulp tests. **B,** Extracted tooth depicts extensive calculus accumulation and root concavity.

A) Radiograph shows J-shaped radiolucency along the root of tooth.

B) Extracted tooth with blood stain is kept on a platform.

The prognosis for teeth affected by periodontitis worsens as the disease process and periodontal destruction progress. Treatment depends on the extent of the periodontitis and on the patient's ability to comply with potential long-term treatment and maintenance therapy. Because this is purely a periodontal problem, the prognosis depends exclusively on the outcome of periodontal therapy.

Primary periodontal lesions with secondary endodontic involvement

As stated earlier, periodontal disease can have an effect on the pulp through dentinal tubules, lateral canals, or both. Primary periodontal lesions with

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secondary endodontic involvement differ from the primary endodontic lesion with secondary periodontal involvement only by the temporal sequence of the disease processes. The tooth with primary periodontal and secondary endodontic disease exhibits deep pocketing, with a history of extensive periodontal disease and, possibly, past treatment. When the pulp becomes involved, the patient often reports accentuated pain and clinical signs of pulpal disease. This situation exists when the apical progression of periodontal disease is sufficient to open and expose the pulp to the oral environment by way of lateral canals or dentinal tubules. On radiographs, these lesions may be indistinguishable from primary endodontic lesions with secondary periodontal involvement. The prognosis depends on continuing periodontal treatment subsequent to endodontic therapy.

True combined lesions

Pulpal and periodontal disease may occur independently or concomitantly in and around the same tooth. Once the endodontic and periodontal lesions coalesce, they may be clinically indistinguishable (Fig. 25.6). The prognosis of multirooted teeth with combined pulpal and periodontal lesions depends largely on the extent of the destruction caused by the periodontal disease component. A necrotic pulp or a failing endodontic treatment, plaque biofilm, calculus, and periodontitis will be present in varying degrees.

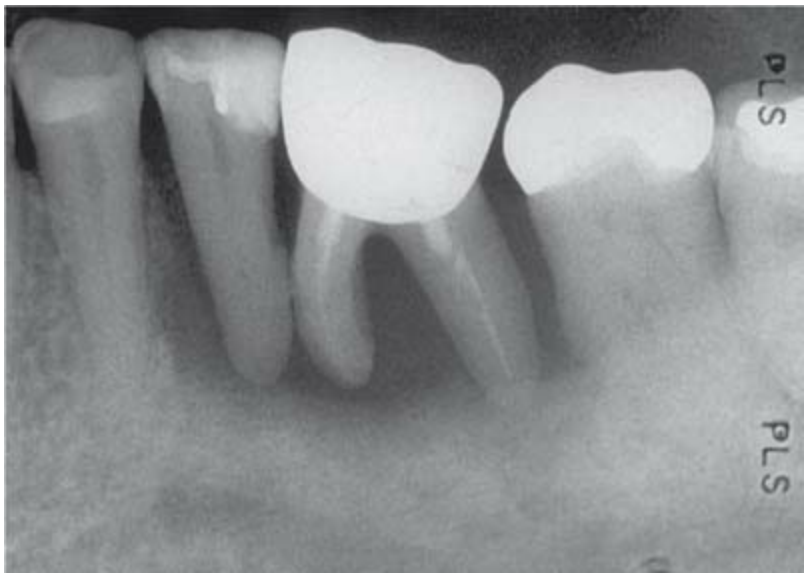

FIG. 25.6 True combined pulpal and periodontal lesions on the

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mandibular second premolar and first molar. Periodontal probing depths were to the apices in both teeth.

Radiograph shows four teeth where the surrounding and bifurcation region of central two teeth are completely radiolucent.

Concomitant pulpal and periodontal lesions

An additional classification has been proposed for lesions that may commonly be seen clinically and reflect the presence of two separate and distinct entities.[6] This is referred to as the *concomitant pulpal and periodontal lesion* (Fig. 25.7). In essence, both disease states exist, but with different causative factors and with no clinical evidence that either disease state has influenced the other. This situation often goes undiagnosed, and treatment is rendered to only one of the diseased tissues in the hope that the other responds favorably. In actuality, the two disease processes must be treated concomitantly, and the prognosis depends on the removal of the individual etiologic factors and the prevention of any further factors that may affect the respective disease processes.

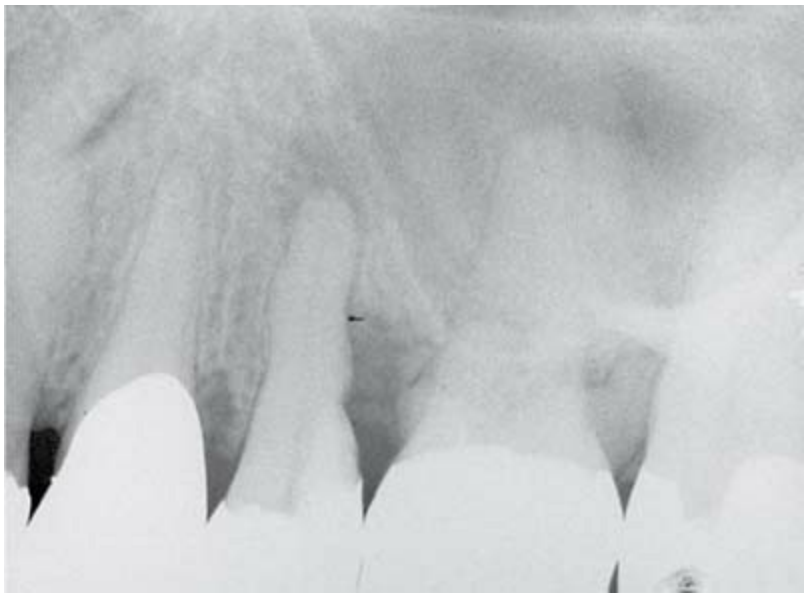

FIG. 25.7 Concomitant pulpal and periodontal lesion on the maxillary second premolar. An endodontic lesion was noted at the apex, with a noncommunicating periodontal pocket on the distal side.

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Radiograph shows the posterior view of four teeth where the second premolar has radiolucent pockets on the gum line and a radiolucent patch at the root tip.

With the application of research findings and clinical experience, pathways of osseous lesion formation may be theorized. These concepts are intended as a guide for the evaluation and understanding of the clinician's successes and failures in treating teeth with osseous lesions. By understanding the nature of their formation, the clinician can better anticipate the healing potential after treatment.

Differential diagnosis

During the course of treatment, clinicians are frequently presented with the dilemma of accurately assessing the contribution of endodontic and periodontal lesions. These lesions may be very separate from each other and present no extraordinary therapeutic consideration. In a few other situations, there is no obvious demarcation between the two lesions, which appear as one, both on radiographs and clinically. In the diagnosis of radiographic osseous lesions, the clinician must resist the temptation to label everything a "combined lesion." Table 25.2 summarizes the differential diagnosis between pulpal and periodontal lesions and highlights a number of common characteristics of these lesions.

Table 25.2

Differential Diagnosis Between Pulpal and Periodontal Disease

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	Pulpal	Periodontal
CLINICAL		
Etiology	Pulp infection	Periodontal infection
Vitality	Nonvital	Vital
Restorative	Deep or extensive	Not related
Plaque biofilm/calculus	Not related	Primary cause
Inflammation	Acute	Chronic
Pockets	Single, narrow	Multiple, wide coronally
pH value	Often acid	Usually alkaline
Trauma	Primary or secondary	Contributing factor
Microbial	Few	Complex
RADIOGRAPHIC		
Pattern	Localized	Generalized
Bone loss	Wider apically	Wider coronally
Periapical	Radiolucent	Not often related
Vertical bone loss	No	Yes
HISTOPATHOLOGIC		
Junctional epithelium	No apical migration	Apical migration
Granulation tissues	Apical (minimal)	Coronal (larger)
Gingival	Normal	Some recession
THERAPY		
Treatment	Root canal therapy	Periodontal treatment

Root fractures, especially vertical root fractures, present particular problems in diagnosis (Fig. 25.8). Symptoms and signs associated with vertical root fractures show a varying character and are frequently difficult to distinguish from those associated with periodontal and endodontic lesions. Breakdown can manifest itself on radiographs in a number of different ways. This may be anything from nothing detectable on radiographs to an area of rapid vertical bone loss, given that there is no way to predict when a patient may present for treatment. It is imperative for the clinician to take more than one radiograph at different angles, especially when no clear diagnosis emerges. In these cases, a minor alteration in the angulation may reveal a tooth fracture or periodontal furcation involvement. The diagnosis of vertical root fractures is often difficult because the fracture is usually not detectable by clinical inspection and radiographic examination unless there is a clear separation of the root fragments. Cemental tears or detachment of cementum from a root surface by means of trauma or aging has been reported.[14,53] The lesion often results in periodontal destruction and endodontic involvement (Fig. 25.9).[54,55]

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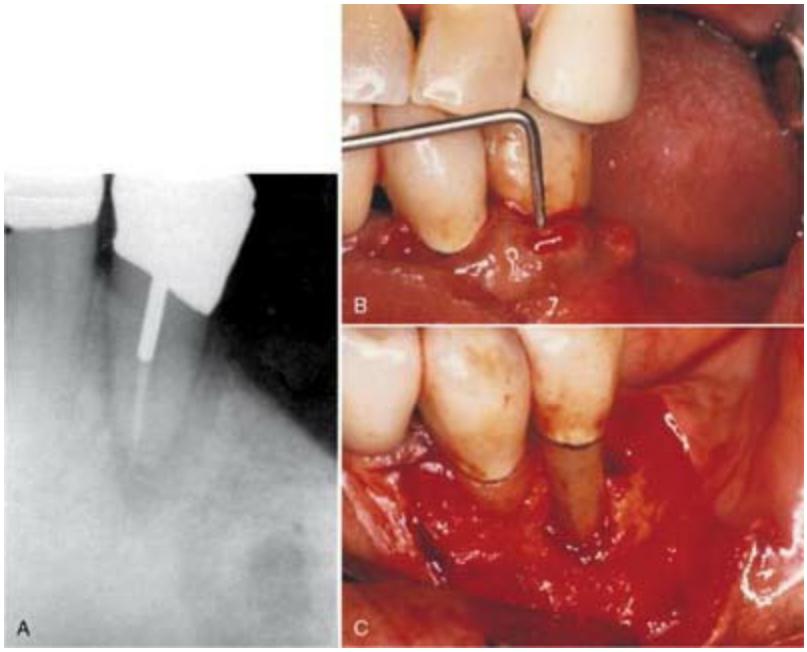

FIG. 25.8 Vertical root fracture. **A,** A radiograph revealed a widened periodontal ligament with a J-shaped radiolucency around the apex. **B,** A periodontal probe indicated more than 12 mm of probing depth. **C,** Exploratory surgery confirmed a vertical root fracture.

Set of radiograph and photographs marked A through C reveals the vertical root fracture extending from the mid root to apex along during the examination of periodontal pocket.

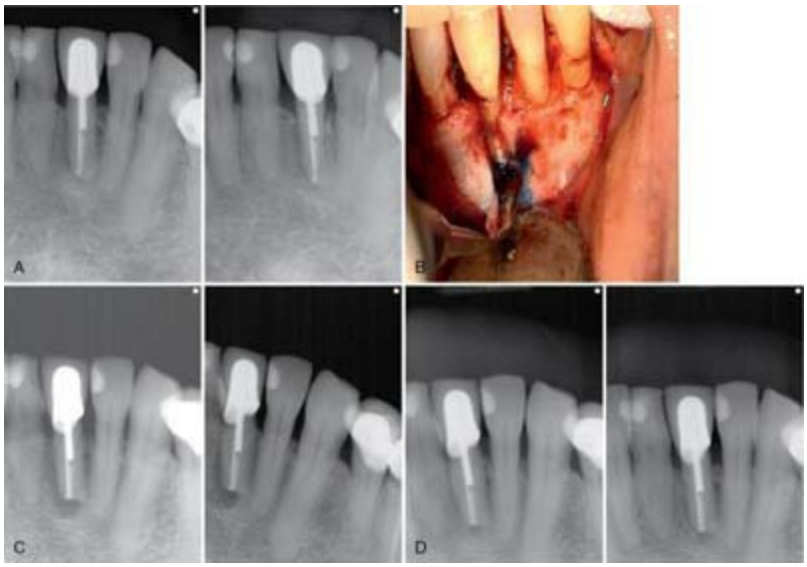

FIG. 25.9 Cemental tear. **A,** Radiographs revealed an endodontically treated tooth with post, separated instrument, and apical osseous

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breakdown; the tooth was very tender to both percussion and palpation. **B,** An "eggshell" piece of cementum on the buccal surface of the root before root-end resection; the cementum was removed with curettage. **C,** Postoperative radiographs after root-end resection, root-end preparation, and root-end filling. **D,** Six-month follow-up radiographs demonstrate periapical healing; the tooth was asymptomatic.

Set of six radiographs and a photograph marked A through D shows the cemental tear in tooth along with its healing.

Sometimes, the definitive diagnosis of vertical root fractures has to be confirmed by exploratory surgical exposure of the root for direct visual examination.[85] Vertical root fractures have been associated with root-filled teeth where excessive lateral forces were applied during compaction or possibly with stress induced by post placement in root-filled teeth.[61] A clinical survey of fractured teeth also reveals that fractures are more common in teeth with extensive restorations, in older patients, and in mandibular posterior teeth.[31] The prognosis for teeth with vertical root fractures that involve the gingival sulcus and periodontal pocket usually is hopeless because of continuous bacterial invasion of the fracture space from the oral environment. Single-rooted teeth are generally extracted. In multirooted teeth, a treatment alternative is hemisection or resection of the fractured root. (See Chapter 22 for further discussion of vertical root fractures.)

Developmental grooves, primarily found in maxillary central and lateral incisors, are also capable of initiating localized periodontal destruction along the root surface.[27,43,52] It is thought that they may be a genetic attempt to form an accessory root, but once plaque biofilm and calculus invade the epithelial attachment, the groove becomes a pathway for microbes and their noxious allies (toxins), with ample substrate from food debris to create a self-supporting periodontal lesion. Bone demineralization follows the path of the groove. Palatogingival grooves are often associated with poor periodontal health because of patients' inability to keep these areas clean, and a poor prognosis is usually assigned regardless of proper conventional therapy.[31,88] It is easy to identify these grooves if the clinician is aware of their existence. Clinically, these grooves may be asymptomatic, or symptomatic periodontal problems (either acute or chronic) may occur. It is thought that the pulp of these teeth may become secondarily involved and demonstrate symptoms of

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pulpal disease (Fig. 25.10). These lesions are often confused with the enamel projections in the furcation area of mandibular molars.[58] The prevalence of cervical enamel projections (Fig. 25.11) ranges from 18% to 45%.[38,58] Depending on the apical extent of these cervical grooves, some authors[38,58] also find a high association with pathologic furcation involvement (up to 82.5%[38]).

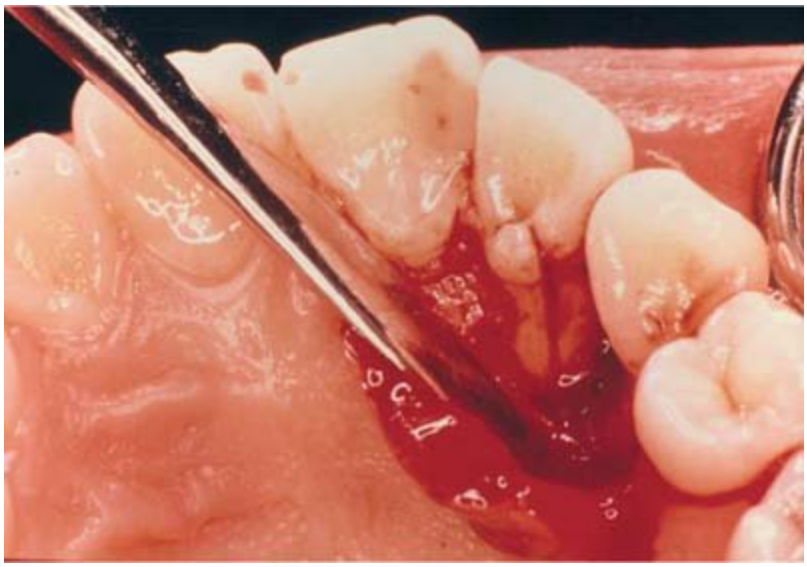

FIG. 25.10 Palatogingival groove on a maxillary lateral incisor with a periodontal defect.

Close-up view of a probe at the site of a large pocket exposes the root of incisor in the upper jaw.

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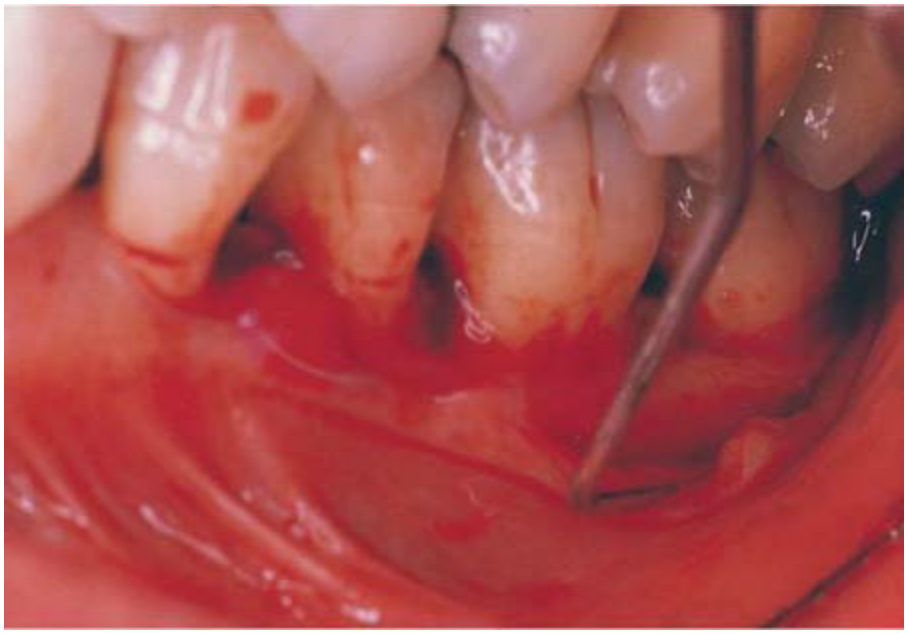

FIG. 25.11 Cervical enamel projection on a mandibular molar.

Close-up of lower jaw shows a probe examining a tooth with enamel projection in the bifurcation region of root.

Lateral periodontal cysts

The clinical presentation of a lateral periodontal cyst is often without symptoms (Fig. 25.12). It may present as a gingival swelling on the facial aspect that may show pain and tenderness on palpation. Radiographic features are a well-circumscribed round or ovoid radiolucent area that usually has a sclerotic margin. Most lateral periodontal cysts are less than 1 cm in diameter and lie somewhere between the apex and cervical margin of a tooth. Three possible etiologies are reported in the literature: (1) reduced enamel epithelium, (2) remnants of dental lamina, or (3) cell rests of Malassez. Histologic evaluation reveals that these cysts are lined by epithelium that closely resembles reduced enamel epithelium lining. The lesion occurs predominantly in the fifth to seventh decades of life, with a predilection for males.[3] The lesion is usually slow growing. Treatment consists of careful excision to help prevent recurrence. The most common location is the mandibular cuspid-bicuspid area, although numerous cases have been reported in the anterior maxilla.

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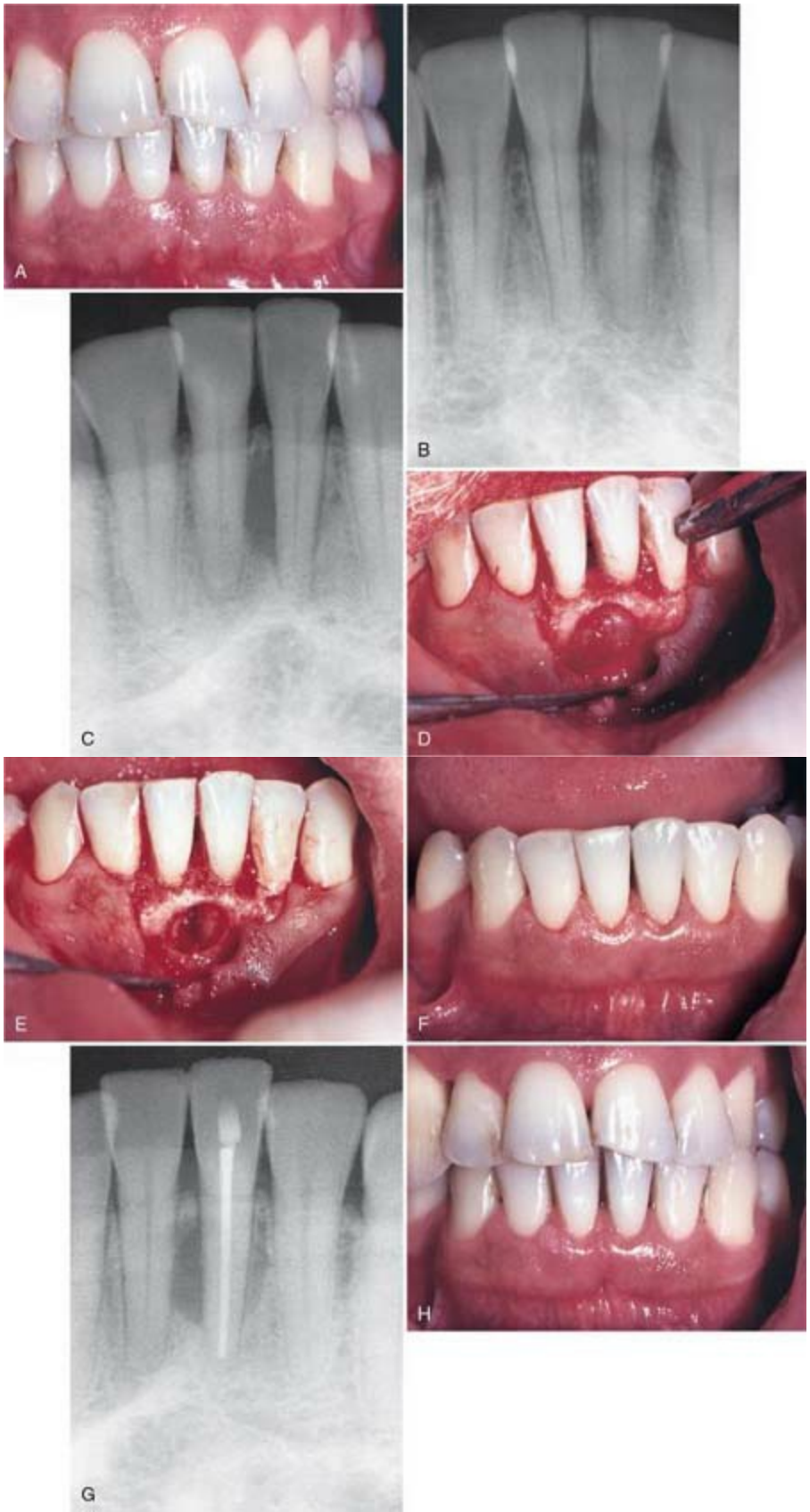

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FIG. 25.12 Lateral periodontal cyst. **A,** Clinical photograph showing mild gingivitis around tooth #24; there is a 5-mm probing defect on the mesial aspect of tooth #24. **B,** A radiograph depicts the beginning lesion 2 years earlier. **C,** A radiograph shows the extent of the lesion at the time of referral to the periodontist. **D,** Initial flap reflection; the lesion is present interproximally between teeth #24 and #25. **E,** The lesion was enucleated and submitted for histologic evaluation; the diagnosis was a lateral periodontal cyst. **F,** Three-month posttreatment clinical photograph; tooth #24 was nonvital and symptomatic and was referred for root canal treatment. **G,** One-year posttreatment radiograph showing completed root canal treatment and osseous defect still present; continuous recall recommended. **H,** One-year posttreatment clinical photograph; probing depths were within normal limits; continuous recall was recommended.

Set of radiographs and photographs marked A through D shows the changes in fluid filled lesion below the gum line of teeth 24 and 25.

Set of photographs and radiographs marked E through H shows a cavity with two oval structures below teeth 24 and 25 and its partial healing after treatment.

Altini and Shear[3] reported finding unicystic and multicystic (including botryoid) varieties. They found that lateral periodontal cysts were lined predominantly or exclusively by thin, reduced enamel, epithelium-like tissue that contained many clear cells, and epithelial thickenings referred to as *plaque biofilms*. Glycogen was present in the epithelium of two thirds of the cases, although not exclusively in the clear cells, many of which were not positive for glycogen. Some of their botryoid variety differed histologically, being lined predominantly by nonkeratinizing stratified squamous epithelium with crowded and pyknotic nuclei and no clear cells. One case contained melanin, and another showed epithelial crypt formation and superficial palisaded low columnar cells, as seen in the glandular odontogenic cyst. This raised the question of whether the latter may form part of the clinicopathologic spectrum of a lateral periodontal cyst. The histogenesis of lateral periodontal cysts is uncertain, but these cysts favor origin from reduced enamel epithelium. Some cases either do not fit a characteristic endodontic or periodontal lesion or do not respond to treatment as expected. A biopsy and histologic analysis are often recommended. Systemic diseases such as scleroderma, metastatic carcinoma, and osteosarcoma can mimic endodontic and periodontal disease visible on a radiograph. *The*

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conscientious clinician must always be alert for lesions of nonendodontic or nonperiodontal origin and look for other causes.

Treatment alternatives

When traditional endodontic and periodontal treatments prove insufficient to stabilize an affected tooth, the clinician must consider treatment alternatives. Generally, a localized periodontal defect associated with an endodontically untreatable tooth or an iatrogenic tooth problem is reason to explore other treatment options. Alternate treatments often consist of resection or regenerative approaches. Resection techniques focus on eliminating the diseased roots or teeth, while regenerative efforts are aimed at restoring lost biologic structures. Resection methods involve removal of affected roots or extraction of involved teeth. When a tooth requires extraction, one of the first options for restoring occlusal function should include placement of dental implants with hybrid prostheses. Bone replacement grafts using guided tissue and bone regeneration techniques are ways to reestablish biologic structures that were lost during this disease process. Fixed partial dentures are still a viable option for some patients and certainly should be considered if abutment teeth already have restorations or endodontic treatment.

Root resection is the removal of a root, with accompanying odontoplasty before or preferably after endodontic treatment.[28] Formerly, it was used when root canal therapy was considered too difficult, but now its indications are restricted to multirooted teeth in which one or more roots cannot be saved. The indications for root resection often include (but are not limited to) root fracture, perforation, root caries, dehiscence, fenestration, external root resorption involving one root, incomplete endodontic treatment of a particular root, stage 3 or 4 periodontitis affecting only one root, and severe grade II or grade III furcation involvement. Root resection is a technique-sensitive procedure (Fig. 25.13) requiring a careful diagnostic process for selection of teeth that would likely be successful candidates, followed by meticulous interdisciplinary treatment. Factors such as occlusal forces, tooth restorability, and the value of the remaining roots must be examined before treatment. A carefully constructed treatment plan is crucial to the success of this resection procedure.[32] Proper reshaping of the occlusal table and

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restoration of the clinical crown are essential, and the root surface must be recontoured to remove the root stump, thus preventing formation of a potential food trap.[44]

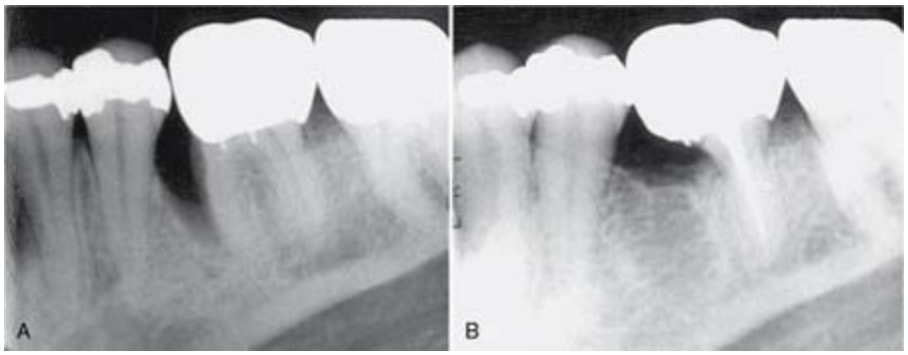

FIG. 25.13 Root resection to correct endodontic and periodontal defect. **A,** Pretreatment radiograph depicts severe periodontal involvement on the mesial. Endodontic treatment was performed before surgical removal of the mesial root. **B,** Five-year follow-up shows the remaining tooth well maintained.

A) Radiograph shows a V-shaped pocket between two teeth.

B) Radiograph shows porous tissue in place of the pocket.

The effectiveness of this approach remains controversial, owing to the disparity of results reported in several long-term studies.[4,10,12,15,25,28,46] Retrospective longitudinal studies have observed the fate of sectioned teeth for time frames ranging from 3 to 12 years and have reported success rates ranging from 62% to 100%, with a low incidence (i.e., 10%) of periodontal breakdown. However, as most long-term studies will point out, the major cause of failure of resection procedures resides in failure of the endodontic and restorative components. Unique anatomic features, such as root length, curvature, shape, size, position of adjacent teeth, and bone density, may influence the end result. For example, root fusion makes resection all but impossible. The removal of roots purely to eliminate a resorptive or traumatic perforation defect, fractured root, or endodontically inoperable root usually results in definitive treatment. If, however, localized or generalized periodontal disease is present, conditions favorable for healing must be created, and concomitant periodontal therapeutic procedures can be

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implemented to restore the health of the periodontium.[32] The final restoration of root-resected teeth depends significantly on the nature of the resection, the amount of remaining tooth structure, the periodontal status, and the patient's occlusion. The prosthetic aspects of tooth restoration must be carefully assessed and integrated into the anticipated surgical procedure to ensure proper positioning of tooth margins relative to the osseous crest and also to manage the anticipated changes in occlusal relationships and masticatory forces.[86] Hemisection is the surgical separation of a multirooted tooth, usually a mandibular molar with severe furcation involvement, through the furcation in such a way that a root and associated portion of the crown may be removed or retained. A fixed partial denture often is placed in the area to restore the lost portion of the occlusal table (Fig. 25.14).

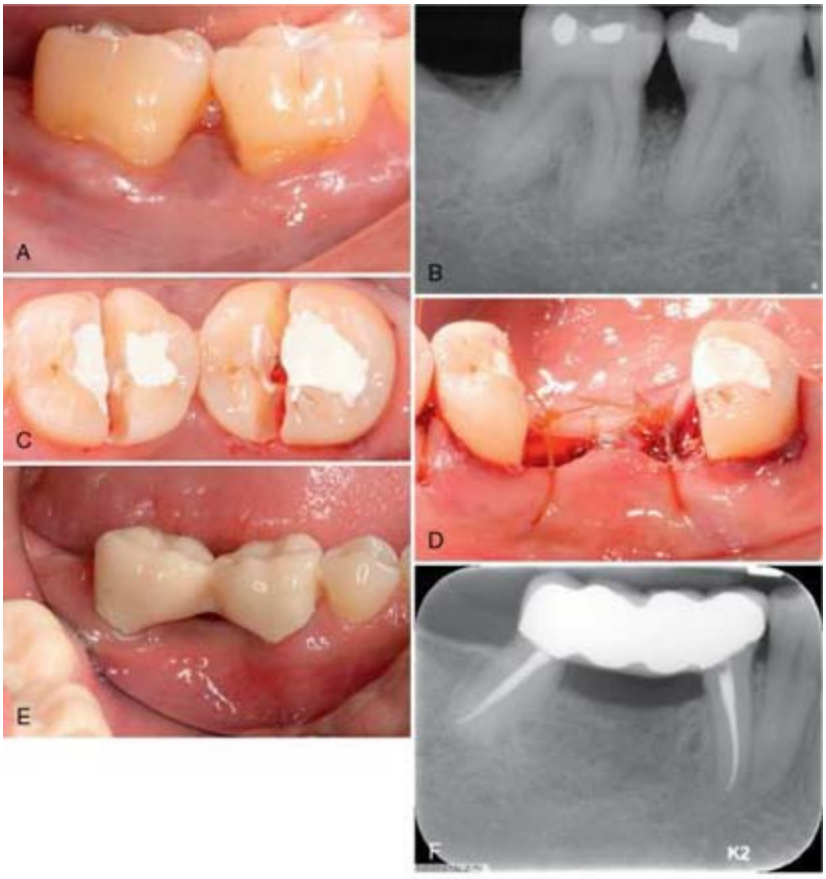

FIG. 25.14 Hemisection. **A,** Initial presentation of mandibular molars with severe attachment loss on the mesial aspect of tooth #31 and the distal aspect of tooth #30. **B,** Radiograph exhibiting interproximal periodontal defects. **C,** Occlusal view of hemisection after endodontic

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treatment. **D,** Suturing after removal of mesial half of tooth #31 and distal half of tooth #30. **E,** Eighteen-month follow-up with fixed restoration in place. **F,** Postoperative radiograph at 18 months showing normal bony and periodontal architecture.

Set of photographs and radiographs marked A through F shows two multi-rooted teeth, followed by the removal of their half parts, resulting in single rooted teeth with fused crowns.

Controversy has also existed regarding the benefits of and need for endodontic therapy before root resection. Instances develop in which exploratory surgery is necessary; should the periodontal problem be more extensive than determined presurgically, removal of a root should be carried out at that time. In these instances, removal of the involved root without endodontic treatment would be acceptable, but root canal therapy should be performed as soon as possible after root removal.[30,78,80] After resection of a vital root, the pulpal opening in the crown may be sealed and restored with a permanent amalgam restoration or sedative base material (e.g., Dycal) as a temporary solution. Investigators evaluated vital-root resection in maxillary molar teeth for 9 years.[28] Amputated pulps were covered with Dycal base and amalgam. At 1 year, 38% of molar teeth remained vital, but at 5 years only 13% maintained vitality. These findings imply that the long-term prognosis for vital-root resection is poor, so *endodontic therapy should be done before or immediately after resection*. However, Haskell[36] reported that vitality of resected teeth can be maintained even after 16 years. Nonetheless, it is generally agreed that whenever possible, endodontics should be performed in advance (before root resection). If this is not possible, the endodontic treatment should be performed as soon as possible after vital-root amputation. Otherwise, pulpal complications may occur, such as internal resorption, pulpal inflammation, and necrosis.[2,79]

The concepts of guided tissue regeneration (GTR) and guided bone regeneration (GBR) have been used to promote bone healing after endodontic surgery.[29,64] Fig. 25.15 illustrates an endodontic defect successfully treated with the GTR approach. Theoretically, the GTR barrier prevents contact of connective tissue with the osseous walls of the defect, protecting the underlying blood clot and stabilizing the wound.[35] An investigator treated large periradicular lesions with GTR barrier membranes and demonstrated

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that periradicular healing occurred more rapidly at membrane sites than at control sites.[63,64] The quality and quantity of the regenerated bone were superior with adjunctive use of the membrane than without such use. Similar findings were published in a case report with histologic examination of biopsy material obtained at barrier removal.[66] In addition, during examination of clinical cases, the closer the lesion is to the gingival margin, the greater the fluid and bacterial contamination from the sulcus (and the greater the risk of mechanical trauma). When GTR is considered, the combined endodontic and periodontal lesion probably has the least favorable prognosis compared with cases having only a periodontal lesion.[63]

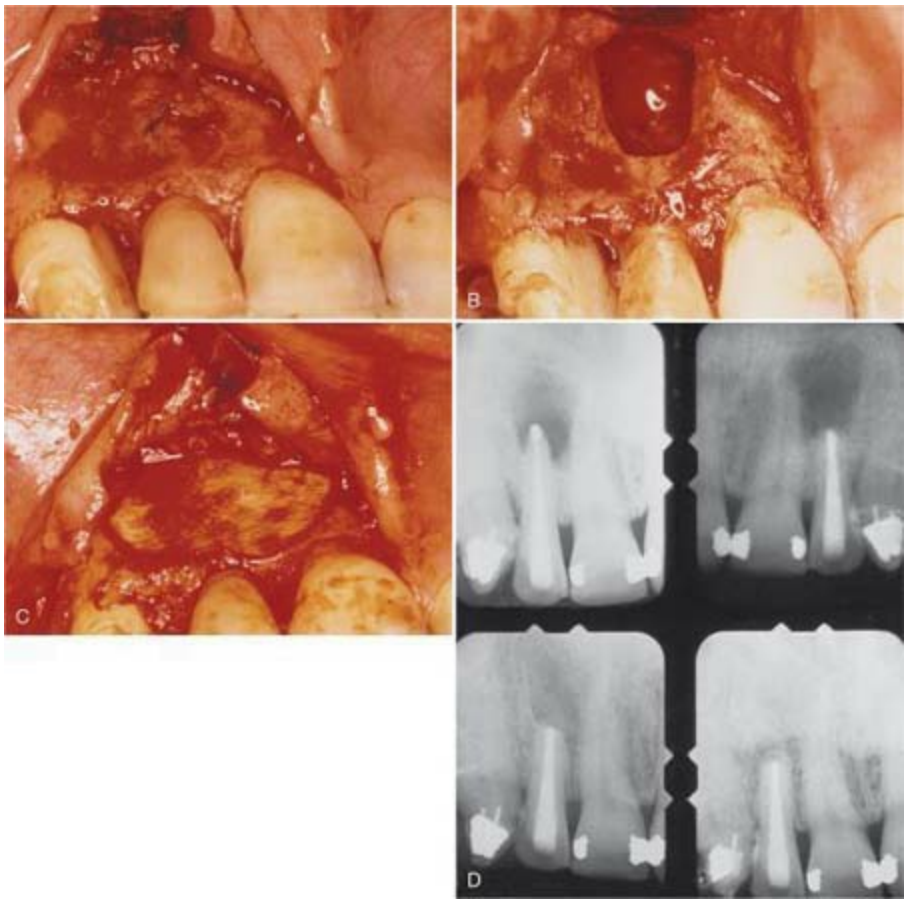

FIG. 25.15 Periradicular endodontic surgery using the combination of bone replacement grafts and collagen guided tissue regeneration barrier membranes. **A,** A buccal flap was reflected (note extensive apical lesion). **B,** An osseous window was created with a bur. **C,** The defect was repaired with demineralized freeze-dried bone allograft and covered with a collagen membrane. **D,** Radiographs show condition

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before surgery, at the time of surgery, and at 6 months and 2 years after surgery. At 2 years, the radiograph reveals complete bone regeneration.

Set of photographs and radiographs marked A through D shows the restoration of tissue damage at the root apex of tooth.

Many varieties of GTR membranes are available for clinical use, but it is logical to use bioresorbable collagen and polymer membranes in endodontic surgeries because there is often no need for a second surgery to retrieve the membrane. Studies reveal similar results achieved with nonresorbable and resorbable membranes.[18] Long-term studies need to be completed to evaluate the success of this approach.

For more than 40 years, bone grafts have been used to treat osseous defects associated with periodontal disease.[70,72] Because the "apicoectomy" (i.e., root-end resection) defect has a surrounding bony wall, the need for adjunctive bone grafting during this procedure is questionable (unless the lesion is exceptionally large in diameter). With the introduction of the GTR concept, a combination of bone replacement graft and GTR membrane (Fig. 25.16) has shown promising results.[1,22,42,66,83] Further studies are required to explore the true benefit of this combination treatment approach in root-end resection procedures.

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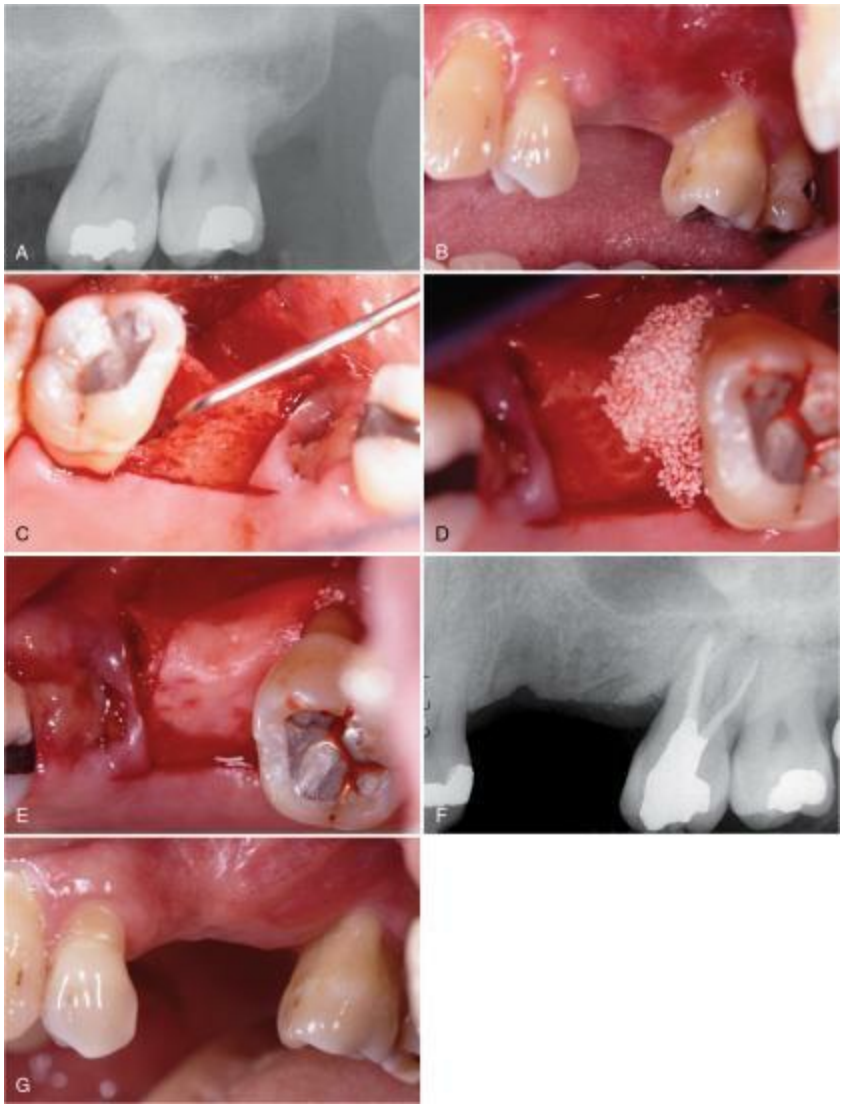

FIG. 25.16 Periodontal surgery using guided bone regeneration. **A,** A pretreatment radiograph demonstrates a significant osseous defect on the mesial aspect of tooth #14. **B,** Pretreatment clinical photograph of teeth #12 to #16. **C,** A full-thickness mucoperiosteal flap reflected with a periodontal probe 7 mm into osseous defect. **D,** Osseous defect filled with BioOss (cancellous bovine bone). Root canal treatment (RCT) was started for the patient before the surgical appointment; RCT was completed 6 weeks after osseous graft placement. **E,** Ossix membrane (collagen) placed over the BioOss graft. **F,** One-year posttreatment radiograph with evidence of osseous repair. **G,** One-year posttreatment clinical photograph of area.

Set of radiographs and photographs marked A through G shows the successful removal of gum pockets and damage tissue in the upper left quadrant.

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Forced eruption or extrusion

Teeth that have fractured, undergone extensive decay, or experienced internal or external root resorption or lateral perforation may be candidates for a forced eruption procedure, especially straight, tapering, single-rooted teeth (Fig. 25.17). To obtain good access for endodontic or restorative procedures, along with reduced probing depths, it may be necessary to perform extensive resection surgery to gain crown lengthening. Forced eruption may offer a better solution than surgical crown lengthening, which may produce poor aesthetics in some cases. A review of the literature reveals that most successfully treated cases are extrusions of less than 4 mm.[5,9,48] The infrequently used treatment method of forced eruption serves as an alternative to the sacrifice of the natural root system. Forced eruption can preserve the natural root system and related periodontal architecture, resulting in years of additional service for the patient. It also can maintain adjacent tooth structure while retaining the option for future implant reconstruction, if needed. Given the reported success of forced eruption, the technique should be used more often by dentists.[23,26] The increased use of implants in clinical treatments has stimulated clinicians' interest in augmenting bone in patients who have deficient alveolar ridges that might preclude ideal implant placement.

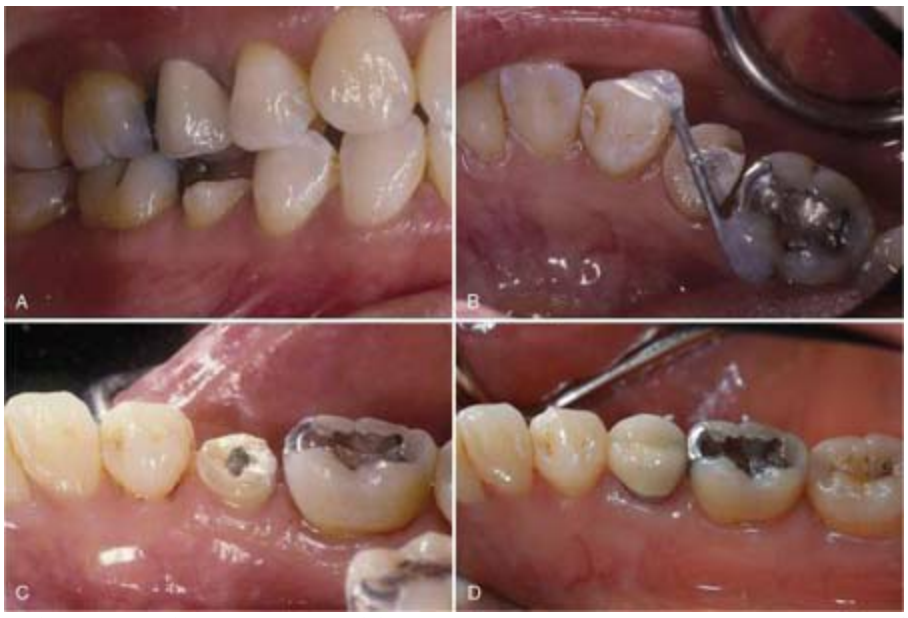

FIG. 25.17 Clinical case of forced eruption. **A,** Initial presentation of tooth #29 (buccal view). **B,** Bonded wire to help extrude tooth #29. **C,**

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Lingual view of tooth #29 after 3 weeks of extrusion. **D,** Lingual view of cemented porcelain crown on tooth #29; 4 months after extrusion and after 1 month of crown retention.

Four close-up views of interior of mouth marked A through D shows the restoration of tooth 29 affected with fractured crown and black residues in the cavity.

A nonsurgical technique for using the available bone for implant site development and fixture placement is orthodontic extrusion or forced eruption. The concept of extruding a tooth coronally by orthodontic forces and the clinical alterations in the soft tissue architecture of the periodontium demonstrated during orthodontic extrusive movement of periodontally compromised teeth have demonstrated probing depth reduction in some cases.[57] An investigator reported that tooth movement in the presence of inflammation might cause deepening of osseous defects. Often, extrusions are accompanied by an immature-appearing tissue that is red compared with adjacent tissue. This results from the eversion of junctional epithelium, which is nonkeratinized in the sulcus; the vasculature is more readily visible until the newly exposed tissue has a chance to keratinize, in about 28 days.[39,40] Most extruded teeth should be retained in place twice as long as it took to force-erupt them to minimize relapse before attempting definitive restorative procedures.

Summary

Endodontic and periodontal lesions result from the close interrelationship of pulp tissue and the periodontium. The major pathways of communication between the two types of tissue are the apical foramina, lateral and accessory canals, and dentinal tubules. The differential diagnosis of endodontic and periodontal lesions is not always straightforward and requires clinical data accumulation from a number of diagnostic tests to obtain a correct diagnosis. When examining and treating the combined or individual lesion in endodontics and periodontics, the clinician must bear in mind that successful treatment depends on a correct diagnosis. Lesions with combined causes require both endodontic and periodontal therapy, and endodontic therapy should usually be completed first. Root resection and regenerative techniques

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offer alternative approaches, enhancing the clinician’s ability to deal with these complex clinical problems.

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References

1. Abramowitz PN, Rankow H, Trope M. *Multidisciplinary approach to apical surgery in conjunction with the loss of buccal cortical plate*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1994;77: 502.
2. Allen AL, Gutmann JL. *Internal root resorption after vital root resection*, J Endod 1977;3: 438.
3. Altini M, Shear M. *The lateral periodontal cyst: an update*, Oral Pathol Med 1992;21: 245.
4. Basten CH-J, Ammons WF Jr, Persson R. *Long-term evaluation of root resected molars: a retrospective study*, Int J Periodontics Restorative Dent 1996;16: 206.
5. Batenhorst K, Bowers G. *Tissue changes resulting from facial tipping and extrusion of incisors in monkeys*, J Periodontol 1974;45: 660.
6. Belk CE, Gutmann JL. *Perspectives, controversies, and directives on pulpal-periodontal relationship*, J Can Dent Assoc 1990;56: 1013.
7. Bender IB, Seltzer S. *The effect of periodontal disease on the pulp*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1972;33: 458.
8. Bergenholtz G, Lindhe J. *Effect of experimentally induced marginal periodontitis and periodontal scaling on the dental pulp*, J Clin Periodontol 1978;5: 59.
9. Berglundh T, Marinello C. *Periodontal tissue reactions to orthodontic extrusion: an experimental study in the dog*, J Clin Periodontol 1991;18: 330.
10. Blomlof L, Jansson L, Applegren R, et al: *Prognosis and mortality of root-resected molars*, Int J Periodontics Restorative Dent 1997;17: 190.

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www.konkur.in

11. Blomlof L, Lengheden A, Linskog S. *Endodontic infection and calcium hydroxide treatment effects on periodontal healing in mature and immature replanted monkey teeth*, J Clin Periodontol 1992;29: 652.
12. Buhler H. *Evaluation of root resected teeth: results after 10 years*, J Periodontol 1988;59: 805.
13. Burch JG, Hulen S. *A study of the presence of accessory foramina and the topography of molar furcations*, Oral Surg Oral Med Oral Pathol 1974;38: 451.
14. Camargo PM, Pirih FQ, Wolinsky LE, et al: *Clinical repair of an osseous defect associated with a cemental tear: a case report*, Int J Periodontics Restorative Dent 2003;23: 79.
15. Carnevale G, DiFebo G, Tonelli MP, et al: *Retrospective analysis of the periodontal-prosthetic treatment of molars and interradicular lesions*, Int J Periodontics Restorative Dent 1991;11: 189.
16. Caton J, Armitage G, Berglundh T, et al: *A new classification scheme for periodontal and peri-implant disease and conditions – Introduction and key changes from the 1999 classifications*, J Periodontol Suppl 1, 2018;89: S1- S8.
17. Chen SY, Wang HL, Glickman GN. *The influence of endodontic treatment upon periodontal wound healing*, J Clin Periodontol 1997;24: 449.
18. Christgau M, Schmalz G, Reich E, et al: *Clinical and radiographical split-mouth study on resorbable versus non-resorbable GTR-membranes*, J Clin Periodontol 1995;22: 306.
19. Cutright DE, Bhaskar SN. *Pulpal vasculature as demonstrated by a new method*, Oral Surg Oral Med Oral Pathol 1969;27: 678.

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www.konkur.in

20. Czarnecki RT, Schilder H. *A histological evaluation of human pulp in teeth with varying degrees of periodontal disease*, J Endod 1979;5: 242.
21. Diem CR, Bower GM, Ferrigno PD, et al: *Regeneration of the attachment apparatus on pulpless teeth denuded of cementum in the rhesus monkey*, J Periodontol 1974;45: 18.
22. Duggins I, Clay J, Himel V, et al: *A combined endodontic retrofill and periodontal guided tissue regeneration for the repair of molar endodontic furcation perforations: report of a case*, Quintessence Int 1994;25: 109.
23. Durham TM, Goddard T, Morrison S. *Rapid forced eruption: a case report and review of forced eruption techniques*, Gen Dent 2004;52: 167.
24. Ehnevid H, Jansson L, Lindskog S, et al: *Endodontic pathogens: propagation of infection through patent dentinal tubules in traumatized monkey teeth*, Endod Dent Traumatol 1995;11: 229.
25. Erpenstein H. *A three year study of hemisectioned molars*, J Clin Periodontol 1983;10: 1.
26. Esposito S. *Rapid forced eruption: a case report and review of forced eruption techniques*, Gen Dent 2003;51: 58.
27. Everett FG, Kramer GM. *The disto-lingual groove in the maxillary lateral incisor: a periodontal hazard*, J Periodontol 1972;443: 352.
28. Filipowicz F, Umstott P, England M. *Vital root resection in maxillary molar teeth: a longitudinal study*, J Endod 1984;10: 264.
29. Garrett S. *Periodontal regeneration around natural teeth*, Ann Periodontol 1996;1: 621.
30. Gerstein K. *The role of vital root resection in*

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www.konkur.in

periodontics, J Periodontol 1977;48: 478.
31. Gher ME, Dunlap RM, Anderson MH, et al: *Clinical survey of fractured teeth*, J Am Dent Assoc 1987;114: 174.
32. Green EN. *Hemisection and root amputation*, J Am Dent Assoc 1986;112: 511.
33. Guldener PH. *The relationship between periodontal and pulpal disease*, Int Endod J 1985;18: 41.
34. Gutmann JL. *Prevalence, location and patency of accessory canals in the furcation region of permanent molars*, J Periodontol 1978;49: 21.
35. Haney JM, Nilveus RE, McMillan PJ, et al: *Periodontal repair in dogs: expanded polytetrafluoroethylene barrier membranes support wound stabilization and enhance bone regeneration*, J Periodontol 1993;64: 883.
36. Haskell EW. *Vital root resection: a case report of long-term follow-up*, Int J Periodontics Restorative Dent 1984;4: 56.
37. Herrera D, Retamal-Valdes B, Alonso B, et al: *Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions*, J Periodontol Suppl 1, 2018;89: S85- S102.
38. Hou GL, Tsai C. *Relationship between periodontal furcation involvement and molar cervical enamel projections*, J Periodontol 1987;58: 715.
39. Ingber JS. *Forced eruption. I. A method of treating isolated one and two wall infrabony osseous defects: rationale and case report*, J Periodontol 1974;45: 199.
40. Ingber JS. *Forced eruption: alteration of soft tissue cosmetic deformities*, Int J Periodontics Restorative Dent 1989;9: 416.
41. Jansson LE, Ehnevid H. *The influence of endodontic infection on periodontal status in mandibular molars*, J

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www.konkur.in

Periodontol 1998;69: 1392.
42. Kellert M, Chalfin H, Solomon C. *Guided tissue regeneration: an adjunct to endodontic surgery*, J Am Dent Assoc 1994;125: 1229.
43. Kerezoudis NP, Sisko GJ, Tsatsas V. *Bilateral buccal radicular groove in maxillary incisors: case report*, Int Endod J 2003;36: 898.
44. Kirchoff DA, Gerstein H. *Presurgical occlusal contouring for root amputation procedures*, Oral Surg 1969;27: 379.
45. Kirkham DB. *The location and incidence of accessory pulpal canals in periodontal pockets*, J Am Dent Assoc 1975;91: 353.
46. Klavan B. *Clinical observation following root amputation in maxillary molar teeth*, J Periodontol 1975;46: 1.
47. Koenigs JF, Brilliant JD, Foreman DW. *Preliminary scanning electron microscope investigations of accessory foramina in the furcation areas of human molar teeth*, Oral Surg Oral Med Oral Pathol 1974;38: 773.
48. Kozlovsky A, Tal H. *Forced eruption combined with gingival fiberotomy: a technique for clinical crown lengthening*, J Clin Periodontol 1988;15: 534.
49. Kramer IR. *The vascular architecture of the human dental pulp*, Arch Oral Biol 1960;2: 177.
50. Langeland K, Rodrigues H, Dowden W. *Periodontal disease, bacteria and pulpal histopathology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1974;37: 257.
51. Lantelme RL, Handelman SL, Herbison RJ. *Dentin formation in periodontally diseased teeth*, J Dent Res 1976;55: 48.
52. Lee KW, Lee EC, Poon KY. *Palato-gingival grooves in maxillary incisors*, Br Dent J 1968;124: 14.
53. Leknes KN, Lie T, Selvig KA. *Cemental tear: a risk*

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factor in periodontal attachment loss, J Periodontol 1996;67: 583.
54. Lin HJ, Chan CP, Yang CY, et al: *Cemental tear: clinical characteristics and its predisposing factors*, J Endod 2011;37: 611.
55. Lin HJ, Chang SH, Chang MC, et al: *Clinical fracture site, morphologic and histopathologic characteristics of cemental tear: role in endodontic lesions*, J Endod 2012;38: 1058.
56. Lowman JV, Burke RS, Pelleu GB. *Patent accessory canals: incidence in molar furcation region*, Oral Surg Oral Med Oral Pathol 1973;36: 580.
57. Mantzikos T, Shamus I. *Forced eruption and implant site development: soft tissue response*, Am J Orthod Dentofacial Orthop 1997;112: 596.
58. Masters DH, Hoskins SW. *Projection of cervical enamel into molar furcations*, J Periodontol 1964;35: 49.
59. Mazur B, Massler M. *Influence of periodontal disease on the dental pulp*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1964;17: 592.
60. Miyashita H, Bergenholtz G, Grondahl K, et al: *Impact of endodontic conditions on marginal bone loss*, J Periodontol 1998;69: 158.
61. Obermayr G, Walton RE, Leary JM, et al: *Vertical root fracture and relative deformation during obturation and post cementation*, J Prosthet Dent 1991;66: 181.
62. Papapanou PN, Sanz M, Buduneli N, et al: *Periodontitis: Consensus report of workgroup 2 of the 2017 world workshop on the classification of periodontal and peri-implant diseases and conditions*, J Periodontol Suppl 1, 2018;89: S173- S182.
63. Pecora G, Baek SH, Rethnam S, et al: *Barrier membrane*

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techniques in endodontic microsurgery, Dent Clin North Am 1997;41: 585.

64. Pecora G, Kim S, Celleti R, et al: *The guided tissue regeneration principle in endodontic surgery: one year postoperative results of large periapical lesions*, Int Endod J 1995;28: 41.
65. Perlmutter S, Tagger M, Tagger E, et al: *Effect of the endodontic status of the tooth on experimental periodontal reattachment in baboons: a preliminary investigation*, Oral Surg Oral Med Oral Pathol 1987;63: 232.
66. Pinto VS, Zuolo ML, Mellonig JT. *Guided bone regeneration in the treatment of a large periapical lesion: a case report*, Pract Periodontics Aesthet Dent 1995;7: 76.
67. Pitts DL, Natkin E. *Diagnosis and treatment of vertical root fractures*, J Endod 1983;9: 338.
68. Polson AM. *Periodontal destruction associated with vertical root fracture: report of four cases*, J Periodontol 1977;48: 27.
69. Rubach WC, Mitchell DF. *Periodontal disease, accessory canals and pulp pathosis*, J Periodontol 1965;36: 34.
70. Sanders JJ, Sepe WW, Bowers GM, et al: *Clinical evaluation of freeze-dried bone allograft in periodontal osseous defects. III. Composite freeze-dried bone allografts with and without autogenous bone grafts*, J Periodontol 1983;54: 1.
71. Saunders RL. *X-ray microscopy of the periodontal and dental pulp vessels in the monkey and in man*, Oral Surg Oral Med Oral Pathol 1966;22: 503.
72. Schallhorn RG. *Long-term evaluation of osseous grafts in periodontal therapy*, Int Dent J 1980;30: 101.
73. Seltzer S, Bender IB, Nazimov H, et al: *Pulpitis induced interradicular periodontal change in experimental*

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animals, J Periodontol 1967;38: 124.
74. Seltzer S, Bender IB, Ziontz M. *The interrelationship of pulp and periodontal disease*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1963;16: 1474.
75. Silverstein L, Shatz PC, Amato AL, et al: *A guide to diagnosing and treating endodontic and periodontal lesions*, Dent Today 1998;17: 112.
76. Simon JHS, Glick DH, Frank AL. *The relationship of endodontic-periodontic lesions*, J Periodontol 1972;43: 202.
77. Simring M, Goldberg M. *The pulpal pocket approach: retrograde periodontitis*, J Periodontol 1964;35: 22.
78. Smukler H, Tagger M. *Vital root amputation: a clinical and histologic study*, J Periodontol 1976;47: 324.
79. Stallard RE. *Periodontic-endodontic relationships*, Oral Surg Oral Med Oral Pathol 1972;34: 314.
80. Tagger M, Perlmutter S, Tagger E, et al: *Histological study of untreated pulps in hemisected teeth in baboons*, J Endod 1988;14: 288.
81. Tonetti MS, Greenwell H, Kornman KS., Staging and grading of periodontitis. *Framework and proposal of a new classification and case definition*, J Periodontol Suppl 1, 2018;89: S159- S172.
82. Torabinejad M, Kiger RD. *A histologic evaluation of dental pulp tissue of a patient with periodontal disease*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1985;59: 198.
83. Tseng CC, Chen YH, Huang CC, et al: *Correction of a large periradicular lesion and mucosal defect using combined endodontic and periodontal therapy: a case report*, Int J Periodontics Restorative Dent 1995;15: 377.
84. Vertucci FJ, Williams RG. *Furcation canals in the human*

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mandibular first molar, Oral Surg Oral Med Oral Pathol 1974;38: 308.

85. Walton RE, Michelich RJ, Smith GN. *The histopathogenesis of vertical root fractures*, J Endod 1984;10: 48.
86. Ward HE. *Preparation of furcally involved teeth*, J Prosthet Dent 1982;48: 261.
87. Whyman RA. *Endodontic-periodontic lesion. I. Prevalence, etiology, and diagnosis*, N Z Dent J 1988;84: 74.
88. Withers J, Brunsvold M, Killoy W, et al: *The relationship of palato-gingival grooves to localized periodontal disease*, J Periodontol 1981;52: 41.

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PART IV
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OUTLINE

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26: Bleaching procedures

Frank Setzer

CHAPTER OUTLINE

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Beauty and perfect appearance have a great value in society. The “perfect smile” is important to many people,[185] including straight and light-colored teeth. Tooth whitening has become an established practice, both in the dental office by providing nonvital and vital in-office bleaching procedures as well as by commercial companies that sell over-the-counter tooth “whitening” products. Vital tooth bleaching aims to lighten entire arches. Nonvital bleaching procedures are most often used to treat individual discolored teeth

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so that they blend in with adjacent teeth.

Etiology of tooth discolorations

A great variety of etiologies may be responsible for tooth discolorations. Discoloration was described as "any change in the hue, color, or translucency of a tooth due to any cause."[196] Great differences exist in severity, localization, and appearance. Intrinsic discolorations are incorporated into the tooth structure. Extrinsic discolorations are attached to the tooth surface. Both intrinsic and extrinsic discolorations can exist in combination and may affect enamel, dentin, or pulp. Some tooth discolorations are caused by hereditary causes, while others are related to patient behavior, age, and disease, or are created iatrogenically by dental or medical treatments (Table 26.1).

Table 26.1

Overview of Intrinsic and Extrinsic Discolorations

Color of Stain	Extrinsic	Intrinsic (Single Tooth)	Intrinsic (Multiple Teeth)
Black	Betel nut Chromogenic bacteria Coffee Dental plaque Metals Tea Tobacco Wine	Amalgam restorations Glass ionomer restorations Pulpal trauma with hemorrhage Minocycline (revascularization procedures) Metals (silver, gold, alloys)	Minocycline (oral) Tetracycline (oral)
Gray		Metals (silver, gold, alloys) Silicate compounds (e.g., MTA)	Dentinogenesis imperfecta Thalassemia

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Brown/reddish	Coffee Dental plaque Doxycycline Drugs (e.g., khat, marihuana) Iodine Metals/halides Oral rinses (e.g., chlorhexidine) Other foods Tea Tobacco Wine	Caries (active, arrested) Cervical root resorption Composite resin, glass ionomer restorations Dental trauma Endodontic irrigation solutions Iodine Minocycline (revascularization procedures) Pulpal trauma with hemorrhage Resorcinol-formaldehyde resin	Amelogenesis imperfecta Congenital erythropoietic porphyria Dental trauma Dentinogenesis imperfecta Fluorosis Infection during enamel formation Tetracycline (oral) Thalassemia
Green	Chromogenic bacteria Metals Tea	—	Hyperbilirubinemia Thalassemia
Orange	Chromogenic bacteria Metals Doxycycline	—	—
Yellow	—	Caries (active) Composite resin, glass ionomer restorations Dental trauma	Aging Amelogenesis imperfecta Dentinogenesis imperfecta Dental traumaDentinal dysplasia Fluorosis Hemolytic diseases Hemoglobin, heme hyperbilirubinemia Nutrition deficiency (e.g., rickets, scurvy)
White	—	Caries (incipient) Dental trauma	Calcification due to trauma Fluorosis Infection during enamel formation Nutrition deficiency (e.g., rickets, scurvy)

MTA, Mineral trioxide aggregate.

Systemic intrinsic causes

Genetic causes

Amelogenesis imperfecta (AI) describes a group of hereditary conditions associated with mutations in five genes—*AMEL* (amelogenin), *ENAM* (enamelin), *MMP20* (matrix metalloproteinaise-20), *KLK4* (kallikrein-4), and *FAM83H*—affecting the structure and appearance of enamel in the primary and secondary dentition.[77] Enamelin defects may cause variable hypoplasia, ranging from local pitting to generalized thinning of the enamel. Amelogenin defects can lead to abnormal maturation and mineralization defects; disorganized, hypoplastic enamel; or other phenotypic variations ranging

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from hypoplasia to hypomaturation or hypomineralization.[77] Based on this clinical appearance, this has led to the classification of four subtypes of AI[70]: AI type 1, hypoplastic enamel, which presents very thin, yet hard and translucent with normal radiographic appearance[251]; AI type 2, hypomaturation, softer than normal enamel, which is prone to rapid wear, and a radiographic density comparable to dentin; AI type 2, hypocalcified enamel of normal thickness, but extremely brittle, very susceptible to staining and rapid wear, and less radioopaque than dentin; and AI type 4, hypomature and hypoplastic enamel associated with taurodontism. The impaired mineralization due to the developmental changes in the enamel matrix formation may allow discoloring agents from the oral cavity easy access to the mineral structure. The tooth color may be brown or yellow. Bleaching attempts may be, although successful, of a temporary nature and often seen as an adjunct to comprehensive restorative treatment. Resin infiltration has been proposed as an alternative treatment for the white opacities present in the hypomature enamel of patients with type 2 AI.[39] Besides discolorations, the clinical appearance and morphologic structure of the enamel may result as well in increased sensitivity and fragility.

Dentinogenesis imperfecta (DGI) and dentin dysplasia (DD) are a group of hereditary autosomal dominant genetic dentin disorders characterized by an abnormal dentin structure.[19] Either the primary dentition solitarily or both primary and secondary dentitions may be affected.[19] DGI is most commonly categorized into three subtypes: DGI type I, DGI type II, and DGI type III; DD is categorized into two subtypes: DD type I and DD type II. DGI type I is associated with osteogenesis imperfecta and involves mutations in the collagen type 1 encoding genes COL1A1 and COL1A2. With the exception of DD type I, all other forms of DGI and DD are related to mutations in the gene encoding dentin sialophosphoprotein (DSPP).[19] All patients affected by osteogenesis imperfecta also suffer from brittle bones. Patients with DGI have weaker teeth that are more prone to fracture. Patients suffering from DGI type I, DGI type II, and DGI type III have teeth with yellow-brown, deep amber, or blue-gray discolorations and higher translucency. Bleaching treatment will have minimal to no effect; comprehensive restorative treatment is indicated, but it is difficult to execute due to the compromised tooth structure.

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Other hereditary diseases causing tooth discoloration include porphyria, erythroblastosis fetalis or thalassemia, and sickle cell anemia. Congenital erythropoietic porphyria is a disease resulting from abnormalities in the synthesis of porphyrins, organic compounds essential for the function of hemoglobin. It may result in reddish or brownish tooth discoloration. Erythroblastosis fetalis is a hemolytic disease wherein fetal erythrocytes in Rh-positive fetuses or newborns of Rh-negative mothers are destroyed by antigens that cross the placenta, leading to hyperbilirubinemia. Bilirubin, a yellow breakdown product of hemoglobin, is incorporated into developing teeth, creating yellow-green and blue-green discolorations. Thalassemia, a blood disease causing characteristic malformations of the skull and long bones, and sickle cell anemia, a hereditary blood disorder displaying an abnormality of the oxygen-carrying hemoglobin molecule causing red blood cell malformations, may result in intrinsic blue, green, or brown discolorations.

Disease-related causes

High fevers experienced during the ages of tooth development may cause chronologic enamel hypoplasia leading to banding type discolorations on the tooth surface. Vitamin and mineral deficiencies may induce hypoplasia. Rickets, a vitamin D deficiency, may lead to white hypoplasias resulting from osteomalacia, developmental anomalies in the bones. Scurvy, a vitamin C deficiency in conjunction with vitamin A deficiency, and disturbances of phosphorus uptake can cause enamel hypoplasias.

Metabolic causes

Enamel hypoplasia may also occur after excessive exposure to fluoride during tooth formation. Fluoride exposure can lead to alterations in the mineralized tooth structure, especially in the enamel matrix, giving the appearance of a mottled tooth. The extent of hypoplasia directly correlates with the fluoride uptake during tooth formation and will impact the degree of discoloration.[46] Shades of discoloration may vary greatly and can range from white spots with opaque or chalky appearances to yellow or brown stains on the facial side of the affected tooth, in severe cases with surface pitting of the enamel. No damage to the shape of the crown can be found. It can be related

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to AI or hereditary hypophosphatemia. Tumors, infections, or trauma may be contributing factors. In situations of moderate severity, external bleaching may be sufficient, whereas in severe cases, a restorative approach may be indicated.

Drug-related causes

Tetracycline is an antibiotic used to treat diseases such as urinary tract infections, chlamydia, acne, and cystic fibrosis. It was frequently used from the 1950s through the early 1980s. In particular in young children, it causes gray or brown, deep, dark stains that may affect the entire tooth or occur as a pattern of horizontal stripes. Many adults also currently suffer from tetracycline stains on their teeth. Tetracycline-induced discolorations in permanent teeth manifest during tooth development, when tetracycline becomes calcified in the tooth, generating the characteristic stains. The discolorations are embedded in enamel and dentin. Children are susceptible to tetracycline discolorations from the time they are in utero until the age of 8. Because tooth development starts prior to birth, women are advised to avoid taking tetracycline during pregnancy. Tetracycline stains are obvious and were viewed as always permanent in the past. However, several studies reported successful cases after application of a walking bleach technique[2,4,7,12,68,95,143,256] and even prolonged bleaching with carbamide peroxide.[30,125,137,151,174,228,253] The walking bleach technique will, however, require intentional devitalization and endodontic treatment of the tooth to allow for the application of the bleaching agent into the pulp chamber.[2,4] A patient must be thoroughly informed about the risks, advantages, and disadvantages of this treatment.[2,4] Today, “power bleaching” and veneers are among the suggested treatments for tetracycline stains. Nevertheless, great care must be taken to avoid issues with bonding systems after the application of bleaching agents. Severe cases of tetracycline discoloration may require full coverage restorations.

Local intrinsic causes

Pulpal hemorrhage

Dental trauma may lead to the rupture of intrapulpal blood vessels with

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subsequent intrapulpal hemorrhage and the release of blood components into the dentinal tubules.[13] The hemolysis of erythrocytes will result in the degradation of hemoglobin into globin and the heme protein, containing an iron atom.[162] The iron, in the form of iron sulfides, may reach dentinal tubules, causing brown/reddish or black stains and discolorations in the surrounding dentin. Depending on whether the pulp recovers or becomes necrotic, the discoloration may revert or persist. If the pulp survives, the tooth may eventually revert to its initial shade; however, in situations where pulp necrosis takes place, the discoloration may remain and become worse. Intracoronal bleaching has proven effective in these situations.[71,206,258]

Pulp necrosis

The dental pulp may be irritated chemically, mechanically, or by microbial insult, especially by bacteria and their toxic by-products. After initial inflammation, the pulp develops local microabscesses followed by tissue necrosis and eventually complete necrosis. Disintegration products from pulp necrosis may become incorporated into dentinal tubules and cause discoloration of the surrounding dentin. The intensity of the discoloration appears proportional to the time the discoloring agents remain in the pulp chamber. These types of discolorations tend to respond favorably to intracoronal bleaching.[196]

Pulp tissue remnants

Remnant pulp tissue that was left behind after endodontic therapy will eventually disintegrate and may discolor tooth structure. As a clinical consequence, pulp horns and all other pulp tissues in the access chambers should be carefully removed during treatment.

Restorative materials

Metallic filling materials, such as amalgam or gold, may cause discolorations. Gold fillings, including gold foil compaction fillings, inlays, or onlays, but also pins or posts, mostly cause color changes, reducing tooth translucency and adding dark hues through thin remaining tooth portions. Amalgam fillings, however, over time, will undergo corrosive changes and degradation, with by-products causing color alterations within the tooth structure.[175] For

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any metallic filling that remains visible through existing tooth structure, removal of the filling and exchange with an esthetic filling material is the preferred choice. Effectively, dentin discolorations by amalgam can be bleached but may be prone to recurrence. Composite resin restorations may leak at the margins and allow for discoloring agents to penetrate into the tooth and dentinal tubules.

Intracanal medicaments and root filling materials

A variety of endodontic medications and filling materials may be responsible for discolorations. Silver points, historically used for root fillings, can cause gray, dark discolorations in teeth and the surrounding tissues due to corrosive processes.[3,33] Gutta-percha was reported to cause a light pink discoloration.[182,250] Resorcinol-formaldehyde resin paste ("Russian Red") was reported to cause discolorations ranging from pink to dark burgundy.[163,212] Phenols or iodine-containing intracanal medications (e.g., camphorated monochlorophenol [CMCP] or iodine-potassium-iodine [IKI]) may reside in the root canal space and start gradual discolorations by penetration of dentinal tubules through oxidation.

Endodontic sealers, particularly those containing metallic compounds (e.g., silver), can cause dark discoloration, either if the material is incompletely removed from pulp chambers and access cavities or because some components interact with moisture from dentin over time (Fig. 26.1).[8,181,255] For example, AH26 (Dentsply De Trey, Konstanz, Germany), an epoxy resin cement, contains bismuth trioxide as a filler and radiopacifier. Within dentin, various bismuth compounds that range in color from green to black may occur and cause discoloration.[255] The corrosion of silver in the sealer may cause a gray to black discoloration.[8,56] Also, incomplete removal of AH26 can cause discoloration in reaction with intracanal medicaments.[232] Epiphany (SybronEndo, Orange, CA), an adhesive root canal cement, showed signs of tooth discoloration.[214]

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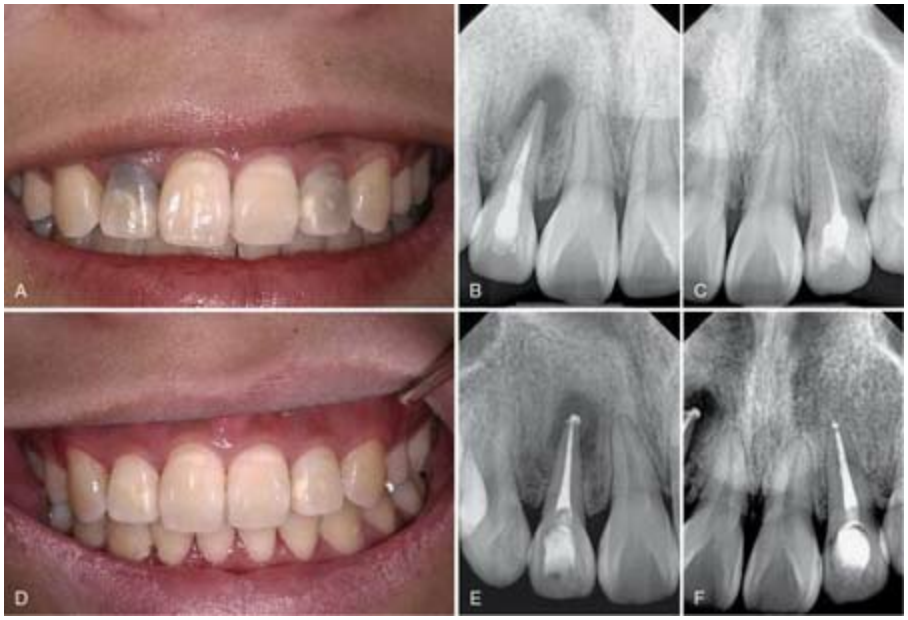

FIG. 26.1 Clinical case of a 19-year-old female patient illustrating discolorations from endodontic filling materials. **A,** Preoperative situation with history of endodontic treatment on both maxillary lateral incisors. **B and C,** Preoperative radiographs prior to nonsurgical retreatment and bleaching procedures. **D,** Postoperative situation after endodontic retreatment, bleaching procedures and new coronal restorations. Shades now match the maxillary arch with A2 (shade guide not shown). **E and F,** Postoperative radiographic control of procedures ca. 4 weeks after completion of endodontic treatment and new restorations.

A) Close-up view of upper teeth shows black spots on the lateral incisors.

B) Radiograph shows a dark patch at the root of lateral incisor.

C) Radiograph shows a high-density tissue in place of the dark patch.

D) Close-up view of teeth shows disappearance of black spots post procedure.

E) Radiograph shows the central tooth with filled root canal and a dark patch at the root tip. The neck region has radiolucent region. The adjacent tooth shows radiolucent root canal and central part of the crown.

F) Radiograph shows two teeth with radiolucent root canal and central part of the crown. The third tooth has a filled radiocanal.

Source: (Courtesy Dr. Melanie Martel, Philadelphia, PA.)

Mineral trioxide aggregate (MTA), a dental material known for excellent biocompatibility, is used for apexifications, perforation repair, root-end

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filling, and revascularization procedures. MTA may discolor teeth and adjacent soft tissues.[22,29,31,114,120,173,222,259] The discoloring effects were shown for both gray and white formulations.

Antibiotic pastes in various combinations are used to initiate revascularization of immature necrotic teeth. Tooth discoloration after regenerative endodontic therapy was extensively reviewed by Kahler and Rossi-Fedele in 2015.[119] In particular, the use of the triple antibiotic combination (clinically used as triple antibiotic paste [TAP]) of metronidazole, minocycline, and ciprofloxacin has produced cases of dark discolorations of hard tissue structure.[119,126] Minocycline, a tetracycline derivate, binds calcium ions of the root dentin and forms an insoluble complex. Discoloration by minocycline-containing pastes affects both teeth with closed apices as well as teeth with open apices.[130] While intracoronal bleaching with sodium perborate has demonstrated whitening after several weeks,[130] these effects cannot be guaranteed. Thus minocyline-containing pastes should be avoided for regenerative therapies if these are addressed from a patient-centered perspective.[119] Other medications shown to induce discolorations include corticosteroid preparations[127] or formocresol and iodoform-based medications.[91,139] Repeated applications of formocresol will penetrate dentin and cementum, particularly in the teeth of younger patients.

Discolorations may also occur due to irrigation solutions. Sodium hypochlorite can discolor dentin based on its destructive effect on erythrocytes and the ability to crystallize on the dentin surface.[91] In contact with chlorhexidine, sodium hypochlorite may form a dark brown precipitate containing para-chloroaniline, which can only be removed by mechanical action.[20,21,109,133,252] MTAD (a mixture of a tetracycline isomer, citric acid, and a detergent; Dentsply Tulsa Dental, Tulsa, OK) has been described as a cause for brown discoloration,[238] probably due to dentinal absorption and the release of doxycycline.

Dental caries

Progressing caries can cause tooth discoloration. Early stages of caries are characterized by white, opaque enamel lesions. If caries arrests, the lesion may darken by taking up pigments from exogenous sources, frequently

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rendering it a deep dark brown or black color.[231] Explanations for discoloration include the formation of melanin or lipofuscin; the sugar-protein reaction also known as the Maillard reaction or nonenzymatic browning[17]; melanin[132] or food dye[124] uptake into the carious defect, or compounds diffusing ahead of the bacterial penetration of demineralized dentin. Dietary chromogens entering the dentin is seen at least as a co-staining factor facilitated by the increased porosity of the hard tissue structures by the carious process. Silver compounds, such as silver diamine fluoride, used to arrest caries may also result in the dark gray or black discolorations typical for silver. These discolorations have been shown not to be very esthetically tolerable by parents if children's anterior teeth are being treated.[45] The use and effects of silver compounds utilized in dentistry for caries management was critically reviewed by Peng et al. in 2012.[188] The addition of the Glutathione Bio-Molecule to the silver diamine fluoride solution has demonstrated some effects in minimizing tooth discoloration in vitro.[210]

Calcific metamorphosis/dystrophic calcification

Calcific metamorphosis can be caused by trauma, resulting in obliteration of the pulp with mineralized tissue. Odontoblasts can become destroyed by the traumatic impact and replaced by cells from adult stem cell populations in the pulp. These cells may initiate rapid deposition of reparative dentin, resulting in yellowish or yellow-brownish discolorations. Anterior teeth are mostly affected. Reparative dentin may occupy the entire pulp chamber, and in certain situations also major parts of the root canal system, resulting in a loss of translucency of the crown. Depending on whether or not the remaining uncalcified portions of the pulp remain vital, endodontic therapy may be indicated, yet difficult to execute, depending on the extent of the calcification.[258] Dystrophic calcification involves the formation of foci of calcification frequently found in the aging pulp, usually in perivascular or perineural locations. Bleaching procedures are not expected to render positive results as the underlying color changes for calcific metamorphosis dystrophic calcification are primarily related to translucency and not actual discolorations. Veneers or full coronal coverage restorations may be indicated in severe situations.

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Root resorption

Cervical root resorptions can lead to pink discolorations of teeth. The granulation tissue of the resorptive defect may be visible through a thin dental enamel layer, giving the characteristic “pink spot” appearance. Treatment involves the removal if the granulation tissue, etching with trichloroacetic acid to remove tissue remnants and an adhesive restoration of the defect. The treatment may involve a surgical exposure of the resorption or, in severe cases, extraction of the tooth.

Aging

Aging-related tooth discolorations are the result of a physiologic process attributed to the uptake of discoloring agents into the hard tissue structure over time.[253] Age-related changes such as incisal wear, craze lines, or cracks allow for easier penetration of discoloring agents. Furthermore, over time, enamel becomes thinner, and the change in ratio between dentin and enamel structure results in additional optical darkening of the teeth. Extracoronal bleaching can partially whiten age-related stains.

Extrinsic causes

Extrinsic stains are food-, beverage-, or smoking-related superficial stains and discolorations. Pigments from beverages such as coffee or tea or tar from smoking may cause dark, brownish discolorations. Extensive consumption of oranges, carrots, licorice, or chocolate may lead to food-related stains. In areas with diverse populations, betel chewing and areca nut use may be prevalent. In particular, people with origins from Southeast and East Asia, India, Nepal, Bangladesh, Sri Lanka, Pakistan, and parts of China, Melanesia, Taiwan, and the Pacific Islands may be affected. Consistent chewing of betel nut and related substances may cause large-scale black discolorations. Discoloring agents are also included in marijuana. The effects of staining may be accelerated or enhanced if demineralization from acidic food or poor oral hygiene created rougher tooth surfaces. In general, extrinsic stains respond well to scaling, polishing, or bleaching.

Treatment planning for internal bleaching

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The etiology of tooth discolorations may vary greatly. Thus the outcome of bleaching procedures is very dependent on the etiology of the discoloration. Prior to any bleaching procedures, the situation must be carefully assessed and potential bleaching outcomes discussed with the patient, together with the patient's expectations.[85] If bleaching procedures are indicated, then they should be integrated into the overall treatment plan.[217] A referring dentist should be made part of the conversation, if applicable. To establish a correct diagnosis, the patient's dental and medical histories need to be reviewed, including family history, genetic or systemic disorders, past and current medications, as well as any history of dental or facial trauma or orthodontic, restorative, and endodontic treatment. Radiographic analysis, including periapical and bitewing radiographs (as well as, if applicable, cone-beam computed tomography [CBCT]) is indicated. The assessment of the pulpal and periapical status is a prerequisite. Particular to treatment planning bleaching procedures, the dentition must be evaluated for caries prevalence and type, plaque control, and tooth surface stains, as well as the type, quality, and extent of existing restorations. Transillumination may aid in finding dental decay or pulp chamber calcifications.

Contraindications of bleaching

Tooth bleaching is not indicated for every situation and every patient. A thorough investigation and diagnosis of the patient's history and clinical situation may bring to the surface issues with the underlying stain that imply that a good esthetic outcome is either impossible or difficult to achieve.[44,189] The patient's esthetic expectations should be critically evaluated and the prognosis for the treatment discussed thoroughly. This includes potentially unrealistic expectations by the patient, either due to the clinical facts or the patient's overexpectations. The clinicians should be aware of any psychological or emotional problems that a patient may have with the current clinical situation and should advise patients if a bleaching procedure is not in their best interest. All findings and discussions with the patient should be well documented for legal purposes and future reference.

The patient must be made aware of additional dental procedures, such as restorative, periodontal, endodontic, or orthodontic treatments that may be

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necessary to achieve a desired esthetic goal.[189,217] Bleaching of teeth with white[26,88] or brown spot[123] discolorations could increase the existing contrast.[225] Teeth with existing decay or discolored or leaking older restorations may require technique variations[61] or new restorative procedures to remove diseased hard tissue structures to match the adjacent tooth esthetically.

Patients with confirmed severe attrition, abrasion, abfractions or erosion, caries, or insufficient restorations may experience dentin hypersensitivity prior to a bleaching procedure. This sensitivity could be strongly increased by an in-office or at-home external bleaching procedure, particularly if patients can control the amount of bleaching agent used. Clinicians are advised to resolve the underlying issues prior to engaging in tooth whitening procedures. Patients with suspected or confirmed bulimia should not receive bleaching procedures. The underlying psychological condition must be resolved prior to treatment. Most patients suffering from bulimia require veneers or full coverage restorations.

If orthodontic treatment with fixed orthodontic appliances is scheduled, no bleaching procedures should take place 2 to 3 weeks prior to any adhesive placement of brackets.[225] The oxygen released from the peroxide compounds will interfere with the bonding strength of the composite resins to hard-tissue structure.[61,171]

Alternatives to bleaching

Microabrasion is an alternative to bleaching with peroxide compounds for superficial irregularities and shallow intrinsic stains in the enamel surface.[90] Briefly, the technique involves mixing 18% hydrochloric acid solution with fine pumice powder to form a thick paste and applying it to the stained tooth surface. A rotating rubber cup in a slow speed handpiece with light pressure is used to distribute the paste. Liquid or sheet rubber dam placement is mandatory. Prior to using the rubber cup, the paste should be placed on the tooth for about 5 seconds, followed by the rubber cup activation for 15 seconds. While the enamel is carefully monitored for any damages, this procedure is repeated for up to 12 to 15 times, depending on the effectiveness of the stain removal. If the stain is not fully removed after this many

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applications, the procedure should be terminated to avoid permanent enamel damage. Copious irrigation with water is used to remove excessive paste material. The application of topical fluoride for several minutes concludes the procedure.[192] Several case reports with successful long-term follow-up have been reported.[223]

Chemistry of bleaching

The most common classification for tooth discoloration was described by Nathoo[175] and includes three classes:

- Nathoo type 1 (N1): The discoloring agent (chromogen) binds to the tooth surface, with a color similar to that of dental stains caused by chromogenic bacteria, coffee, tea, wine, and metals.
- Nathoo type 2 (N2): The discoloring agent is a Nathoo type 1 food stain that changes color to darker after binding to the tooth surface for a certain amount of time.
- Nathoo type 3 (N3): A prechromogen or, in its base state, colorless material binds to the tooth and causes a stain after a chemical reaction. These types of stains are often due to carbohydrate-rich foods (e.g., apples, potatoes), stannous fluoride, or chlorhexidine.

Tooth bleaching involves the alteration of light-absorbing or light-reflecting properties of enamel and dentin stains, resulting in apparent whitening. In-office bleaching, internally and externally, has been practiced since the early 20th century employing peroxide compounds. At-home bleaching procedures were only introduced in the 1980s. Historically, a variety of bleaching materials has been tried, including substances such as oxalic acid or chlorine.[26,83,175] Requirements for active tooth bleaching ingredients include a quick reaction with the stain components, fewer reactions with oral hard and soft tissues, ease of use, and an acceptable shelf life. Alkaline bleaching solutions may cause less demineralization damage than acidic solutions and are less likely to penetrate the pulp.[75,76] Bleaching materials should cause as minimal oxidative damage to tissues as possible (Fig. 26.2).[69,200]

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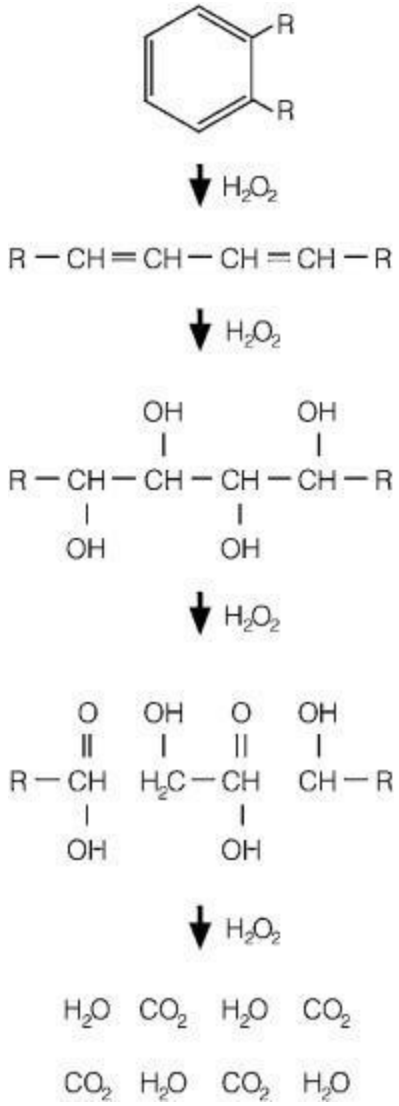

FIG. 26.2 Chemistry of tooth whitening with hydrogen peroxide. Breakdown of pigmented organic materials consisting of carbon ring structures with unsaturated, double carbon bonds to hydrophilic, nonpigmented carbon structures with saturated carbon bonds, resulting in effective whitening. Further degradation over this step may complete disintegration of the organic matrix with the release of carbon dioxide and water.

A benzene ring with two R groups at C1 and C2 in the presence of hydrogen peroxide leads to R single bonded to CH double bonded to CH single bonded to CH double bonded to CH single bonded to CH, which further in the presence of hydrogen peroxide leads to a compound that has a 4-carbon chain. Each carbon is single bonded to a hydroxyl group. The terminal carbon atoms are each single bonded to an R group. This compound further in the presence of hydrogen peroxide yields three compounds. The first compound has a central CH group double bonded to an oxygen atom, single bonded to a hydroxyl group and an R group. The second compound has a methylene group

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single bonded to a hydroxyl group and a CH group, which is double bonded to an oxygen atom and single bonded to a hydroxyl group. The third compound has a CH group single bonded to a hydroxyl group and an R group. Further, these compounds in the presence of hydrogen peroxide yield four water molecules and four carbon dioxide molecules each.

Source: (Modified from *Cohen's Pathways of the Pulp*, ed 8, Part III, Chapter 21, Fig. 21.1. Editors: Cohen S, Burns C, Mosby, St. Louis, 2002.)

Contemporary bleaching techniques rely on oxidative processes that actively break down large pigmenting molecules. Oxidation is initiated by active peroxide compounds.[84,96] Peroxides and oxygen radicals are reactive free agents that are formed in all body cells that use oxygen. Reactive oxygen is, however, usually only found in low concentrations. Natural antioxidants, such as vitamin E, prevent the accumulation.[40,154] Cell damage may occur if reactive oxygen reacts with iron or copper ions.[69]

The active peroxides utilized by contemporary bleaching techniques primarily derive from hydrogen peroxide and carbamide peroxide for external, vital bleaching and sodium peroxide for internal, nonvital bleaching. Hydrogen peroxide (H_2O_2) is the simplest peroxide. In its pure form, it is a colorless liquid of a slightly higher viscosity than water. It is suitable for in-office as well as at-home bleaching procedures. Temperature increase accelerates the speed of the bleaching reaction. Hydrogen peroxide is more effective as a bleaching agent at pH values that are close to the dissociation constant.[65] Enzymes such as peroxidases can break down hydrogen peroxide into oxygen and water. High concentrations of hydrogen peroxide must be handled with great care. If accidentally brought into contact with soft tissues, it is caustic and may cause chemical burns by free oxygen radicals. For external bleaching procedures, the use of rubber dam with additional block out isolation around the gingival margins is mandatory to avoid iatrogenic complications. Typically, at-home concentrations range from 3% to 7.5%; however, some exceptionally strong concentrations can reach 14%. In-office concentrations may range from 25% to 38%. Hydrogen peroxide was shown not to induce significant changes in the organic and inorganic relative contents of enamel, but to whiten teeth by oxidizing the organic matrix.[62]

Carbamide peroxide ($CH_6N_2O_3$), or urea hydrogen peroxide, will break

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down into carbamide and hydrogen peroxide in aqueous solution.[175] It is an efficient bleaching agent.[248] The carbamide portion urea is well tolerated by the human body.[202] Carbamide peroxide is available as crystallized powder or white crystals that contain H_2O_2 in a concentration of about 35%. Most common home bleaching products contain carbamide peroxide at about 10% strength, but it can reach up to 30% (equivalent to 3.5% to 8.6% hydrogen peroxide). Carbamide peroxide can be used for internal bleaching.[248] At 35%, it shows very low extraradicular diffusion rates compared to hydrogen peroxide and sodium perborate.[145,158] Common bleaching preparations may contain glycerin or propylene glycol, sodium stannate, phosphoric or citric acid, and flavor additives. Carbopol, a water-soluble polyacrylic acid polymer, may be added as a thickening agent. It prolongs the release of active peroxide. At room and oral temperatures, the reaction of carbamyl peroxide takes place at a slower rate compared to hydrogen peroxide.[98] The release of oxygen from hydrogen peroxide occurs within seconds of contacting tooth surfaces, whereas carbamide peroxide remains active for 40 to 90 minutes.[175]

Sodium perborate ($NaBO_3$) comes in different preparations, mostly as a stable, dry powder or as a gel. It is available as a monohydrate, trihydrate, and tetrahydrate, with varying contents of oxygen. Depending on the oxygen content of the preparation, the compounds have a different bleaching efficiacy.[260] The perborate ion comprises 95% of the molecule and provides close to 10% of the available oxygen. Acids, water, and warm air initiate its decomposition to sodium metaborate, hydrogen peroxide, and nascent oxygen. The pH of sodium perborate solutions used for dental bleaching is alkaline. Because sodium perborate can be more easily controlled, it is considered safer to work with than hydrogen peroxide and more popular for intracoronal bleaching.

In an aqueous solution, due to heat or in an acidic environment, both carbamide peroxide and sodium perborate decompose and release hydrogen peroxide. Hydrogen peroxide release from sodium perborate has been shown to reach a peak concentration within 27 hours of mixing it with distilled deionized water, reaching a plateau after about 3 days, with only low levels of hydrogen peroxide present up to 28 days.[241] Hydrogen peroxide then diffuses through enamel and dentin. Subsequently, the oxidative processes

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provide for lightening of stains in comparison to the surrounding tooth structure. Double bonds of complex molecular chains with high-molecular-weight pigments are broken down by the chemical reactions with the free radicals. As a consequence, the change in pigment configuration and size leads to a change in the wavelength of the reflected light and the change of the stain to a lighter color.[75,76,175] In an ideal scenario, this leads to an effective reduction or elimination of the discoloration. However, after the bleaching process, a shade rebound effect may be observed, resulting from a reformation of double bonds of the molecules.

Walking bleach technique

Early accounts of the walking bleach technique described it as leaving a mix of sodium perborate and distilled water in the pulp chamber for a period of several days, with the access cavity sealed with a temporary restoration.[208,220] Later, the use of 30% hydrogen peroxide instead of water was suggested to provide better bleaching effectiveness.[179] A mix of sodium perborate and water or, alternatively, anesthetic solution remains the most commonly used technique for internal bleaching of nonvital teeth. The enhancement of the mixture with 30% hydrogen peroxide is being used less due to concerns of cervical root resorptions but remains an option for stains resistant to whitening that require stronger chemical compounds to achieve good bleaching results.[16,27,170,180,213]

Walking bleach clinical protocol

1. Prior to initiating a bleaching procedure, a diagnosis must be made following thorough evaluation of the etiology and origin of the stain. The patient must be informed about the history, the procedures involved in the treatment, the expected outcome of the procedure, and a potential for rediscoloration.
2. The endodontic status of the tooth must be assessed clinically and radiographically. In case of clinical symptoms, the presence of periapical pathology, or an insufficient root filling, retreatment of the existing endodontic filling is advised, unless the apical periodontitis is on a tooth with a history of recent, well-executed root canal

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treatment or is verifiably in the process of healing.

3. Existing coronal restorations need be checked for adequate seal and shade and replaced if defective. In certain situations, the reason for discoloration may be a leaking restoration or discolored filling materials. Replacement of a defective restoration and cleaning of the access chamber may resolve the discoloration.
4. Clinical photographs should be taken at the beginning, throughout, and at the end of the treatment, with the tooth next to a shade guide to act as a reference for the practitioner and the patient.
5. Rubber dam isolation is mandatory. It is important to provide a seal at the gingival margin to prevent leakage of bleaching agents into the surrounding soft tissues. Depending on whether a single tooth or multiple teeth are being isolated, this may involve the placement of clamps, wedges, ligatures, floss, or light curing blockout material. Local or topical anesthesia may be applied if the patient is sensitive to the discomfort exerted during the isolation procedures.
6. The access cavity needs to be prepared so that all restorative filling materials, root-filling material and sealer remnants, and necrotic pulp tissues remnants are completely removed. This procedure requires good visualization through cavity refinement and the elimination of all undercuts, especially in the area of pulp horns. The practitioner should exercise care to avoid labial perforations.
7. Intracoronal materials should be removed to a level just below the labial gingival margin. Solvents such as xylene, eucalyptus oil, or orange solvent may be used to help clean the pulp chamber. Also, rinsing the cavity with sodium hypochlorite was suggested for cleanliness and disinfection or, alternatively, with alcohol to reduce surface tension.[16] Although some authors have recommended cavity treatment with 37% phosphoric acid or ethylenediaminetetraacetic acid (EDTA) to allow for deeper penetration of the bleaching agents, others have suggested that phosphoric acid was unnecessary and offered no better prognosis and no improvement in the bleaching effectiveness of either sodium perborate or a high concentration of hydrogen peroxide.[16,41] Acidic pretreatment may increase the risk adverse effects on the periodontium.[110]

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8. A protective layer of at least a 2-mm thickness should be placed over the root filling material. To avoid further pigmentation the protective layer should be white or tooth colored. Without this protective layer, bleaching materials may penetrate the root filling toward the apical foramen.[52,93,218] Materials that had been suggested include glass-ionomer cements, intermediate restorative material (IRM), Cavit and Coltosol, resin composites, photo-activated temporary resin materials such as Fermit, zinc oxide–eugenol cements, polycarboxylate cements, and zinc phosphate cements.[221] Cavit and IRM provide better sealing than zinc phosphate cement,[165] and Cavit offers a better leakage barrier than IRM.[93] Cavit and Coltosol also provided the most favorable seal when tightly compacted and proved to be superior to Fermit.[111] A protective barrier at the cementoenamel junction (CEJ) will furthermore reduce the risk of cervical root resorption and damage to the periodontal ligament.[221] Ideally, the vertical outline of the protective barrier should be 1 mm coronally to the CEJ to cover lingual, labial, and proximal CEJ areas.[207] Clinically, however, this may be difficult to achieve in confined spaces. A barrier at level with the labial CEJ will leave a substantial area of proximal dentinal tubules unprotected. Clinically, Cavit is also easily removable using ultrasonic instruments and chlorhexidine, thus minimizing the amount of tooth structure loss. Glass-ionomer cements may remain in place after the bleaching procedure is completed and do not need to be removed (Figs. 26.3 and 26.4).
9. Sodium perborate (tetrahydrate) is mixed with an inert liquid (distilled water, saline, or anesthetic solution) to achieve the consistency of wet sand. Mixing the paste instead with hydrogen peroxide is an option to accelerate the speed of the bleaching process, yet this technique is risky due to the increased possibility of cervical root resorptions. If it is required, due to the severity of the stain, then it should be used at 3% strength,[179] and it should be refrained from being increased to a 30% solution. The long-term results of a sodium perborate and water mix do not significantly differ from mixes with hydrogen peroxide.[108,202,206] A plastic instrument or amalgam carrier can be used to place the material in the pulp chamber. It is then

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carefully condensed with a plugger, without missing areas such as pulp horns (Fig. 26.5).

10. Excessive bleaching material should be removed, the paste carefully dapped with a cotton pellet without removing too much moisture, and a temporary filling material that is ideally 3 mm thick with well sealing margins placed (IRM, Cavit). Though some have advocated the use an acid-etched adhesive as a temporary filling to avoid recontamination, restaining, and to offer the best possible prevention from leaking walking bleach,[254] this may not be practical in small cavities where it becomes difficult to selectively etch the enamel without the risk of opening dentinal tubules for hydrogen peroxide and the subsequent risk of cervical resorption. Moreover, because this procedure would have to be repeated at every visit, it would add to the amount of tooth structure loss. Any walking bleach remnants on the external tooth surfaces or the rubber dam isolation need to be removed to avoid irritation of the soft tissues.
11. The rubber dam is removed and all tissues inspected.
12. The patient should be informed about the procedure. Depending on the reaction of the stain to the bleaching agent, the time to expect results may vary. Usually, a patient should be instructed to return between 3 and 10 days to evaluate the results and to change the walking bleach if necessary. To avoid overbleaching,[16] the patient needs to be instructed to self-monitor the color status of the tooth and to report back earlier than the scheduled appointment date if the color change has reached the desired result or if the temporary filling material is starting to wash out. Although a rarely documented event, it was advocated to instruct the patient about an increased susceptibility to tooth fracture in the time that the tooth is filled with a bleaching agent and not a supportive buildup.[18]
13. If desired results cannot be obtained, a mix with 3% H_2O_2 can be considered. However, the patient needs to be informed about the risks and that most good bleaching results can be accomplished by a sodium perborate and water/anesthetic mix.[206] If the esthetic outcome with the results is acceptable, a potential for resorption may be minimized.

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14. If a clinical case does not show improvement after three to four attempts, the diagnosis and treatment plan should be reevaluated for a different etiology.
15. At the conclusion of the bleaching procedure, a postoperative radiograph should be taken to check for any adverse effects of the bleaching agents. A definitive adhesive restoration should be placed about 1 to 3 weeks after the last appointment[42,215,216,246] (see the separate paragraph regarding definitive restoration after bleaching procedures a bit later).
16. The patient should be scheduled for yearly follow-ups to include a clinical examination and periapical radiographs.[49]

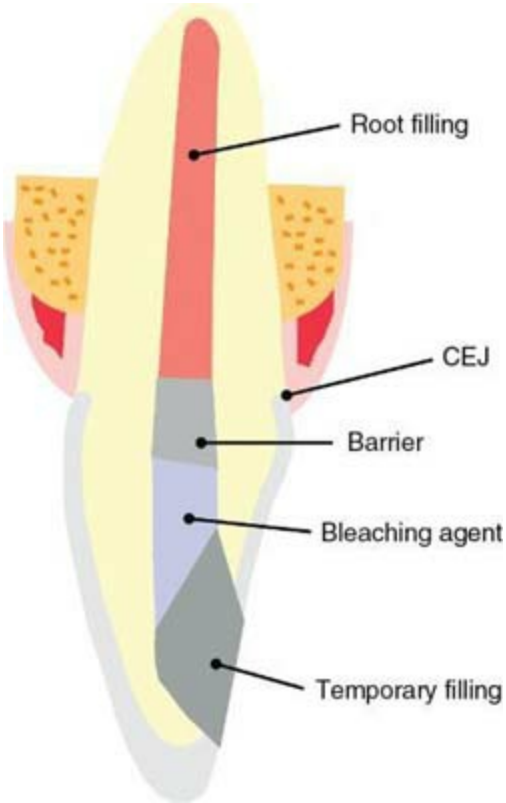

FIG. 26.3 Walking bleach technique: Placement of barrier material at the cementoenamel junction *(CEJ)* level, bleaching agent, and coronal seal with temporary filling material.

Cross-section diagram of tooth shows bleaching agent placed between temporary filling (bottom) and barrier (top) at the center with labels marked for root filling and C E J.

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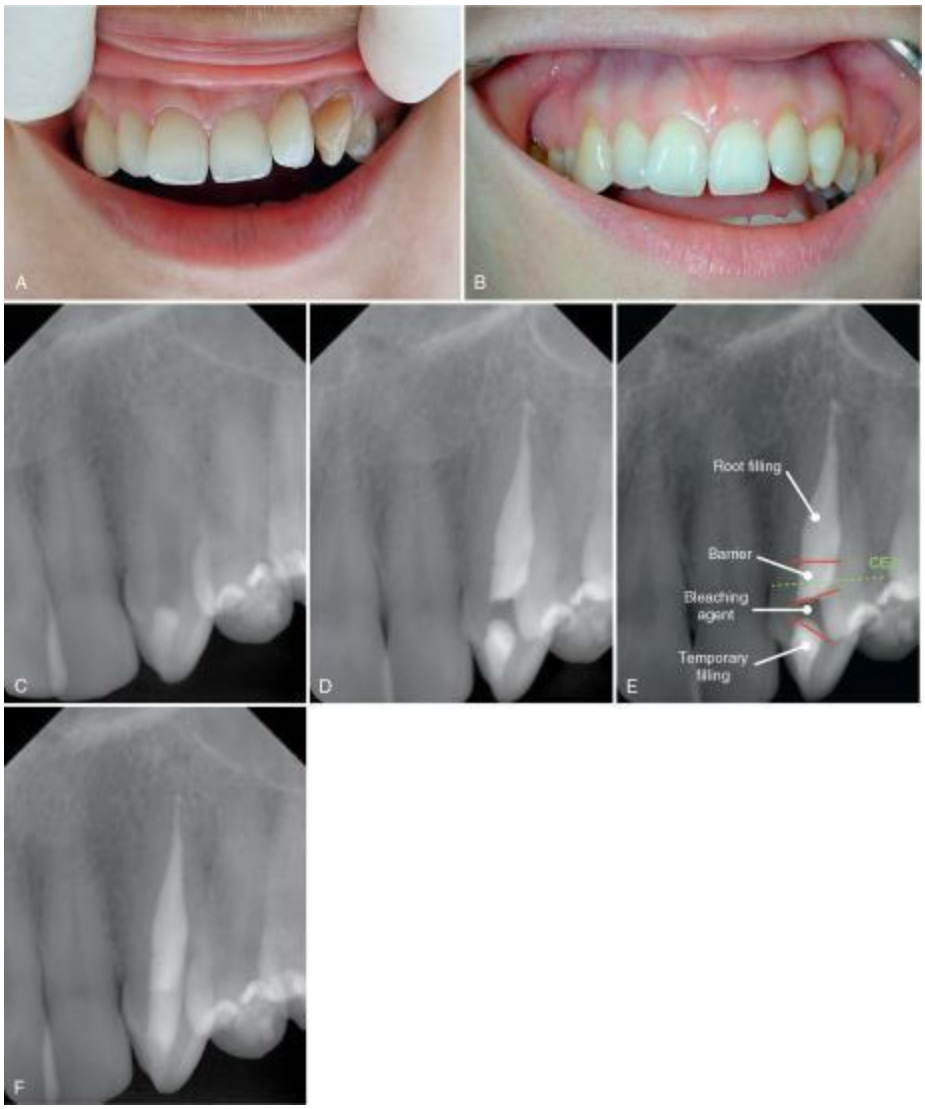

FIG. 26.4 Clinical case illustrating the walking bleach technique. **A,** Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left canine due to pulp necrosis. **B,** Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C,** Preoperative situation. **D,** Control radiograph after application of sodium perborate for the walking bleach technique. **E,** Illustration of part D showing placement of barrier material at the cementoenamel junction *(CEJ)* level, bleaching agent, and coronal seal with temporary filling material. **F,** Postoperative situation after bleaching, definitive coronal seal with adhesive restoration.

A) Close-up view of upper teeth shows pale canine on the left.

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B) Close-view of upper teeth shows yellowish staining at the gum line of lateral incisor and canine on the left.

C) Radiograph has radiolucent patch at the center extending from the crown in the central tooth.

D) Radiograph shows filled root canal of the central tooth where the region just above the neck is radiolucent.

E) Labels corresponding to radiolucent region (D) are as follows: root filling, barrier, bleaching agent, temporary filling, and CEJ.

F) Radiograph shows complete filling without gaps.

Source: (Courtesy Dr. Helmut Walsch, Munich, Germany.)

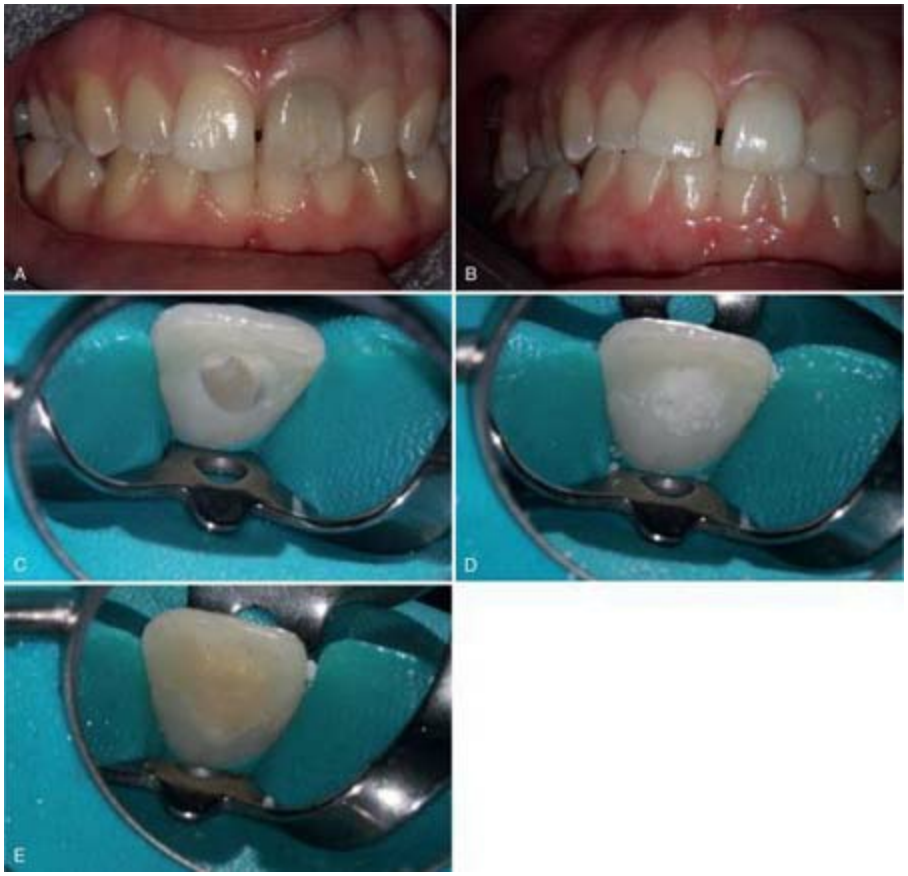

FIG. 26.5 Clinical case illustrating clinical steps of the walking bleach technique. **A,** Situation prior to endodontic treatment and internal bleaching. Discolored maxillary left central incisor due to dental trauma. **B,** Postoperative situation after endodontic treatment and internal bleaching using the walking bleach technique. **C,** Access cavity after

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barrier placement. **D,** Application of sodium perborate. **E,** Temporary coronal seal with adhesive restoration.

Set of five illustrations marked A through E depict five steps involved in the walking bleach technique.

Source: (Courtesy Dr. Mohammed Al-Harbi, Jeddah, Saudi-Arabia.)

Thermocatalytic bleaching

Heat-activated Superoxol had been advocated for its increased bleaching effect for nonvital bleaching of endodontically treated teeth.[112] Hydrogen peroxide with a concentration of 30% to 35% is placed in the pulp chamber and activated via heat application with electric heating devices or special lamps.[27,28,37,113,134,162,230] All preparatory steps follow the clinical protocol described previously for the walking bleach technique. However, in lieu of placing a sodium perborate/water mix, the pulp chamber is filled with 30% to 35% hydrogen peroxide and a heating device applied (e.g., Touch'n Heat, System B; Analytic Technology, Orange, CA; SuperEndo Alpha, B&L, Fairfax, VA). The application of heat will result in foaming of the hydrogen peroxide, with a subsequent release of free radical oxygen. This procedure is repeated in three to four office visits. In addition, some protocols advocate the placement of 30% to 35% hydrogen peroxide in the pulp chamber between chair-side appointments.[47,160,261] Temporary filling placement follows the same steps as outlined earlier for the walking bleach technique. If this technique is used, great care needs to be taken to avoid overheating of teeth, periodontal ligament, and gingival tissues. Regular cooling breaks are recommended. Products such as Vaseline, Orabase, or cocoa butter have been suggested for additional thermal insulation. However, due to the increased risk of cervical root resorption when using this clinical technique, the walking bleach technique is seen as more favorable today. If a decision is made to apply the thermocatalytic technique, the patient must be thoroughly informed about the risks and potential long-term consequences.

Definitive restoration of teeth following internal bleaching

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A well-sealing definitive restoration is one of the foundations for long-term success, as it prevents coronal microleakage from the access cavity and avoids the risk of renewed discoloration through open dentinal tubules.[18,264] It is generally accepted that an acid-etched and bonded composite resin restoration provides the most favorable results. For preexisting resin composite restorations, a rebonding of the filling has been suggested to reduce the risk of microleakage at the dentinal margins after bleaching with 15% carbamide peroxide.[169] The restoration must be placed at a depth that provides both the adequate seal for the access cavity as well as incisal support. The placement of a white base material below the resin restoration was viewed favorably in situations where the composite resin restoration may compromise the translucency of the tooth. Lighter shade composites were suggested for teeth with a not entirely satisfactory bleaching result.[4,81] Consensus exists that the adhesive bond strength between glass-ionomer cements and composite resins to dentin and enamel is temporarily compromised following bleaching procedures[60,78,117,234–236] due to remnants of peroxide or free oxygen, which inhibit resin polymerization.[60,239] Several remediation techniques have been suggested to overcome the negative influence of hydrogen peroxide containing bleaching agents, including dehydrating agents such as 80% alcohol and the use of acetone-containing adhesives,[118] the application of sodium hypochlorite to dissolve remnants of peroxide,[202] catalases,[195] the antioxidant sodium ascorbate,[121,142] or the use of alpha-tocopherol.[262] At least 1 week of contact with an aqueous solution was recommended to achieve adequate bonding.[5,234,240] Optimal bonding to bleached dentin and enamel was shown to be reestablished after 3 weeks.[42,216] Calcium hydroxide medication in the pulp cavity was advocated to achieve a buffering effect for an acidic pH that may be present after internal bleaching[18,122]; however, it may be unnecessary after a walking bleach technique, due to weaker nature of sodium perborate compared to Superoxol. Light curing of the composite resin restoration should occur from labial so that shrinkage toward the axial walls can reduce a potential risk of microleakage.[147]

External bleaching

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External bleaching may involve single teeth or entire arches. Whereas internal bleaching requires endodontic treatment of the tooth, external bleaching can also be applied to vital teeth. Steady advancements have been made in external bleaching techniques since their inception.[100,115] Various techniques exist, including in-office and at-home methods.

In-office external bleaching

The in-office or chairside techniques are completely in the hand of the dental professional. Almost all techniques involve the application of hydrogen peroxide gels of concentrations between 25% and 38%. Liquid solutions at higher concentrations are associated with higher complication rates of soft-tissue damage, as they are more difficult to control in terms of handling. High concentrations of hydrogen peroxide solutions are thermodynamically unstable and may explode if not stored in dark bottles in a refrigerator. It was also demonstrated in vitro that hydrogen peroxide gel concentrations above the 15% level did not increase bleaching effectiveness; they may, however, negatively affect enamel hardness.[87]

Although early techniques also employed heat activation, electric currents, or combinations with other chemicals to make hydrogen peroxide more effective,[153] contemporary techniques commonly use bleaching gels applied on their own or in combination with light activation by special lamps. Patients should undergo a hygiene treatment with cleaning and polishing of the tooth surfaces prior to external bleaching, as well as a careful evaluation of the nature of the stain or the possibility that the discoloration is related to an existing restoration.

If external bleaching is indicated, the treatment involves the placement of rubber dam, as described in the protocol for the walking bleach technique. Great care must be taken to avoid soft-tissue damage. If liquid hydrogen peroxide solutions and no gels are used, the additional placement of gauze to act as a reservoir for hydrogen peroxide is recommended to keep the solution in the proximity of the teeth. More gauze can be placed underneath the rubber dam as an additional safety to catch excess solution before it can damage gingiva, mucosa, or lips. However, these complications can be greatly reduced by a gel application with or without light activation.

Light sources used for bleaching include conventional ultraviolet (UV)

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bleaching lights, tungsten-halogen and Xe-halogen lights, plasma arc lamps, light-emitting diodes (LEDs), or laser lights. The wavelengths of the various light sources may range from the high UV to low visible blue light spectra, and invisible infrared light, such as employed by a CO_2 laser.[37,194] Although all of these lights have been used to activate the bleaching agent or expedite the whitening effect, there are great differences among them. Limited research on bleaching lights exists, sometimes with controversial results. Although some studies emphasized certain effects after light-activated bleaching, others reported no differences between teeth bleached with and without light activation.[37,138,194,227] In the 1980s, the Fuji HiLite dual-cure material containing 35% hydrogen peroxide was used. When the paste was mixed, it had a green color that would turn white upon activation with a standard UV light upon the release of oxygen. In comparison to other light activation techniques, this was a more time-consuming process and less comfortable for the patient. In addition to light activation alone, various photocatalysts had been suggested, including methylene blue and UV or visible light-activated titanium dioxide.[224]

Traditionally, tungsten-halogen curing lights are the standard dental curing lights that provide heat and stimulate the initiation of the chemical reaction by activating light-sensitive chemicals in the bleaching agent. Depending on manufacturer recommendations, the individual bleaching time with activation of the gel is up to 30 to 60 seconds per application per tooth, which is rather time consuming for the patient and the dental professional because up to three passes are recommended. Some products available were based on a premixed 35% hydrogen peroxide gel with carotene that converts light energy to heat upon activation and increased the breakdown of the bleaching agent into active free radicals. Xenon-halogen curing lights are newer generation dental curing lights used to the same effect. Some specialized bleaching lights offer full-arch adaptors. A randomized clinical trial of in-office tooth whitening identified blue light-activated bleaching as the one technique with results most favored by patients.[9]

Xenon Plasma Arc Lamp systems are primarily based on thermal activation and the activation of chemical catalysts in the bleaching gel. The light employed is of high intensity, produces great heat energy, and limits the application to 3-second intervals for up to three passes. Due to the high heat

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intensity, there is a potential for dental trauma to the pulp and the surrounding tissues, which is why these lights have to be used with great care. Some manufacturers produced lights that allowed an on-off mode setting over 5-second bursts to have a safer and lower-intensity output.

LED lamps emit cold blue light with a wavelength of around 465 nm, which allows activation of hydrogen peroxide gels and accelerates the bleaching process. LED lights are placed close to the patient's teeth for 15- to 20-minute treatments, which may have to be repeated up to three times. Cosmetic specialists with no dental background have adopted this method of bleaching because it offers low risk, as no heat activation is involved. Green LED lights demonstrated the lowest intrapulpal temperature increase compared to conventional halogen, hybrid lights with or without a laser component, or high-intensity LED lights.[167] However, a systematic review investigating the influence of the light source on pulpal responses during bleaching procedures concluded that there was not sufficient evidence to extrapolate the true human situation.[23]

Lasers have been in use for tooth bleaching at around 810 to 830 nm and 970 to 980 nm wavelengths in combination with 30% to 35% hydrogen peroxide gel. Gels prepared for laser activation may contain fumed silica and a blue dye, the latter absorbing the laser wavelength, leading to an increase in temperature and a controlled breakdown of the hydrogen peroxide. Gels are commonly applied in a 2- to 3-mm thickness over the teeth. An application of 1 to 2 W of laser energy for 30 seconds per tooth has been recommended. Dental professionals and patients are required to wear protective eyewear while operating lasers. Some studies reported no significant advantage to using laser light for bleaching,[37] whereas others demonstrated a significant increase in whiteness and decrease in yellowness compared to conventional power bleaching[66] or showed a decrease in color regression after a combination of at-home bleaching with an in-office laser-assisted bleaching session in comparison to an at-home bleaching regimen on its own.[6]

Power bleaching

The technique normally termed *power bleaching* refers to accelerated vital in-office tooth whitening procedures that employ xenon plasma arc-curing lights or lasers. Dental hygienists or assistants may carry out this procedure,

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depending on individual state licensing regulations. The use of laser lights might be more strictly regulated and may not be permitted to be performed by dental professionals other than the dentist. For a delegated procedure, liquid rubber dam is often applied in lieu of a sheet of rubber dam, which would require active retention techniques, such as wedges, floss, or ligatures. Liquid rubber dam can be applied more easily without assistance, but it does not give additional protection to gingival tissues or the mucosa.

Power bleaching clinical protocol

1. Prior to initiating the bleaching procedure, a diagnosis must confirm that the discolorations can be resolved by external bleaching. The patient must be informed about the history, the procedures involved in the treatment, the expected outcome of the procedure, and the potential for rediscoloration.
2. A thorough clinical examination should be performed to detect caries, developmental defects, endodontic or periodontal diseases, and other pathologic conditions in the oral cavity. Existing restorations need be checked for quality and shade and replaced if defective. For certain teeth, the reason for discoloration may be leaking restorations or discolored filling materials. Replacement of a defective restoration may resolve most of the discoloration.
3. Patients should have a hygiene appointment prior to the bleaching session to check for sound gingival tissues and ensure that areas to be bleached are free of calculus or plaque.
4. Clinical photographs should be taken in the beginning, throughout, and at the end of the treatment with the tooth next to a shade guide to act as a reference for the practitioner and the patient.
5. The patient and dental professionals should wear proper protective eyewear (sunglasses). The patient should be prepared to signal if a tingling sensation to the gingiva, mucosa, lips, or teeth is felt or if the temperature is perceived as too hot. As a precaution, vitamin E capsules, a powerful antioxidant, should be ready at hand. In case of an emergency, these can be cut open and the oil inside the capsules applied to the injured tissues with a cotton pellet.
6. The teeth should be cleaned again with rubber cups and pumice. The

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cheeks should be retracted with photo retractors or cotton rolls. The cotton rolls serve as an additional safety feature if any hydrogen peroxide is leaking. Rubber dam isolation is mandatory. This can be either liquid rubber dam (light-cured resin dam) around the gingival margins or ligated sheet rubber dam. Liquid rubber dam bears a higher risk of tissue injury. It is important to create a tight seal at the gingival margin to prevent leakage of bleaching agents. If a sheet rubber dam is used, the inversion of the rubber dam in the sulcus with an air syringe followed by dental loss ligature is recommended.

7. The power bleach gel is mixed according to the manufacturer's recommendation and applied on the labial surfaces of the teeth in a 2- to 3-mm thickness with a disposable brush.
8. Depending on the light source, each individual tooth is exposed for up to three passes for 3 to 10 seconds, according to manufacturers' recommendations. The gel may stay on the teeth for another 3 to 5 minutes without light activation.
9. The gel should be removed with a wet gauze and copious amounts of water, then cleaned with pumice for a second time, and rinsed again to ensure all remnants of the bleaching gel were removed.
10. The rubber dam and any remaining cotton rolls and retractors are removed. Water rinsed is used again, and all tissues inspected.
11. The teeth should be polished and a neutral pH sodium fluoride gel be applied.
12. The patient should be informed about the procedure. Depending on the individual situation, results may vary. The patient should be instructed that increased sensitivity of the teeth may be present for 2 to 3 days.
13. The patient ought to be instructed to refrain from tobacco, coffee, tea, cola, and wine for a period of 2 weeks.[51]

At-home external bleaching

For patients who do not want to undergo an in-office procedure, the possibility of at-home external bleaching with custom-made bleaching trays exist. These may be readily available from a pharmacy, requiring only crude adaptation after softening in hot water, or can be custom-fabricated in a

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dental laboratory. Over-the-counter products typically come with bleaching gels, although some come with pastes. Some products are offered as simple, low-strength hydrogen peroxide strips or pastes that can be brushed onto the teeth. Occasionally, pre-rinses are offered. However, pre-rinses may have a rather acidic pH, as the solutions may become unstable more easily.

Professional home bleaching involves the fabrication of a well-adapted custom tray after impressions are taken and models are poured. The patient will wear gel loaded in a tray that slowly releases the bleaching agents for several hours or overnight every day for 2 to 6 weeks until a satisfactory result has been achieved.[44,96–100]

Trays are usually made using alginate impressions. Upon delivery, trays must be checked for fit, smoothness at the margins, and occlusion. The patient should place several drops of bleaching gel into the tray before every application. The most common bleaching agent for at-home bleaching is 10% to 22% carbamide peroxide with an effective yield of 4% to 7.5% hydrogen peroxide. Patients who wear the trays during the daytime may choose to replace the gel every 2 hours. If any disturbance (e.g., thermal sensitivity, abnormal taste, or tissue irritation) occurs, the patient should stop the procedure and seek advice from the dentist. Patients who use the trays in the daytime should be seen once per week for 3 weeks; nighttime users should be seen every other week for 6 weeks.

The success of at-home bleaching mostly depends on the patient's cooperation. Risks include potential compliance issues and the possibility of overuse of the bleaching agent. Results with at-home bleaching techniques tend to remain stable for 1 to 10 years.[149]

Risks and complications associated with tooth bleaching

Risks associated with internal tooth bleaching

Tooth bleaching may cause localized adverse effects on hard and soft tissues, such as the risk of external cervical resorption, interaction with adhesive bonding systems, and dental material solubility.[11]

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Cervical root resorption

External or cervical root resorption is a serious complication of whitening procedures with peroxide compounds (Fig. 26.6). Cervical root resorptions were seen in 6% to 8% of cases when 35% hydrogen peroxide was used, and 18% to 25% if the hydrogen peroxide was heat activated.[54,73,74,94,104,141,204] The etiology for external root resorption to occur is complex.[86,94,136,141] It is suggested that a combination of predisposing factors—such as a cementum deficiency exposing dentin, a periodontal ligament injury that is triggering an inflammatory response, or an infection that is sustaining inflammation—has to be present.[18,74,178,244] Cervical resorptions are also often seen in patients who had bleaching treatment at a young age[7,12,74,104,108,141] or who suffered from a traumatic injury.[94,242]

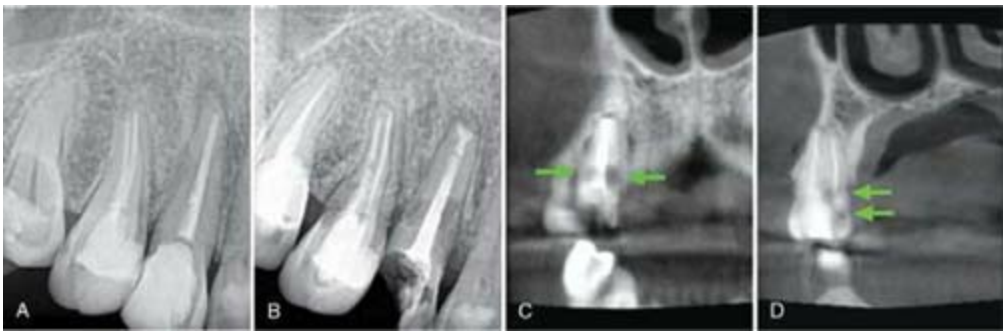

FIG. 26.6 Clinical case of a 33-year-old female patient illustrating extensive resorptive defects after nonvital bleaching efforts. **A,** Postoperative situation after endodontic treatments of the maxillary right canine and first premolar. The patient was referred back to the primary care provider for definitive restorations. **B,** Situation 12 months after the original endodontic treatments. The patient had spent 1 year overseas and received endodontic treatment on the second premolar, root-end surgery on the canine, as well as nonvital bleaching of the maxillary right canine and first premolar. Note the extensive resorptive defects on both teeth that received bleaching procedures and the missing restoration on the canine. **C** and **D,** Coronal CBCT views of canine and first premolar. Note location and dimensions of the resorptions *(arrows)*.

Four radiographs are marked A through D.

A) It shows radiolucent canal of tooth on the right.

B) It shows filled root canal of tooth on the right. The crown of left tooth is

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broken.

C) Two arrows point toward the radiolucent spots close to the root canal in the broken tooth.

D) Two arrows point toward partial radio-opaque regions close to the root canal.

Source: (Courtesy Dr. Mariana Lis Robles, Mendoza, Argentina.)

Hydrogen peroxide itself was suggested to be responsible for the induction of the resorptive process.[94] The body can handle hydrogen peroxide effectively at lower concentrations.[92]

Proinflammatory agents activate reduced nicotinamide adenine dinucleotide phosphate oxidase, which produces superoxides that can react with hydrogen peroxide in the presence of inflammation.[89] This may allow the formation of hypochlorous acid, N-chloramines, and reactive hydroxyl ions, and cause a resorptive process.[89] Hydrogen peroxide may reach the bone by diffusion via dentinal tubules, cementum, and the periodontal ligament.[89,184,257] Within the bone, the acidic environment induced by the low pH value of hydrogen peroxide at high concentrations may facilitate osteoclastic activity.[74,249] Significant increases in the levels of the bone resorption markers RANK-L and IL-1β were observed in the gingival crevicular fluid of teeth undergoing nonvital bleaching with either 35% hydrogen peroxide or 37% carbamide peroxide.[24] After application to the enamel surface, 25% hydrogen peroxide was also shown to cross enamel and the dentinoenamel junction (DEJ), reaching the dentin.[245]

About 10% of all teeth have a natural defect at the cementoenamel junction, exposing dentin.[229] These cementum defects, as well as unusual morphology at the CEJ, can favor the penetration of hydrogen peroxide into the cervical area of a tooth.[135,177,204] The placement of 30% hydrogen peroxide in the pulp chamber of teeth with missing parts of the cementum layer resulted in the diffusion of 82% of the bleaching agent.[204] Dentin exposure may also be a result of defects in the cementum layer following traumatic injuries or an incomplete formation of the cementoenamel junction.[161,168] The permeability of the dentin can then facilitate the penetration of bacteria or bacterial endotoxins from an infected root canal

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system or pulp chambers to the surface of the root, potentially causing inflammatory processes in the periodontal ligament.[105,170] If highly concentrated bleaching agents are used, in particular, 30% to 35% hydrogen peroxide, a diffusion of highly reactive hydroxyl radicals via unprotected dentinal tubules and cemental defects has been suggested.[135,204]

Free oxygen radicals are also able to break down constituents of connective tissue, in particular collagen and hyaluronic acid, which may be another likely mechanism responsible for periodontal tissue destruction and root resorptions after internal bleaching.[55] The oxidizing agents were shown to induce dentin protein denaturation,[141] either by local changes in the pH[58,80,168] or by heat.[71,94,161] Pretreatment is with EDTA, a chelating complex binding iron. Heat activation of 30% to 35% hydrogen peroxide is the characteristic step of the thermocatalytic method. It may result in widening of the dentinal tubules, allowing for a diffusion of free radicals into the dentin[183,203] and an enhancement of resorptive processes.[203] The hydroxyl radicals released from hydrogen peroxide are extremely reactive and have been shown to degrade components of connective tissue.[55] As a consequence, the thermocatalytic technique is nowadays disfavored due to the high risk of external root resorption.

The potential for cervical resorption may be minimized by using sodium perborate with water instead of hydrogen peroxide for internal bleaching techniques, because similar acceptable outcomes have been reported[206] and because it did not cause cervical resorption, even 1 year after bleaching.[161] Sodium perborate-water suspensions were also shown to be less toxic to periodontal ligament cells than 30% hydrogen peroxide or sodium perborate mixed with 30% hydrogen peroxide.[129,181] Moreover, a strong association was found between tooth discoloration related to blood components and an increased presence of hydroxyl radicals.[55]

Treatment of cervical resorption may range from simple fillings to extraction, depending on the severity of the resorptive defect. An early detection of a resorptive process increases the chances of avoiding unnecessary tooth loss because the prognosis for cases with advanced resorptions is doubtful.[59] Evaluation by means of a CBCT may greatly enhance the ability for correct treatment planning.[64] If resorptions are related

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to internal bleaching, the placement of calcium hydroxide into the root canal space has been suggested[74] to change the low pH present in inflammatory osteolytic processes characteristic for hard-tissue resorption.[164] Reparative hard-tissue formation was suggested to take place following this treatment.[243] Some cervical resorption can be repaired with direct restorations, such as composite resin or glass ionomer-composite compomers (e.g., Geristore, DenMat, Lompoc, CA). This treatment requires a surgical exposure of the defect, followed by degranulation by curettage and the application of 90% trichloroacetic acid to remove remnant tissues prior to adhesive restorations.[72,166] Patients should be informed about the potential for gingival recession if the surgical access requires a full thickness flap in the anterior area.[64,80] Rapid orthodontic extrusion in combination with fiberotomy and restoration was suggested for resorptions with a substantial subcrestal component.[103,144]

The free radicals released by peroxides can cause chemical burns and tissue sloughing on gingiva, mucosa, or other oral soft tissues. This may occur during the internal bleaching procedure in the dental chair during both walking bleach and thermocatalytic technique appointments after a patient has left the office if a walking bleach technique is used. In any case, prevention is the best cure, and avoiding accidents is the priority. In general, the use of higher concentrations of peroxides, such as 30% to 35% hydrogen peroxide, in particular in liquid form, is more dangerous than the use of a mixed paste or gel of sodium carbamide peroxides that have lower effective hydrogen peroxide concentrations and can be used in a more controlled way. However, in the dental office, great care has to be taken so that the bleaching agents do not leak through the rubber dam and all excessive bleaching material is carefully removed after the end of the procedure (see details in Walking Bleach Clinical Protocol). For emergency situations, clinicians should have vitamin E on hand, because this tough antioxidant reverses the strong oxidation processes on oral soft tissues and minimizes damage (see details in Power Bleaching Clinical Protocol). To avoid out-of-office accidents, it must be assured that the temporary filling material is sealing tightly and is placed in an adequate thickness to cover the bleaching agent. Patients should be advised to return immediately after a temporary restoration starts to fail or wash out.

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Enamel and dentin damage

A number of in vitro studies reported changes in enamel microhardness and morphology,[25,43,87,159,172,197,199,263,266] as well as in the cementum.[237,265] It has been suggested that peroxide components alter the proportions of organic to inorganic hard tissue components.[96,105,152,191,] A treatment with 10% carbamide peroxide and 7.5% hydrogen peroxide did alter the enamel surface micromorphology but not its microhardness.[209] Hydrogen peroxide demonstrated a different chemical affinity to each specific dental tissue, rather than just to diffuse through interprismatic spaces into the dentinal tubules when applied to the enamel surface.[245] It was also shown to modify the organic compounds of dentin.[245]

At least in vital teeth, however, these changes appear not to be permanent[15,79] and can be reduced by the application of fluoride before or during remineralization.[36,79] Bleaching with hydrogen peroxide increased the demineralization in teeth affected by incipient caries lesions.[34] The bleached enamel surface may show an increased porosity and slight erosion when observed under scanning electron microscopy (SEM). These changes were mostly seen when acidic prerinses or bleaching gels with a low pH had been applied.[247] One study found no differences between bleached and unbleached human enamel over a 6-month period when a 10% carbamide peroxide gel was used.[102] No significant clinical evidence of negative effects on enamel by professionally administered at-home bleaching techniques was seen. A few case reports described significant enamel damage after over-the-counter products had been applied by patients, possibly the result of overuse.[53]

Inhibition of adhesive bonding

Polymerization and bonding strength of composite resins to dentin and enamel are temporarily compromised by the oxygen released during a bleaching process.[14,60,226,233,] Scanning electron microscope images have shown an increased porosity of the enamel after bleaching.[235] This may present a clinical issue in terms of leaking restorations with the potential for caries activity and rediscoloration. Several remedial solutions were suggested, including rinsing the cavity with sodium hypochlorite,[202] which, however, was demonstrated to have similar effects on bonding, alcohol,[118]

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chlorhexidine, catalase, and sodium ascorbate.[48,121,142] Clinicians are generally advised to wait for 2 to 3 weeks prior to placing a definitive adhesive restoration to allow time to reverse the effects and have all remnants of hydrogen peroxide removed from the pulp chamber.[48]

Risks associated with external tooth bleaching

External tooth bleaching (including professional at-home tray bleaching techniques) is, generally speaking, a safe procedure that allows tooth whitening to be retained for several years.[149] Nevertheless, concerns have been raised since the introduction of external bleaching,[101,154–157] mostly related to the caustic effects of free oxygen radicals that may cause tissue damage in oral soft and hard tissues. The reaction with proteins, lipids, and nucleic acids may eventually lead to cell damage. Human dental pulp cells in contact with enamel/dentine discs treated with 17.5% hydrogen peroxide gel demonstrated significant overexpression of inflammatory mediators, as well as significant cell viability reduction.[219] At-home external bleaching did not demonstrate significant long-term complications if 10% carbamide peroxide was used as the bleaching agent. However, since at-home bleaching does not give the practitioner control over the patient's behavior, higher concentrations of carbamide peroxide than recommended may be used and could more likely cause damage to the gingival and periodontal tissues.[106] In general, caution is advised with regard to the quality of product, techniques used, and treatment rendered.

Tooth sensitivity

The presence of hypersensitivity is an indication of inflammatory changes in the dental pulp.[156] Histologically, however, few pulps have demonstrated actual signs of inflammation after external bleaching with hydrogen peroxide gels.[128] Nevertheless, changes in sensitivity after external bleaching of vital teeth have been reported in up to 50% of the teeth.[101,153] Most of the time the change in sensitivity is mild to moderate and is transient over a period of 2 to 3 days.[116,150,190] The use of 37% carbide peroxide compared to 35% hydrogen peroxide for in-office bleaching demonstrated reduced sensitivity in a randomized controlled trial.[187] A systematic review and meta-analysis

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assessing in-office versus at-home vital bleaching in adult patients did not identify differences regarding the risk or intensity of tooth sensitivity nor the effectiveness of the bleaching treatment.[57] In the case of at-home bleaching, the incidence of hypersensitivity increases with the frequency of changing the bleaching trays, particularly if they are changed more than once daily.[150] For the best immediate remediation, the bleaching periods should be shortened. Both topical fluoridation and desensitizing toothpaste may aid in reducing the hypersensitivity after bleaching.

Hydrogen peroxide that is released from the bleaching trays or from the gel during power bleaching may reach the pulp chamber via craze lines, enamel defects, as well as enamel and dentin cracks.[32,50,82,193] However, it was shown that 50,000 µg of hydrogen peroxide are required to inhibit pulpal enzymes.[92] From bleaching gels containing 12% carbamide peroxide, less than 30 µg of hydrogen peroxide were shown to reach the pulp chamber after tray applications for up to 7 hours. Therefore at least for at-home external bleaching, it is unlikely to create long-term or significant damage. Nevertheless, to avoid complications, active bleaching should be avoided whenever restorations with ill-adapting margins or exposed dentin areas are present. The microhardness of methacrylate-based composite resins can be affected by carbamide peroxide in home bleaching concentrations.[63] It has been suggested that desensitizers be used in addition to the bleaching gel. Fluoride, casein phosphopeptide-amorphous calcium phosphate, potassium nitrate, or nano-hydroxyapatite suspension applied after the in-office bleaching process or mixed into bleaching agent were shown to recover the microhardness of the enamel after 14 days.[140] Caution should also be applied regarding children and adolescents due to the larger pulpal volume present in immature, respectively younger teeth.[146]

Gingival irritation

The most commonly observed adverse effect after external bleaching is gingival irritation, which may be associated with an increase in tooth sensitivity.[148,205,211] Most of the gingival irritations are mild to moderate and disappear after 2 to 3 days without causing significant discomfort for the patient. For in-office bleaching, these issues are mostly related to soft-tissue exposures to excessive bleaching gel or liquid hydrogen peroxide in amounts

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less than necessary to cause severe discomfort or tissue damage. However, when a patient reports tissue irritation, the soft tissues should be checked immediately. Warning signs other than the patient's sensation are air bubbles rising from the gingival margins. The area should be copiously rinsed without delay. A vitamin E preparation should be on hand to serve as an emergency dressing that will provide an immediate antioxidative effect. If an at-home technique is used, gingival irritations may be related to badly adapting trays, and the issue should be resolved after correcting the defective trays. As for gingival hypersensitivity, the increase of gingival irritation is related to the frequency of changing the gel in the custom trays. For more severe cases, topical anesthesia, limited movement, and good oral hygiene can increase the speed of soft-tissue healing.

Mercury release from amalgam restorations

Several studies have reported an increased mercury release from amalgam restorations after the application of bleaching agents, in particular carbamide peroxide.[198,201] Although mercury is toxic and can cause damage in larger doses, no health implications due to the induced release of mercury by tooth bleaching have been reported.

Prognosis of bleaching

Most studies addressing the prognosis of internal bleaching refer to the immediate postoperative whitening result.[4,7,12,108,112,162,178] The higher concentrations of bleaching agents used for in-office vital bleaching versus at-home bleaching products were demonstrated to produce greater bleaching effectiveness.[131] A long-term follow-up of teeth bleached with the thermocatalytic technique reported an 80% success rate after 1 year and a 45% success rate after 6 years, confirming the redarkening of bleached teeth in many instances.[67] External bleaching may show color regressions after 1 to 3 years.[38] A randomized clinical trial evaluating CIELab parameters of teeth bleached using the walking bleach or an inside-outside technique demonstrated no significant changes in color and lightness comparing the short-term bleaching results with the 1-year follow-up.[186] Al-Tarakemah and Darvell reevaluated a data set of 300 patients, coming to the conclusion that some patients who started out with lighter color shades at the beginning of a

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bleaching procedures may be worse off after 1 year of follow-up, attributing this to cumulative damages to the organic matrix of the enamel.[10] Discolorations induced by trauma or pulp necrosis appear to have a good prognosis of around 95%, indicating that bleaching reaches the desired result, whereas discolorations due to medications or restorations have a much less successful outcome.[176] Discoloration caused by restorative materials has an uncertain prognosis,[250] particularly if metallic ions such as silver, mercury, copper, or iodine are involved. Although some authors disagree,[1,35,113] there may be a higher possibility for successful bleaching in younger patients due to the wider open dentinal tubuli when compared to older patients.[2,107,178] Gray or light yellow discolorations may be easier to bleach than other discolorations.[1] Well-sealing composite restorations are recommended to prevent renewed discoloration.[1]

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References

1. Abbott P, Heah SY. *Internal bleaching of teeth: an analysis of 255 teeth*, Aust Dent J 2009;54: 326.
2. Abou-Rass M. *The elimination of tetracycline discoloration by intentional endodontics and internal bleaching*, J Endod 1982;8: 101.
3. Abou-Rass M. *Evaluation and clinical management of previous endodontic therapy*, J Prosthet Dent 1982;47: 528.
4. Abou-Rass M. *Long-term prognosis of intentional endodontics and internal bleaching of tetracycline-stained teeth*, Compend Contin Educ Dent 1998;19: 1034.
5. Adibfar A, Steele A, Torneck CD, et al: *Leaching of hydrogen peroxide from bleached bovine enamel*, J Endod 1992;18: 488.
6. Al Quran FA, Mansour Y, Al-Hyari S, et al: *Efficacy and persistence of tooth bleaching using a diode laser with three different treatment regimens*, Eur J Esthet Dent 2011;6: 436.
7. Aldecoa EA, Mayordomo FG. *Modified internal bleaching of severe tetracycline discoloration: a 6-year clinical evaluation*, Quintessence Int 1992;23: 83.
8. Allan NA, Walton RC, Schaeffer MA. *Setting times for endodontic sealers under clinical usage and in vitro conditions*, J Endod 2001;27: 21.
9. Alomari Q, El Daraa E. *A randomized clinical trial of in-office dental bleaching with or without light activation*, J Contemp Dent Pract 2010;11: E017.
10. Al-Tarakemah Y, Darvell BW. *On the permanence of tooth bleaching*, Dent Mater 2016;32: 1281.

Telegram: @dental_k

www.konkur.in

11. Anderson DG, Chiego DJ Jr, Glickman GN, McCauley LK. *A clinical assessment of the effects of 10% carbamide peroxide gel on human pulp tissue*, J Endod 1999;25: 247.
12. Anitua E, Zabalegui B, Gil J, et al: *Internal bleaching of severe tetracycline discolorations: four-year clinical evaluation*, Quintessence Int 1990;21: 783.
13. Arens D. *The role of bleaching in esthetics*, Dent Clin North Am 1989;33: 319.
14. Attin T, Hannig C, Wiegand A, et al: *Effect of bleaching on restorative materials and restorations—a systematic review*, Dent Mater 2004;20: 852.
15. Attin T, Hartmann O, Hilgers RD, et al: *Fluoride retention of incipient enamel lesions after treatment with a calcium fluoride varnish in vivo*, Arch Oral Biol 1995;40: 169.
16. Attin T, Paque F, Ajam F, et al: *Review of the current status of tooth whitening with the walking bleach technique*, Int Endod J 2003;36: 313.
17. Banting DW. *Management of dental caries in the older patient*, Papas ASN Niessen LC Chauncey HH Geriatric dentistry 1991; Mosby Year Book St. Louis 141-167.
18. Baratieri LN, Ritter AV, Monteiro S Jr, et al: *Nonvital tooth bleaching: guidelines for the clinician*, Quintessence Int 1995;26: 597.
19. Barron MJ, McDonnell ST, Mackie I, et al: *Hereditary dentin disorders: dentinogenesis imperfecta and dentin dysplasia*, Orphanet J Rare Dis 2008;3: 31.
20. Basrani BR, Manek S, Mathers D, et al: *Determination of 4-chloroaniline and its derivatives formed in the interaction of sodium hypochlorite and chlorhexidine by using gas chromatography*, J Endod 2010;36: 312.
21. Basrani BR, Manek S, Sodhi RN, et al: *Interaction*

Telegram: @dental_k

www.konkur.in

between sodium hypochlorite and chlorhexidine gluconate, J Endod 2007;33: 966.

22. Belobrov I, Parashos P. *Treatment of tooth discoloration after the use of white mineral trioxide aggregate*, J Endod 2011;37: 1017.
23. Benetti F, Lemos CAA, de Oliveira Gallinari M, et al: *Influence of different types of light on the response of the pulp tissue in dental bleaching: a systematic review*, Clin Oral Investig 2018;22: 1825.
24. Bersezio C, Vildósola P, Sáez M, et al: *Does the use of a "walking bleaching" technique increase bone resorption markers?*, Oper Dent 2018;43: 250.
25. Bitter NC. *A scanning electron microscope study of the long-term effect of bleaching agents on the enamel surface in vivo*, Gen Dent 1998;46: 84.
26. Black GV, McKay FS. *Mottled teeth—an endemic developmental imperfection of the teeth heretofore unknown in the literature of dentistry*, Dental Cosmos 1916;58: 132.
27. Boksman L, Jordan RE, Skinner DH. *Non-vital bleaching—internal and external*, Aust Dent J 1983;28: 149.
28. Boksman L, Jordan RE, Skinner DH. *A conservative bleaching treatment for the nonvital discolored tooth*, Compend Contin Educ Dent 1984;5: 471.
29. Bortoluzzi EA, Araujo GS, Guerreiro Tanomaru JM, et al: *Marginal gingiva discoloration by gray MTA: a case report*, J Endod 2007;33: 325.
30. Botelho MG, Chan AWK, Newsome PRH, et al: *A randomized controlled trial of home bleaching of tetracycline-stained teeth*, J Dent 2017;67: 29.
31. Boutsioukis C, Noula G, Lambrianidis T. *Ex vivo study of the efficiency of two techniques for the removal of mineral*

Telegram: @dental_k

www.konkur.in

trioxide aggregate used as a root canal filling material, J Endod 2008;34: 1239.
32. Bowles WH, Ugwuneri Z. *Pulp chamber penetration by hydrogen peroxide following vital bleaching procedures*, J Endod 1987;13: 375.
33. Brady JM, del Rio CE. *Corrosion of endodontic silver cones in humans: a scanning electron microscope and X-ray microprobe study*, J Endod 1975;1: 205.
34. Briso AL, Gonçalves RS, Costa FB, et al: *Demineralization and hydrogen peroxide penetration in teeth with incipient lesions*, Braz Dent J 2015;26: 135.
35. Brown G. *Factors influencing Successful bleaching of the discolored root-filled tooth*, Oral Surg Oral Med Oral Pathol 1965;20: 238.
36. Bruun C, Givskov H. *Formation of CaF2 on sound enamel and in caries-like enamel lesions after different forms of fluoride applications in vitro*, Caries Res 1991;25: 96.
37. Buchalla W, Attin T. *External bleaching therapy with activation by heat, light or laser—a systematic review*, Dent Mater 2007;23: 586.
38. Burrows S. *A review of the efficacy of tooth bleaching*, Dent Update 2009;36: 537.
39. Cagetti MG, Cattaneo S, Hu YQ, et al: *Amelogenesis imperfecta: a non-invasive approach to improve esthetics in young patients: report of two cases*, J Clin Pediatr Dent 2017;41: 332.
40. Carlsson J. *Salivary peroxidase: an important part of our defense against oxygen toxicity*, J Oral Pathol 1987;16: 412.
41. Casey LJ, Schindler WG, Murata SM, et al: *The use of dentinal etching with endodontic bleaching procedures*, J

Telegram: @dental_k

www.konkur.in

Endod 1989;15: 535.
42. Cavalli V, Reis AF, Giannini M, et al: *The effect of elapsed time following bleaching on enamel bond strength of resin composite*, Oper Dent 2001;26: 597.
43. Chng HK, Ramli HN, Yap AU, et al: *Effect of hydrogen peroxide on intertubular dentin*, J Dent 2005;33: 363.
44. Christensen GJ. *Bleaching teeth: report of a survey, 1997*, J Esthet Dent 1998;10: 16.
45. Crystal YO, Janal MN, Hamilton DS, et al: *Parental perceptions and acceptance of silver diamine fluoride staining*, J Am Dent Assoc 2017;148: 510.
46. Clarkson J. *Review of terminology, classifications, and indices of developmental defects of enamel*, Adv Dent Res 1989;3: 104.
47. Cohen S. *A simplified method for bleaching discolored teeth*, Dent Dig 1968;74: 301.
48. Comlekoglu ME, Gokce B, Kaya AD, et al: *Reversal of reduced bond strength after bleaching*, Gen Dent 2010;58: 258.
49. *Consensus report of the European Society of Endodontology on quality guidelines for endodontic treatment*, Int Endod J 1994;27: 115.
50. Cooper JS, Bokmeyer TJ, Bowles WH. *Penetration of the pulp chamber by carbamide peroxide bleaching agents*, J Endod 1992;18: 315.
51. Cortes G, Pini NP, Lima DA, et al: *Influence of coffee and red wine on tooth color during and after bleaching*, Acta Odontol Scand 2013;71: 1475.
52. Costas FL, Wong M. *Intracoronal isolating barriers: effect of location on root leakage and effectiveness of bleaching agents*, J Endod 1991;17: 365.
53. Cubbon T, Ore D. *Hard tissue and home tooth whiteners*,

Telegram: @dental_k

www.konkur.in

CDS Rev 1991;84: 32.
54. Cvek M, Lindvall AM. *External root resorption following bleaching of pulpless teeth with oxygen peroxide*, Endod Dent Traumatol 1985;1: 56.
55. Dahlstrom SW, Heithersay GS, Bridges TE. *Hydroxyl radical activity in thermo-catalytically bleached root-filled teeth*, Endod Dent Traumatol 1997;13: 119.
56. Davis MC, Walton RE, Rivera EM. *Sealer distribution in coronal dentin*, J Endod 2002;28: 464.
57. de Geus JL, Wambier LM, Kossatz S, et al: *At-home vs in-office bleaching: a systematic review and meta-analysis*, Oper Dent 2016;41: 341.
58. Demarco FF, Freitas JM, Silva MP, et al: *Microleakage in endodontically treated teeth: influence of calcium hydroxide dressing following bleaching*, Int Endod J 2001;34: 495.
59. Discacciati JA, de Souza EL, Costa SC, et al: *Invasive cervical resorption: etiology, diagnosis, classification and treatment*, J Contemp Dent Pract 2012;13: 723.
60. Dishman MV, Covey DA, Baughan LW. *The effects of peroxide bleaching on composite to enamel bond strength*, Dent Mater 1994;10: 33.
61. do Rego MV, dos Santos RM, Leal LM, et al: *Evaluation of the influence of dental bleaching with 35% hydrogen peroxide in orthodontic bracket shear bond strength*, Dental Press J Orthod 2013;18: 95.
62. Eimar H, Siciliano R, Abdallah MN, et al: *Hydrogen peroxide whitens teeth by oxidizing the organic structure*, J Dent Suppl 2, 2012;40: e25.
63. Esmaeili B, Abolghasemzadeh F, Gholampor A, et al: *The effect of home bleaching carbamide peroxide concentration on the microhardness of dental composite*

Telegram: @dental_k

www.konkur.in

resins, Gen Dent 2018;66: 40.
64. Estevez R, Aranguren J, Escorial A, et al: *Invasive cervical resorption class III in a maxillary central incisor: diagnosis and follow-up by means of cone-beam computed tomography*, J Endod 2010;36: 2012.
65. Fasanaro TS. *Bleaching teeth: history, chemicals, and methods used for common tooth discolorations*, J Esthet Dent 1992;4: 71.
66. Fekrazad R, Alimazandarani S, Kalhori KA, et al: *Comparison of laser and power bleaching techniques in tooth color change*, J Clin Exp Dent 2017;9: e511.
67. Feiglin B. *A 6-year recall study of clinically chemically bleached teeth*, Oral Surg Oral Med Oral Pathol 1987;63: 610.
68. Fields JP. *Intracoronal bleaching of tetracycline-stained teeth: a case report*, J Endod 1982;8: 512.
69. Floyd RA. *The effect of peroxides and free radicals on body tissues*, J Am Dent Assoc Suppl, 1997;128: 37S.
70. Fonseca RB, Sobrinho LC, Neto AJ, et al: *Enamel hypoplasia or amelogenesis imperfecta – a restorative approach*, Braz J of Oral Sci 2006;5: 941.
71. Freccia WF, Peters DD, Lorton L, et al: *An in vitro comparison of nonvital bleaching techniques in the discolored tooth*, J Endod 1982;8: 70.
72. Friedman S. *Surgical-restorative treatment of bleaching-related external root resorption*, Endod Dent Traumatol 1989;5: 63.
73. Friedman S. *Internal bleaching: long-term outcomes and complications*, J Am Dent Assoc Suppl, 1997;128: 51S.
74. Friedman S, Rotstein I, Libfeld H, et al: *Incidence of external root resorption and esthetic results in 58 bleached pulpless teeth*, Endod Dent Traumatol 1988;4: 23.

Telegram: @dental_k

www.konkur.in

75. Frysh H, Bowles WH, Baker F, et al: *Effect of pH on bleaching efficiency*, J Dent Res 1993;72: 384.
76. Frysh H, Bowles WH, Baker F, et al: *Effect of pH on hydrogen peroxide bleaching agents*, J Esthet Dent 1995;7: 130.
77. Gadhia K, McDonald S, Arkutu N, et al: *Amelogenesis imperfecta: an introduction*, Br Dent J 2012;212: 377.
78. Garcia-Godoy F, Dodge WW, Donohue M, et al: *Composite resin bond strength after enamel bleaching*, Oper Dent 1993;18: 144.
79. Gelhard TB, ten Cate JM, Arends J. *Rehardening of artificial enamel lesions in vivo*, Caries Res 1979;13: 80.
80. Gimlin DR, Schindler WG. *The management of postbleaching cervical resorption*, J Endod 1990;16: 292.
81. Glockner K, Hulla H, Ebeleseder K, et al: *Five-year follow-up of internal bleaching*, Braz Dent J 1999;10: 105.
82. Gokay O, Yilmaz F, Akin S, et al: *Penetration of the pulp chamber by bleaching agents in teeth restored with various restorative materials*, J Endod 2000;26: 92.
83. Goldstein RE. *In-office bleaching: where we came from, where we are today*, J Am Dent Assoc Suppl, 1997;128: 11S.
84. Goldstein RE, Garber DA, Goldstein CE, et al: *Esthetic update: the changing esthetic dental practice*, J Am Dent Assoc 1994;125: 1447.
85. Goldstein RE, Lancaster JS. *Survey of patient attitudes toward current esthetic procedures*, J Prosthet Dent 1984;52: 775.
86. Goon WW, Cohen S, Borer RF. *External cervical root resorption following bleaching*, J Endod 1986;12: 414.
87. Grazioli G, Valente LL, Isolan CP, et al: *Bleaching and enamel surface interactions resulting from the use of*

Telegram: @dental_k

www.konkur.in

highly-concentrated bleaching gels, Arch Oral Biol 2018;87: 157.

88. Griffin RE Jr, Grower MF, Ayer WA. *Effects of solutions used to treat dental fluorosis on permeability of teeth*, J Endod 1977;3: 139.
89. Grisham MB. *Oxidants and free radicals in inflammatory bowel disease*, Lancet 1994;344: 859.
90. Gupta A, Dhingra R, Chaudhuri P, et al: *A comparison of various minimally invasive techniques for the removal of dental fluorosis stains in children*, J Indian Soc Pedod Prev Dent 2017;35: 260.
91. Gutierrez JH, Guzman M. *Tooth discoloration in endodontic procedures*, Oral Surg Oral Med Oral Pathol 1968;26: 706.
92. Halliwell B, Clement MV, Ramalingam J, et al: *Hydrogen peroxide. Ubiquitous in cell culture and in vivo?*, IUBMB Life 2000;50: 251.
93. Hansen-Bayless J, Davis R. *Sealing ability of two intermediate restorative materials in bleached teeth*, Am J Dent 1992;5: 151.
94. Harrington GW, Natkin E. *External resorption associated with bleaching of pulpless teeth*, J Endod 1979;5: 344.
95. Hayashi K, Takamizu M, Momoi Y, et al: *Bleaching teeth discolored by tetracycline therapy*, Dent Surv 1980;56: 17.
96. Haywood VB. *History, safety, and effectiveness of current bleaching techniques and applications of the nightguard vital bleaching technique*, Quintessence Int 1992;23: 471.
97. Haywood VB. *Achieving, maintaining, and recovering successful tooth bleaching*, J Esthet Dent 1996;8: 31.
98. Haywood VB. *Nightguard vital bleaching: current concepts and research*, J Am Dent Assoc Suppl, 1997;128: 19S.

Telegram: @dental_k

www.konkur.in

99. Haywood VB. *Current status of nightguard vital bleaching*, Compend Contin Educ Dent Suppl 28, 2000; S10.
100. Haywood VB, Heymann HO. *Nightguard vital bleaching*, Quintessence Int 1989;20: 173.
101. Haywood VB, Heymann HO. *Nightguard vital bleaching: how safe is it?*, Quintessence Int 1991;22: 515.
102. Haywood VB, Leonard RH, Dickinson GL. *Efficacy of six months of nightguard vital bleaching of tetracycline-stained teeth*, J Esthet Dent 1997;9: 13.
103. Heithersay GS. *Combined endodontic-orthodontic treatment of transverse root fractures in the region of the alveolar crest*, Oral Surg Oral Med Oral Pathol 1973;36: 404.
104. Heithersay GS, Dahlstrom SW, Marin PD. *Incidence of invasive cervical resorption in bleached root-filled teeth*, Aust Dent J 1994;39: 82.
105. Heling I, Parson A, Rotstein I. *Effect of bleaching agents on dentin permeability to Streptococcus faecalis*, J Endod 1995;21: 540.
106. Herrin JR, Squier CA, Rubright WC. *Development of erosive gingival lesions after use of a home care technique*, J Periodontol 1987;58: 785.
107. Hodosh M, Mirman M, Shklar G, et al: *A new method of bleaching discolored teeth by the use of a solid state direct heating device*, Dent Dig 1970;76: 344.
108. Holmstrup G, Palm AM, Lambjerg-Hansen H. *Bleaching of discoloured root-filled teeth*, Endod Dent Traumatol 1988;4: 197.
109. Homayouni H, Majd NM, Zohrehei H, et al: *The effect of root canal irrigation with combination of sodium hypo-chlorite and chlorhexidine gluconate on the sealing ability*

Telegram: @dental_k

www.konkur.in

of obturation materials, Open Dent J 2014;8: 184.
110. Horn DJ, Hicks ML, Bulan-Brady J. *Effect of smear layer removal on bleaching of human teeth in vitro*, J Endod 1998;24: 791.
111. Hosoya N, Cox CF, Arai T, et al: *The walking bleach procedure: an in vitro study to measure microleakage of five temporary sealing agents*, J Endod 2000;26: 716.
112. Howell RA. *Bleaching discoloured root-filled teeth*, Br Dent J 1980;148: 159.
113. Howell RA. *The prognosis of bleached root-filled teeth*, Int Endod J 1981;14: 22.
114. Jacobovitz M, de Lima RK. *Treatment of inflammatory internal root resorption with mineral trioxide aggregate: a case report*, Int Endod J 2008;41: 905.
115. Joiner A. *The bleaching of teeth: a review of the literature*, J Dent 2006;34: 412.
116. Jorgensen MG, Carroll WB. *Incidence of tooth sensitivity after home whitening treatment*, J Am Dent Assoc 2002;133: 1076.
117. Josey AL, Meyers IA, Romaniuk K, et al: *The effect of a vital bleaching technique on enamel surface morphology and the bonding of composite resin to enamel*, J Oral Rehabil 1996;23: 244.
118. Kalili KT, Caputo AA, Yoshida K. *Effect of alcohol of composite bond strength to bleached enamel*, J Dent Res special issue, 1993;72: 285.
119. Kahler B, Rossi-Fedele G. *A review of tooth discoloration after regenerative endodontic therapy*, J Endod 2016;42: 563.
120. Karabucak B, Li D, Lim J, et al: *Vital pulp therapy with mineral trioxide aggregate*, Dent Traumatol 2005;21: 240.
121. Kaya AD, Turkun M, Arici M. *Reversal of compromised*

Telegram: @dental_k

www.konkur.in

bonding in bleached enamel using antioxidant gel, Oper Dent 2008;33: 441.

122. Kehoe JC. *pH reversal following in vitro bleaching of pulpless teeth*, J Endod 1987;13: 6.
123. Kendell RL. *Hydrochloric acid removal of brown fluorosis stains: clinical and scanning electron micrographic observations*, Quintessence Int 1989;20: 837.
124. Kidd EA, Joyston-Bechal S, Smith MM. *Staining of residual caries under freshly-packed amalgam restorations exposed to tea/chlorhexidine in vitro*, Int Dent J 1990;40: 219.
125. Kihn PW, Barnes DM, Romberg E, et al: *A clinical evaluation of 10 percent vs. 15 percent carbamide peroxide tooth-whitening agents*, J Am Dent Assoc 2000;131: 1478.
126. Kim JH, Kim Y, Shin SJ, et al: *Tooth discoloration of immature permanent incisor associated with triple antibiotic therapy: a case report*, J Endod 2010;36: 1086.
127. Kim ST, Abbott PV, McGinley P. *The effects of Ledermix paste on discolouration of mature teeth*, Int Endod J 2000;33: 227.
128. Kina JF, Huck C, Riehl H, et al: *Response of human pulps after professionally applied vital tooth bleaching*, Int Endod J 2010;43: 572.
129. Kinomoto Y, Carnes DL Jr, Ebisu S. *Cytotoxicity of intracanal bleaching agents on periodontal ligament cells in vitro*, J Endod 2001;27: 574.
130. Kirchhoff AL, Raldi DP, Salles AC, et al: *Tooth discolouration and internal bleaching after the use of triple antibiotic paste*, Int Endod J 2015;48: 1181.
131. Klaric Sever E, Budimir Z, Cerovac M, et al: *Clinical and*

Telegram: @dental_k

www.konkur.in

patient reported outcomes of bleaching effectiveness, Acta Odontol Scand 2018;76: 30.

132. Kleter GA, Damen JJ, Buijs MJ, et al: *Modification of amino acid residues in carious dentin matrix*, J Dent Res 1998;77: 488.
133. Kolosowski KP, Sodhi RN, Kishen A, et al: *Qualitative analysis of precipitate formation on the surface and in the tubules of dentin irrigated with sodium hypochlorite and a final rinse of chlorhexidine or QMiX*, J Endod 2014;40: 2036.
134. Kopp RS. *A safe, simplified bleaching technic for pulpless teeth*, Dent Surv 1973;49: 42.
135. Koulaouzidou E, Lambrianidis T, Beltes P, et al: *Role of cementoenamel junction on the radicular penetration of 30% hydrogen peroxide during intracoronal bleaching in vitro*, Endod Dent Traumatol 1996;12: 146.
136. Kravitz LH, Tyndall DA, Bagnell CP, et al: *Assessment of external root resorption using digital subtraction radiography*, J Endod 1992;18: 275.
137. Kugel G, Gerlach RW, Aboushala A, et al: *Long-term use of 6.5% hydrogen peroxide bleaching strips on tetracycline stain: a clinical study*, Compend Contin Educ Dent 2011;32: 50.
138. Kugel G, Papathanasiou A, Williams AJ 3rd, et al: *Clinical evaluation of chemical and light-activated tooth whitening systems*, Compend Contin Educ Dent 2006;27: 54.
139. Kupietzky A, Waggoner WF, Galea J. *The clinical and radiographic success of bonded resin composite strip crowns for primary incisors*, Pediatr Dent 2003;25: 577.
140. Kutuk ZB, Ergin E, Cakir FY, et al: *Effects of in-office bleaching agent combined with different desensitizing*

Telegram: @dental_k

www.konkur.in

agents on enamel, J Appl Oral Sci 2018;8: e20180233.
141. Lado EA, Stanley HR, Weisman MI. *Cervical resorption in bleached teeth*, Oral Surg Oral Med Oral Pathol 1983;55: 78.
142. Lai SC, Tay FR, Cheung GS, et al: *Reversal of compromised bonding in bleached enamel*, J Dent Res 2002;81: 477.
143. Lake FT, O'Dell NL, Walton RE. *The effect of internal bleaching on tetracycline in dentin*, J Endod 1985;11: 415.
144. Latcham NL. *Management of a patient with severe postbleaching cervical resorption: a clinical report*, J Prosthet Dent 1991;65: 603.
145. Lee GP, Lee MY, Lum SO, et al: *Extraradicular diffusion of hydrogen peroxide and pH changes associated with intracoronal bleaching of discoloured teeth using different bleaching agents*, Int Endod J 2004;37: 500.
146. Lee SS, Zhang W, Lee DH, et al: *Tooth whitening in children and adolescents: a literature review*, Pediatr Dent 2005;27: 362.
147. Lemon RR. *Bleaching and restoring endodontically treated teeth*, Curr Opin Dent 1991;1: 754.
148. Leonard RH Jr. *Efficacy, longevity, side effects, and patient perceptions of nightguard vital bleaching*, Compend Contin Educ Dent 1998;19: 766.
149. Leonard RH Jr. *Long-term treatment results with nightguard vital bleaching*, Compend Contin Educ Dent 4A, 2003;24: 364.
150. Leonard RH Jr, Haywood V, Phillips C. *Risk factors for developing tooth sensitivity and gingival irritation associated with nightguard vital bleaching*, Quintessence Int 1997;28: 527.
151. Leonard RH, Sharma A, Haywood VB. *Use of different*

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concentrations of carbamide peroxide for bleaching teeth: an in vitro study, Quintessence Int 1998;29: 503.

152. Lewinstein I, Hirschfeld Z, Stabholz A, et al: *Effect of hydrogen peroxide and sodium perborate on the microhardness of human enamel and dentin*, J Endod 1994;20: 61.
153. Li Y. *Biological properties of peroxide-containing tooth whiteners*, Food Chem Toxicol 1996;34: 887.
154. Li Y. *Toxicological considerations of tooth bleaching using peroxide-containing agents*, J Am Dent Assoc Suppl, 1997;128: 31S.
155. Li Y. *Tooth bleaching using peroxide-containing agents: current status of safety issues*, Compend Contin Educ Dent 1998;19: 783.
156. Li Y. *The safety of peroxide-containing at-home tooth whiteners*, Compend Contin Educ Dent 4A, 2003;24: 384.
157. Li Y. *Safety controversies in tooth bleaching*, Dent Clin North Am 2011;55: 255.
158. Lim MY, Lum SO, Poh RS, et al: *An in vitro comparison of the bleaching efficacy of 35% carbamide peroxide with established intracoronal bleaching agents*, Int Endod J 2004;37: 483.
159. Lopes GC, Bonissoni L, Baratieri LN, et al: *Effect of bleaching agents on the hardness and morphology of enamel*, J Esthet Restor Dent 2002;14: 24.
160. Lowney JJ. *Simplified technique for bleaching a discoloured tooth*, Dent Dig 1964;70: 446.
161. Madison S, Walton R. *Cervical root resorption following bleaching of endodontically treated teeth*, J Endod 1990;16: 570.
162. Marin PD, Bartold PM, Heithersay GS. *Tooth discoloration by blood: an in vitro histochemical study*,

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Endod Dent Traumatol 1997;13: 132.
163. Matthews JD Jr. *Pink teeth resulting from Russian endodontic therapy*, J Am Dent Assoc 2000;131: 1598.
164. McCormick JE, Weine FS, Maggio JD. *Tissue pH of developing periapical lesions in dogs*, J Endod 1983;9: 47.
165. McInerney ST, Zillich R. *Evaluation of internal sealing ability of three materials*, J Endod 1992;18: 376.
166. Meister F Jr, Haasch GC, Gerstein H. *Treatment of external resorption by a combined endodontic-periodontic procedure*, J Endod 1986;12: 542.
167. Mondelli RF, Soares AF, Pangrazio EG, et al: *Evaluation of temperature increase during in-office bleaching*, J Appl Oral Sci 2016;24: 136.
168. Montgomery S. *External cervical resorption after bleaching a pulpless*, Oral Surg Oral Med Oral Pathol 1984;57: 203.
169. Moosavi H, Ghavamnasiri M, Manari V. *Effect of postoperative bleaching on marginal leakage of resin composite and resin-modified glass ionomer restorations at different delayed periods of exposure to carbamide peroxide*, J Contemp Dent Pract 2009;10: E009.
170. Muller CJ, van Wyk CW. *The amelo-cemental junction*, J Dent Assoc S Afr 1984;39: 799.
171. Mullins JM, Kao EC, Martin CA, et al: *Tooth whitening effects on bracket bond strength in vivo*, Angle Orthod 2009;79: 777.
172. Murchison DF, Charlton DG, Moore BK. *Carbamide peroxide bleaching: effects on enamel surface hardness and bonding*, Oper Dent 1992;17: 181.
173. Naik S, Hegde AH. *Mineral trioxide aggregate as a pulpotomy agent in primary molars: an in vivo study*, J Indian Soc Pedod Prev Dent 2005;23: 13.

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174. Nathoo S, Stewart B, Petrone ME, et al: *Comparative clinical investigation of the tooth whitening efficacy of two tooth whitening gels*, J Clin Dent 2003;14: 64.
175. Nathoo SA. *The chemistry and mechanisms of extrinsic and intrinsic discoloration*, J Am Dent Assoc Suppl, 1997;128: 6S.
176. Nathoo SA, Gaffar A. *Studies on dental stain induced by antibacterial agents and rationale approaches for bleaching dental stains*, Adv Dent Res 1994;9: 462.
177. Neuvald L, Consolaro A. *Cementoenamel junction: microscopic analysis and external cervical resorption*, J Endod 2000;26: 503.
178. Niederman R, Ferguson M, Urdaneta R, et al: *Evidence-based esthetic dentistry*, J Esthet Dent 1998;10: 229.
179. Nutting EB, Poe GS. *A new combination for bleaching teeth*, J South Californian Dent Assoc 1963;31: 289.
180. Nutting EB, Poe GS. *Chemical bleaching of discolored endodontically treated teeth*, Dent Clin North Am 1967;655:.
181. Parsons JR, Walton RE, Ricks-Williamson L. *In vitro longitudinal assessment of coronal discoloration from endodontic sealers*, J Endod 2001;27: 699.
182. Partovi M, Al-Havvaz AH, Soleimani B. *In vitro computer analysis of crown discolouration from commonly used endodontic sealers*, Aust Endod J 2006;32: 116.
183. Pashley DH, Livingston MJ. *Effect of molecular size on permeability coefficients in human dentin*, Arch Oral Biol 1978;23: 391.
184. Pashley DH, Thompson SM, Stewart FP. *Dentin permeability: effects of temperature on hydraulic conductance*, J Dent Res 1983;62: 956.
185. Pavicic DK, Kolceg M, Lajnert V, et al: *Changes in*

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quality of life induced by tooth whitening are moderated by perfectionism: a randomized, double-blind, placebo-controlled trial, Int J Prosthodont 2018;31: 394.

186. Pedrollo Lise D, Siedschlag G, Bernardon JK, et al: *Randomized clinical trial of 2 nonvital tooth bleaching techniques: A 1-year follow-up*, J Prosthet Dent 2018;119: 53.
187. Peixoto AC, Vaez SC, Pereira NAR, et al: *High-concentration carbamide peroxide can reduce the sensitivity caused by in-office tooth bleaching: a single-blinded randomized controlled trial*, J Appl Oral Sci 2018;26: e20170573.
188. Peng JJ, Botelho MG, Matinlinna JP. *Silver compounds used in dentistry for caries management: a review*, J Dent 2012;40: 531.
189. Plotino G, Buono L, Grande NM, et al: *Nonvital tooth bleaching: a review of the literature and clinical procedures*, J Endod 2008;34: 394.
190. Pohjola RM, Browning WD, Hackman ST, et al: *Sensitivity and tooth whitening agents*, J Esthet Restor Dent 2002;14: 85.
191. Powell LV, Bales DJ. *Tooth bleaching: its effect on oral tissues*, J Am Dent Assoc 1991;122: 50.
192. Price RB, Loney RW, Doyle MG, et al: *An evaluation of a technique to remove stains from teeth using microabrasion*, J Am Dent Assoc 2003;134: 1066.
193. Pugh G Jr, Zaidel L, Lin N, et al: *High levels of hydrogen peroxide in overnight tooth-whitening formulas: effects on enamel and pulp*, J Esthet Restor Dent 2005;17: 40.
194. Reyto R. *Laser tooth whitening*, Dent Clin North Am 1998;42: 755.
195. Rotstein I. *Role of catalase in the elimination of residual*

Telegram: @dental_k

www.konkur.in

hydrogen peroxide following tooth bleaching, J Endod 1993;19: 567.

196. Rotstein I. *Tooth discoloration and bleaching*, Ingle JIB Bakland LK Endodontics ed 5 2002; BC Decker Hamilton, Ontario, Canada 845-860.
197. Rotstein I, Dankner E, Goldman A, et al: *Histochemical analysis of dental hard tissues following bleaching*, J Endod 1996;22: 23.
198. Rotstein I, Dogan H, Avron Y, et al: *Mercury release from dental amalgam after treatment with 10% carbamide peroxide in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 216.
199. Rotstein I, Lehr Z, Gedalia I. *Effect of bleaching agents on inorganic components of human dentin and cementum*, J Endod 1992;18: 290.
200. Rotstein I, Lewinstein I, Zuwabi O, et al: *Effect of cervical coating of ethyl cellulose polymer and metacrylic acid copolymer on the radicular penetration of hydrogen peroxide during bleaching*, Endod Dent Traumatol 1992;8: 202.
201. Rotstein I, Mor C, Arwaz JR. *Changes in surface levels of mercury, silver, tin, and copper of dental amalgam treated with carbamide peroxide and hydrogen peroxide in vitro*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83: 506.
202. Rotstein I, Mor C, Friedman S. *Prognosis of intracoronal bleaching with sodium perborate preparation in vitro: 1-year study*, J Endod 1993;19: 10.
203. Rotstein I, Torek Y, Lewinstein I. *Effect of bleaching time and temperature on the radicular penetration of hydrogen peroxide*, Endod Dent Traumatol 1991;7: 196.
204. Rotstein I, Torek Y, Misgav R. *Effect of cementum*

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defects on radicular penetration of 30% H2O2 during intracoronal bleaching, J Endod 1991;17: 230.

205. Rotstein I, Wesselink PR, Bab I. *Catalase protection against hydrogen peroxide-induced injury in rat oral mucosa*, Oral Surg Oral Med Oral Pathol 1993;75: 744.
206. Rotstein I, Zalkind M, Mor C, et al: *In vitro efficacy of sodium perborate preparations used for intracoronal bleaching of discolored non-vital teeth*, Endod Dent Traumatol 1991;7: 177.
207. Rotstein I, Zyskind D, Lewinstein I, et al: *Effect of different protective base materials on hydrogen peroxide leakage during intracoronal bleaching in vitro*, J Endod 1992;18: 114.
208. Salvas CJ. *Perborate as a bleaching agent*, J Am Dent Assoc 1938;25: 324.
209. Sasaki RT, Arcanjo AJ, Florio FM, et al: *Micromorphology and microhardness of enamel after treatment with home-use bleaching agents containing 10% carbamide peroxide and 7.5% hydrogen peroxide*, J Appl Oral Sci 2009;17: 611.
210. Sayed M, Matsui N, Hiraishi N, et al: *Effect of glutathione bio-molecule on tooth discoloration associated with silver diammine fluoride*, Int J Mol Sci 2018;29: 19.
211. Schulte JR, Morrissette DB, Gasior EJ, et al: *Clinical changes in the gingiva as a result of at-home bleaching*, Compendium 1993;14: 1362.
212. Schwandt NW, Gound TG. *Resorcinol-formaldehyde resin "Russian Red" endodontic therapy*, J Endod 2003;29: 435.
213. Serene TP, Snyder DE. *Bleaching technique (pulpless anterior teeth)*, J South Calif Dent Assoc 1973;41: 30.
214. Shahrami FZ, Zaree M, Bejeh M, et al: *Comparison of*

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tooth crown discoloration with Epiphany and AH26 sealer in terms of chroma and value: an in vitro study, Brazilian J Oral Sci 2011;10: 171.

215. Shinohara MS, Peris AR, Rodrigues JA, et al: *The effect of nonvital bleaching on the shear bond strength of composite resin using three adhesive systems*, J Adhes Dent 2004;6: 205.
216. Shinohara MS, Rodrigues JA, Pimenta LA. *In vitro microleakage of composite restorations after nonvital bleaching*, Quintessence Int 2001;32: 413.
217. Sibilla P, Cogo E, Turrini R, et al: *The timing and operational management of the variables of bleaching in cases of rehabilitation in the esthetic field*, Int J Esthet Dent 2014;9: 436.
218. Smith JJ, Cunningham CJ, Montgomery S. *Cervical canal leakage after internal bleaching*, J Endod 1992;18: 476.
219. Soares DG, Basso FG, Scheffel DS, et al: *Responses of human dental pulp cells after application of a low-concentration bleaching gel to enamel*, Arch Oral Biol 2015;60: 1428.
220. Spasser HF. *A simple bleaching technique using sodium perborate*, N Y State Dent J 1961;27: 332.
221. Steiner DR, West JD. *A method to determine the location and shape of an intracoronal bleach barrier*, J Endod 1994;20: 304.
222. Subay RK, Ilhan B, Ulukapi H. *Mineral trioxide aggregate as a pulpotomy agent in immature teeth: long-term case report*, Eur J Dent 2013;7: 133.
223. Sundfeld RH, Sundfeld-Neto D, Machado LS, et al: *Microabrasion in tooth enamel discoloration defects: three cases with long-term follow-ups*, J Appl Oral Sci 2014;22:

Telegram: @dental_k

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347.
224. Suyama Y, Otsuki M, Ogisu S, et al: *Effects of light sources and visible light-activated titanium dioxide photocatalyst on bleaching*, Dent Mater J 2009;28: 693.
225. Swift EJ Jr. *Restorative considerations with vital tooth bleaching*, J Am Dent Assoc 1997;128(Suppl): 60S.
226. Swift EJ Jr, Perdigao J. *Effects of bleaching on teeth and restorations*, Compend Contin Educ Dent 1998;19: 815.
227. Tavares M, Stultz J, Newman M, et al: *Light augments tooth whitening with peroxide*, J Am Dent Assoc 2003;134: 167.
228. Teixeira EC, Hara AT, Serra MC. *Use of 37% carbamide peroxide in the walking bleach technique: a case report*, Quintessence Int 2004;35: 97.
229. Ten Cate AR. Oral histology: development, structure, and function ed 2 1985; Mosby St. Louis, MO.
230. Tewari A, Chawla HS. *Bleaching of non-vital discoloured anterior teeth*, J Indian Dent Assoc 1972;44: 130.
231. Thylstrup AF, Fejerskov O. *Clinical and pathological features of dental caries*, Thylstrup AF Fejerskov O Textbook of clinical cariology ed 2 1995; Munksgaard Copenhagen.
232. Tinaz AC, Kivanc BH, Gorgul G. *Staining potential of calcium hydroxide and monochlorophenol following removal of AH26 root canal sealer*, J Contemp Dent Pract 2008;9: 56.
233. Titley KC, Torneck CD, Ruse ND. *The effect of carbamide-peroxide gel on the shear bond strength of a microfil resin to bovine enamel*, J Dent Res 1992;71: 20.
234. Titley KC, Torneck CD, Ruse ND, et al: *Adhesion of a resin composite to bleached and unbleached human*

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enamel, J Endod 1993;19: 112.
235. Titley KC, Torneck CD, Smith DC, et al: *Adhesion of composite resin to bleached and unbleached bovine enamel*, J Dent Res 1988;67: 1523.
236. Titley KC, Torneck CD, Smith DC, et al: *Adhesion of a glass ionomer cement to bleached and unbleached bovine dentin*, Endod Dent Traumatol 1989;5: 132.
237. Tong LS, Pang MK, Mok NY, et al: *The effects of etching, micro-abrasion, and bleaching on surface enamel*, J Dent Res 1993;72: 67.
238. Torabinejad M, Cho Y, Khademi AA, et al: *The effect of various concentrations of sodium hypochlorite on the ability of MTAD to remove the smear layer*, J Endod 2003;29: 233.
239. Torneck CD, Titley KC, Smith DC, et al: *The influence of time of hydrogen peroxide exposure on the adhesion of composite resin to bleached bovine enamel*, J Endod 1990;16: 123.
240. Torneck CD, Titley KC, Smith DO, et al: *Effect of water leaching the adhesion of composite resin to bleached and unbleached bovine enamel*, J Endod 1991;17: 156.
241. Tran L, Orth R, Parashos P, et al: *Depletion rate of hydrogen peroxide from sodium perborate bleaching agent*, J Endod 2017;43: 472.
242. Tredwin CJ, Naik S, Lewis NJ, et al: *Hydrogen peroxide tooth-whitening (bleaching) products: review of adverse effects and safety issues*, Br Dent J 2006;200: 371.
243. Tronstad L, Andreasen JO, Hasselgren G, et al: *pH changes in dental tissues after root canal filling with calcium hydroxide*, J Endod 1981;7: 17.
244. Trope M. *Cervical root resorption*, J Am Dent Assoc 1997;128(Suppl): 56S.

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245. Ubaldini AL, Baesso ML, Medina Neto A, et al: *Hydrogen peroxide diffusion dynamics in dental tissues*, J Dent Res 2013;92: 661.
246. Unlu N, Cobankara FK, Ozer F. *Effect of elapsed time following bleaching on the shear bond strength of composite resin to enamel*, J Biomed Mater Res B Appl Biomater 2008;84: 363.
247. Uthappa R, Suprith ML, Bhandary S, et al: *A comparative study of different bleaching agents on the morphology of human enamel: an in vitro SEM study*, J Contemp Dent Pract 2012;13: 756.
248. Vachon C, Vanek P, Friedman S. *Internal bleaching with 10% carbamide peroxide in vitro*, Pract Periodontics Aesthet Dent 1998;10: 1145.
249. Vaes G. *On the mechanisms of bone resorption. The action of parathyroid hormone on the excretion and synthesis of lysosomal enzymes and on the extracellular release of acid by bone cells*, J Cell Biol 1968;39: 676.
250. van der Burgt TP, Plasschaert AJ. *Tooth discoloration induced by dental materials*, Oral Surg Oral Med Oral Pathol 1985;60: 666.
251. Visram S, McKaig S. *Amelogenesis imperfecta—clinical presentation and management: a case report*, Dent Update 2006;33: 612-614.
252. Vivacqua-Gomes N, Ferraz CC, Gomes BP, et al: *Influence of irrigants on the coronal microleakage of laterally condensed gutta-percha root fillings*, Int Endod J 2002;35: 791.
253. Vogel RI. *Intrinsic and extrinsic discoloration of the dentition (a literature review)*, J Oral Med 1975;30: 99.
254. Waite RM, Carnes DL Jr, Walker WA 3rd. *Microleakage of TERM used with sodium perborate/water and sodium*

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www.konkur.in

perborate/superoxol in the "walking bleach" technique, J Endod 1998;24: 648.

255. Walsh LJ, Athanassiadis B. *Endodontic aesthetic iatrodontics*, Australasian Dental Practice 2007;18: 62.
256. Walton RE, O'Dell NL, Lake FT, et al: *Internal bleaching of tetracycline-stained teeth in dogs*, J Endod 1983;9: 416.
257. Wang JD, Hume WR. *Diffusion of hydrogen ion and hydroxyl ion from various sources through dentin*, Int Endod J 1988;21: 17.
258. Watts A, Addy M. *Tooth discolouration and staining: a review of the literature*, Br Dent J 2001;190: 309.
259. Watts JD, Holt DM, Beeson TJ, et al: *Effects of pH and mixing agents on the temporal setting of tooth-colored and gray mineral trioxide aggregate*, J Endod 2007;33: 970.
260. Weiger R, Kuhn A, Lost C. *In vitro comparison of various types of sodium perborate used for intracoronal bleaching of discolored teeth*, J Endod 1994;20: 338.
261. Weisman MI. *Efficient bleaching procedure for the pulpless tooth*, Dent Dig 1963;69: 347.
262. Whang HJ, Shin DH. *Effects of applying antioxidants on bond strength of bleached bovine dentin*, Restor Dent Endod 2015;40: 37.
263. White DJ, Kozak KM, Zoladz JR, et al: *Peroxide interactions with hard tissues: effects on surface hardness and surface/subsurface ultrastructural properties*, Compend Contin Educ Dent 2002;23: 42.
264. Wilcox LR, Diaz-Arnold A. *Coronal microleakage of permanent lingual access restorations in endodontically treated anterior teeth*, J Endod 1989;15: 584.
265. Zalkind M, Arwaz JR, Goldman A, et al: *Surface morphology changes in human enamel, dentin and*

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cementum following bleaching: a scanning electron microscopy study, Endod Dent Traumatol 1996;12: 82.

266. Zanolla J, Marques A, da Costa DC, et al: *Influence of tooth bleaching on dental enamel microhardness: a systematic review and meta-analysis*, Aust Dent J 2017;62: 276.

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27: Endodontic records and legal responsibilities

Edwin J. Zinman

CHAPTER OUTLINE

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Endodontic record excellence

Importance

Endodontic therapy records serve as an important map to document and guide the clinician's journey down the correct diagnostic and treatment path. Documentation is essential to attaining endodontic excellence.

The dental record must contain sufficient information to identify the patient, support the diagnosis, justify the treatment, and document the course and result of treatment, and it should be designed to protect the patient's welfare. Records also are fundamental means of communication among health care professionals.

Content

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Endodontic treatment records should include the following information:

1. Name of patient
2. Date of visit
3. Medical (periodically updated) and dental history
4. Allergies and adverse drug reactions
5. Chief complaints
6. Radiographs of diagnostic quality
7. Pulpal and periodontal diagnostic tests performed
8. Clinical examination findings
9. Differential diagnoses and final diagnosis
10. Treatment plan
11. Prognosis
12. Referrals, including patient refusals (if any)
13. Communications with other health care providers
14. Progress notes (including complications)
15. Completion notes
16. Canceled or missed appointments and stated reasons
17. Emergency treatment
18. Patient concerns and dissatisfactions
19. Planned follow-ups
20. Drug and laboratory prescriptions
21. Patient noncompliance
22. Consent and informed refusal forms
23. Accounting ledger
24. Recall notifications
25. Name or initials of author for each treatment entry
26. E-mail address of patient
27. Emergency contact persons, addresses, and phone numbers
28. Patient phone numbers including home, work, and cell
29. Log of records and radiographs received or sent

Office records

1. Sign-in and discharge sheets (optimal)
2. Periodic bacteriological monitoring of waterlines

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3. Continuing Education certificates[a]
4. Office procedure manuals, that is, tray setups
5. Weekly spore testing of autoclaves

Function

Dental records should document the following information:

1. Course of therapy by recorded diagnosis, including differential diagnoses, informed consent or refusal, treatment, and prognosis
2. Communications among the treating clinician, other health care providers, consultants, subsequent treating practitioners, and third-party carriers
3. Necessity and reasonableness of diagnosis and treatment capable of peer review and dental insurance carrier evaluation
4. Standard of care compliance
5. Adverse reactions or events with explanation
6. Follow-up comments to medical history–positive responses

Patient information form

A patient information form provides essential data for patient identification and office communication, which should be updated periodically (Fig. 27.1). The patient's name; home, business, and e-mail addresses; and telephone and fax numbers are needed to contact the patient for scheduling purposes and inquire about postoperative sequelae.[178] Location information about the patient's spouse, relative, or a close friend who can be notified in an emergency is also suggested. In the event the patient is a minor, the responsible parent or guardian should provide the information. Questions about dental insurance and financial responsibility are included on the form to avoid any later misunderstandings and help fulfill federal requirements of the Truth in Lending law, applicable if four or more installment payments are arranged (whether or not there are interest or late-payment charges).[104]

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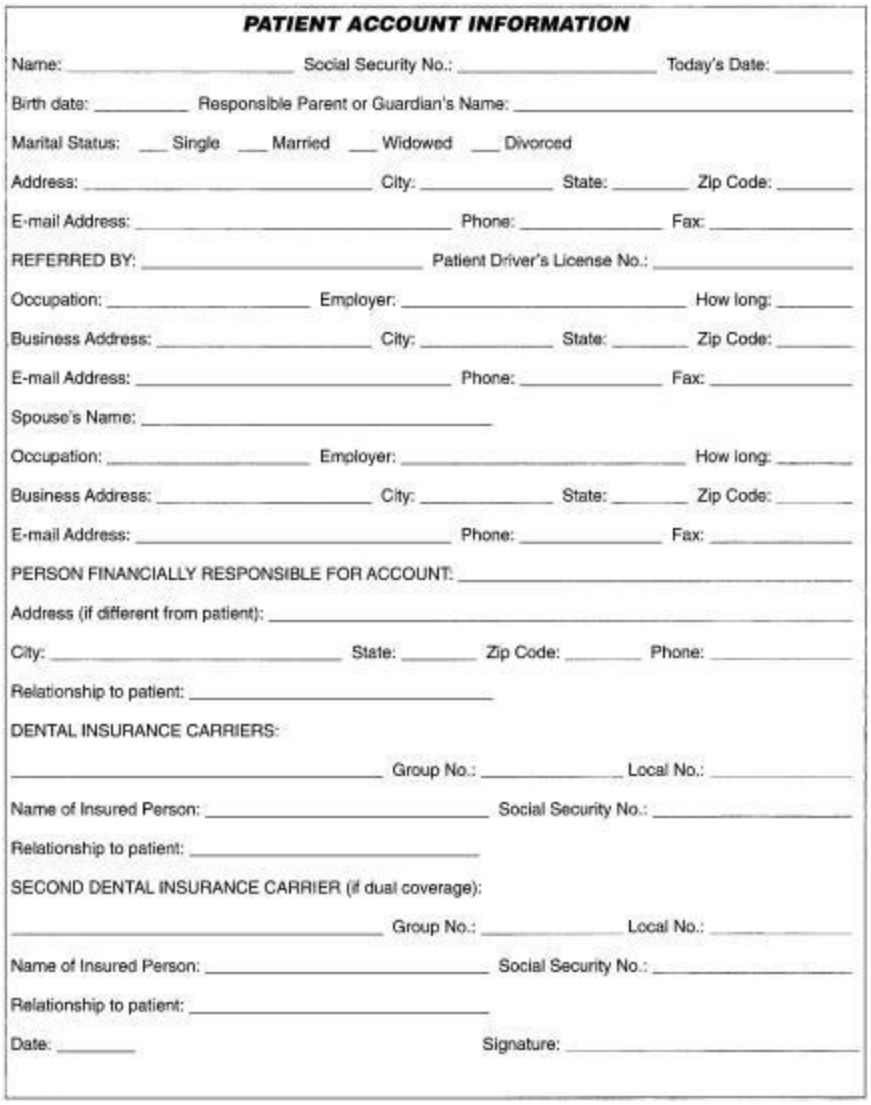

PATIENT ACCOUNT INFORMATION

Name: _____ Social Security No.: _____ Today's Date: _____

Birth date: _____ Responsible Parent or Guardian's Name: _____

Marital Status: ___ Single ___ Married ___ Widowed ___ Divorced

Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

REFERRED BY: _____ Patient Driver's License No.: _____

Occupation: _____ Employer: _____ How long: _____

Business Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

Spouse's Name: _____

Occupation: _____ Employer: _____ How long: _____

Business Address: _____ City: _____ State: _____ Zip Code: _____

E-mail Address: _____ Phone: _____ Fax: _____

PERSON FINANCIALLY RESPONSIBLE FOR ACCOUNT: _____

Address (if different from patient): _____

City: _____ State: _____ Zip Code: _____ Phone: _____

Relationship to patient: _____

DENTAL INSURANCE CARRIERS:

_____ Group No.: _____ Local No.: _____

Name of Insured Person: _____ Social Security No.: _____

Relationship to patient: _____

SECOND DENTAL INSURANCE CARRIER (if dual coverage):

_____ Group No.: _____ Local No.: _____

Name of Insured Person: _____ Social Security No.: _____

Relationship to patient: _____

Date: _____ Signature: _____

FIG. 27.1 Patient information form.

Form, titled patient account information, is as follows:

Name, social security number, today's date, birth date, responsible parent or guardian's name, marital status: (single, married, widowed, or divorced), address, city, state, zip code, e-mail address, phone, fax.

Referred By: Patient driver's license number, occupation, employer, how long, business address, city, state, zip code, e-mail address, phone, fax, spouse's Name, occupation, employer, how long, business address, city, state, zip code, e-mail address, phone, fax.

Person financially responsible for account: Address (if different from patient), city, state, zip Code, phone, relationship to patient.

Dental insurance carriers: Group number, local number, name of insured person, social security number, relationship to patient.

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Second dental insurance carrier (if dual coverage): Group number, local number, name of insured person, social security number, and relationship to patient.

Each option is followed by blank space.

At the bottom, blank spaces for date and signature are given.

Medical health history

The clinician should thoroughly review the patient's past and present health status before proceeding so that dental treatment can be safely initiated. Health questionnaires open avenues for discussion about problems of major organ systems and important biochemical mechanisms, such as blood coagulation, allergy, immunocompromised status, need for antibiotic prophylaxis, and disease susceptibility. The clinician may request that the patient be examined by a physician or undergo laboratory testing under medical supervision to determine whether a suspected medical problem may require attention before endodontic therapy proceeds or if drug sensitivity or an allergy mandates treatment modifications.[43,165,188] Knowledge about current medications, medical therapy, and the name and address of the treating physician is essential.

Medical histories must be updated periodically (or at least annually or sooner, as the need arises). The patient should be asked to review the previous and current medical history (Fig. 27.2). If no changes are necessary, the patient should date and sign the history form. If any changes occurred, the patient should identify each updated medical change, the date, and sign the form where indicated for medical update information. Periodically the patient should provide an entirely new updated form rather than changing data on the old form. Earlier medical histories should be retained in the chart for future reference. If physician communication for treatment occurs, the clinician should record these contacts. In addition, the clinician should verify physician approvals for treatment by fax, with verification of receipt or letter or preferably both, and retain copies in the chart.

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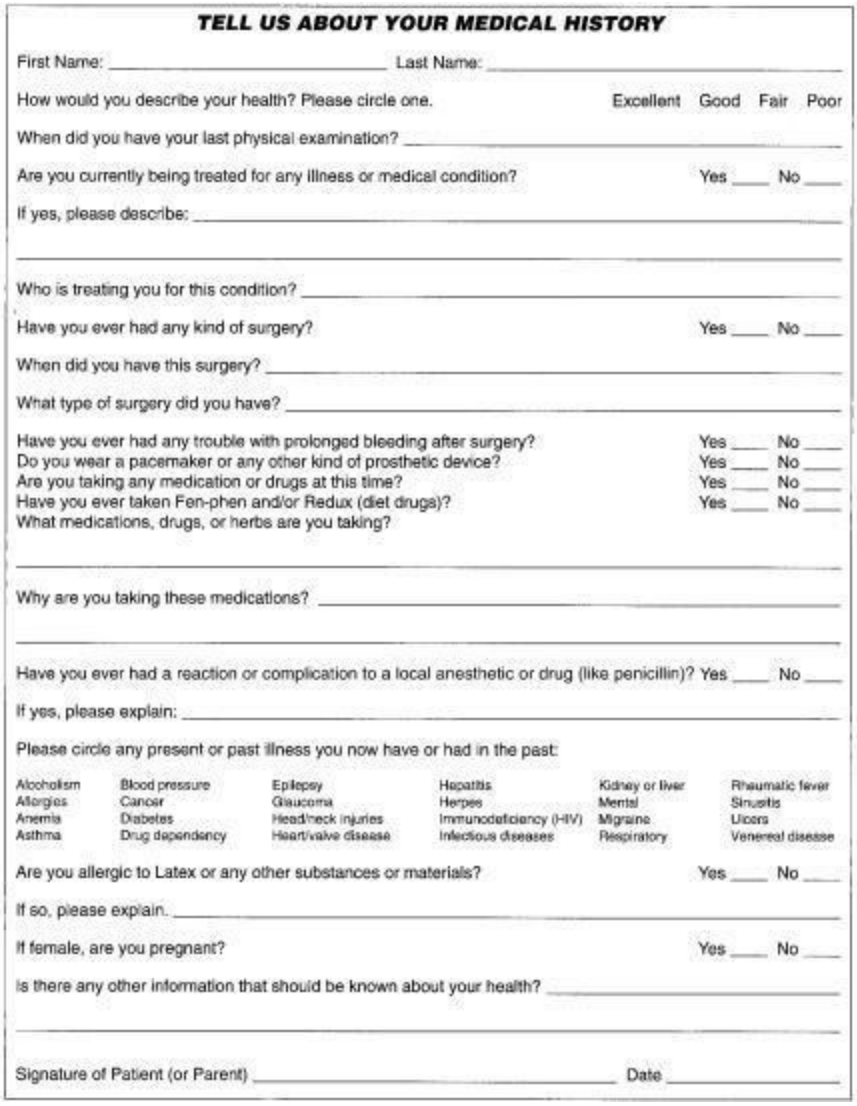

TELL US ABOUT YOUR MEDICAL HISTORY

First Name: _____ Last Name: _____

How would you describe your health? Please circle one. Excellent Good Fair Poor

When did you have your last physical examination? _____

Are you currently being treated for any illness or medical condition? Yes ___ No ___

If yes, please describe: _____

_____

Who is treating you for this condition? _____

Have you ever had any kind of surgery? Yes ___ No ___

When did you have this surgery? _____

What type of surgery did you have? _____

Have you ever had any trouble with prolonged bleeding after surgery? Yes ___ No ___
Do you wear a pacemaker or any other kind of prosthetic device? Yes ___ No ___
Are you taking any medication or drugs at this time? Yes ___ No ___
Have you ever taken Fen-phen and/or Redux (diet drugs)? Yes ___ No ___
What medications, drugs, or herbs are you taking?

_____

Why are you taking these medications? _____

_____

Have you ever had a reaction or complication to a local anesthetic or drug (like penicillin)? Yes ___ No ___

If yes, please explain: _____

Please circle any present or past illness you now have or had in the past:

Alcoholism	Blood pressure	Epilepsy	Hepatitis	Kidney or liver	Rheumatic fever
Allergies	Cancer	Glaucoma	Herpes	Mental	Sinusitis
Anemia	Diabetes	Head/neck injuries	Immunodeficiency (HIV)	Migraine	Ulcers
Asthma	Drug dependency	Heart/valve disease	Infectious diseases	Respiratory	Venereal disease

Are you allergic to Latex or any other substances or materials? Yes ___ No ___

If so, please explain. _____

If female, are you pregnant? Yes ___ No ___

Is there any other information that should be known about your health? _____

_____

Signature of Patient (or Parent) _____ Date _____

FIG. 27.2 Medical history form.

Form, titled tell us about your medical history, is as follows:

First Name, last Name, How would you describe your health? Please circle one (Excellent, good, fair, and poor), When did you have your last physical examination? Are you currently being treated for any illness or medical condition? (Yes or no), If yes, please describe, Who is treating you for this condition? Have you ever had any kind of surgery? (Yes or no), When did you have this surgery? What type of surgery did you have? Have you ever had any trouble with prolonged bleeding after surgery? (Yes or no), do you wear a pacemaker or any other kind of prosthetic device? (Yes or no), Are you taking any medication or drugs at this time? (Yes or no), Have you ever taken Fen-pen and/or Redux (diet drugs)? (Yes or no), What medications, drugs, or herbs are you taking? Why are you taking these medications? Have you ever had a reaction or complication to a local anesthetic or drug (like penicillin)? (Yes or no), If yes, please explain.

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Please circle any present or past illness you now have or had in the past:

Alcoholism, blood pressure, epilepsy, hepatitis, kidney or liver, rheumatic fever, allergies, cancer, glaucoma, herpes, mental, sinusitis, anemia, diabetes, head/neck injuries, immunodeficiency (HIV), migraine, ulcers, asthma, drug dependency, Heart or valve disease, infectious diseases, respiratory, and venereal disease.

Are you allergic to Latex or any other substances or materials? (Yes or no), If so, please explain. If female, are you pregnant? (Yes or no), Is there any other information that should be known about your health?

Each query or option is followed by blank space.

At the bottom, blank spaces for signature of patient (or parent) and date are given.

Updating the medical history requires the practitioner to be apprised of changes in the patient's medical condition and any new medications the patient is taking, including over-the-counter or herbal medications or supplements.[257] A patient untrained in medical science may not appreciate the fact that new medications may suggest new diseases or changes in existing disease status. For instance, certain regurgitating valvular heart diseases may require antibiotic prophylaxis.[334] New medications may also cause a synergistic effect with other medications the patient is using or another treating clinician is prescribing.

Clinicians who pass the buck by claiming, "Oh it was my secretary or my new assistant—I could do nothing about it," are nonetheless legally liable for their staff's actions or inactions.[335] A clinician exclusively relying on staff members to obtain medical histories in a waiting room full of patients is making a mistake. Instead, the clinician must check and then follow up regarding the accuracy of the histories. Training and monitoring staff are duties clinicians cannot afford to ignore. President Harry Truman's sage advice, "The buck stops here," applies to treating clinicians who overdelegate to nonclinician staff members who lack the requisite dental education for adequate medical history follow-up.

Dental history

The dental history should include past dental difficulties, name and address

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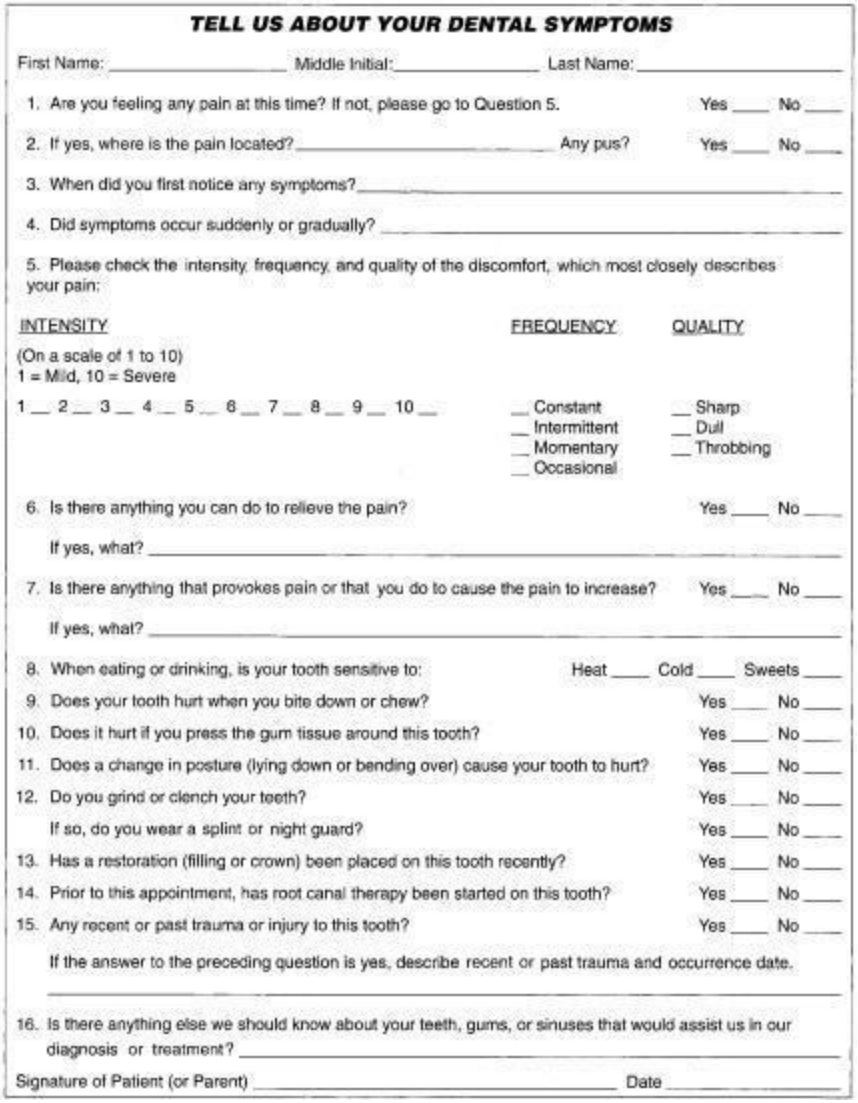
of current or most recent treating clinician, chief complaint (including onset, duration, frequency, type, and intensity of any pain or numbness), relevant prior dental treatment, and attitude regarding teeth retention. Positive responses suggest further patient consultation and consideration for obtaining records (by written release) for elucidation from the patient's previous or present treating clinician.[227] After receiving positive responses, the clinician may also wish to obtain prior and concurrent treaters' radiographs for current comparison with any progressive changes (Fig. 27.3).



TELL US ABOUT YOUR DENTAL SYMPTOMS

First Name: _____ Middle Initial: _____ Last Name: _____

1. Are you feeling any pain at this time? If not, please go to Question 5. Yes ___ No ___
2. If yes, where is the pain located? _____ Any pus? Yes ___ No ___
3. When did you first notice any symptoms? _____
4. Did symptoms occur suddenly or gradually? _____
5. Please check the intensity, frequency, and quality of the discomfort, which most closely describes your pain:

INTENSITY	FREQUENCY	QUALITY
(On a scale of 1 to 10) 1 = Mild, 10 = Severe		
1 __ 2 __ 3 __ 4 __ 5 __ 6 __ 7 __ 8 __ 9 __ 10 __	__ Constant __ Intermittent __ Momentary __ Occasional	__ Sharp __ Dull __ Throbbing

6. Is there anything you can do to relieve the pain? Yes ___ No ___
   If yes, what? _____
7. Is there anything that provokes pain or that you do to cause the pain to increase? Yes ___ No ___
   If yes, what? _____
8. When eating or drinking, is your tooth sensitive to: Heat ___ Cold ___ Sweets ___
9. Does your tooth hurt when you bite down or chew? Yes ___ No ___
10. Does it hurt if you press the gum tissue around this tooth? Yes ___ No ___
11. Does a change in posture (lying down or bending over) cause your tooth to hurt? Yes ___ No ___
12. Do you grind or clench your teeth? Yes ___ No ___
    If so, do you wear a splint or night guard? Yes ___ No ___
13. Has a restoration (filling or crown) been placed on this tooth recently? Yes ___ No ___
14. Prior to this appointment, has root canal therapy been started on this tooth? Yes ___ No ___
15. Any recent or past trauma or injury to this tooth? Yes ___ No ___
    If the answer to the preceding question is yes, describe recent or past trauma and occurrence date.
    _____
16. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis or treatment? _____

Signature of Patient (or Parent) _____ Date _____

FIG. 27.3 Dental history record.

Form, titled tell us about your dental symptoms, is as follows:

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First Name, middle initial, and last name 1. Next, questions are asked as follows: 1. Are you feeling any pain at this time? If not, please go to Question 5. (Yes or no) 2. If yes, where is the pain located? any pus? (Yes or no) 3. When did you first notice any symptoms? 4. Did symptoms occur suddenly or gradually? 5. Please check the intensity (on a scale of 1 to 10 where 1 equals mild and 10 equals severe), frequency (constant, intermittent, momentary, and occasional), and quality of the discomfort (sharp, dull, and throbbing), which most closely describes your pain. 6. Is there anything you can do to relieve the pain? (Yes or no) If yes, what? 7. Is there anything that provokes pain or that you do to cause the pain to increase? (Yes or no) If yes, what? 8. When eating or drinking, is your tooth sensitive to: (Heat, cold or sweets) 9. Does your tooth hurt when you bite down or chew? (Yes or no) 10. Does it hurt if you press the gum tissue around this tooth? (Yes or no) 11. Does a change in posture (lying down or bending over) cause your tooth to hurt? (Yes or no) 12. Do you grind or clench your teeth? (Yes or no) If so, do you wear a splint or night guard? (Yes or no) 13. Has a restoration (filling or crown) been placed on this tooth recently? (Yes or no) 14. Prior to this appointment, has root canal therapy been started on this tooth? (Yes or no) 15. Any recent or past trauma or injury to this tooth? (Yes or no) If the answer to the preceding question is yes, describe recent or past trauma and occurrence date. 16. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis or treatment?

Each query or option is followed by blank space.

At the bottom, blank spaces for signature of patient (or parent) and date are given.

Diagnostic and progress records

Diagnostic and progress records often combine fill-in and check-off types of forms. Fill-in or essay forms allow greater latitude of response to a question, resulting in a more detailed description. However, one drawback is that these forms are open to oversights unless a clinician is conscientious in following up with further clinical information.

Using only an essay-type health history is insufficient. Often a patient may not appreciate the significance of important symptoms. A check-off format is efficient and more practical. Forms with questions that reveal pertinent data alert the clinician to medical or dental conditions that warrant further evaluation or consultation.[43] Moreover, such records can document any missing medical information the patient failed to provide orally. At the end of the check-off portion of the medical history, an essay question allows the

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patient to provide any omitted pertinent medical information or elaborate on checkmarked items.[273,286]

Electronic records

Electronic records represent current methodology for recording patient information (see Chapter 28 for details).[307] Several safeguards are essential for their use. First, patient confidentiality and security must be protected. Consider using advanced encryption standard (AES) encryption with at least 2048 bit key. Be certain that only the clinician and authorized staff view patient data in conformance with the Health Insurance Portability and Accountability Act (HIPAA) security standards, effective April 2005.[149] Second, data backup of patient records, including digital radiographs and account ledgers, should be stored off site. The entire hard drive should be periodically backed up for additional protection, which can be accomplished with cloud technology autosave backup systems.[94] Multiple copies of the backup data should be stored in more than one geographic location, and fault protection such as redundant array of inexpensive disks (RAID) level 1 or 5 should be installed to prevent data loss.

Radiographs

Radiographs are essential for diagnosis and also serve as additional documentation of the patient's pretreatment condition. A panographic radiograph is not diagnostically accurate for endodontics and therefore should be used only as a screening device (see Chapter 2).[143,271,344] Diagnostic-quality periapical plain film or digital radiographs are essential aids for diagnosis, for working films (e.g., measuring the length of root canals, fitting gutta-percha cones), to verify the final fill, and for follow-up comparisons at recall examinations. Therefore the clinician should retake any radiographs that lack diagnostic quality. Retain all radiographs, including working films.

Digital radiography is recommended because it increases endodontic efficiency at the chair. Also, images may be forwarded via e-mail to either the patient or another clinician. No developing time is needed, so procedural radiographs can be viewed instantly and approved or retaken with little time in between (no downtime is required to wait for a film to exit the film

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processor). Newer digital x-rays with improved sensor technology use high-resolution charged coupling device (CCD) chips, rather than complementary metal-oxide semiconductor (CMOS) technology and provide close-up zooming with improved image quality as described in Chapters 5 and 28.

Three-dimensional (3D) cone-beam computed tomography (CBCT) imaging has greater information compared with conventional radiography. Conventional periapical radiographs do not facilitate accurate predictions in the preoperative assessment for the potential perforation of the maxillary sinus. CBCT has greatly aided the diagnosis of pain sources caused by root fractures or incompletely filled, overfilled, or missed extra canals that two-dimensional (2D) periapical imaging may obscure.[20,75,97,157,169,201,204]

According to the U.S. Food and Drug Administration (FDA), switching from D to E speed results in a 30% to 40% reduction in radiation exposure. F-speed film reduces exposure 20% to 50% compared to E-speed film, without compromising diagnostic quality.[19] Digital imaging reduces radiation dose by 40% to 60%.[5,124]

A rectangular collimator decreases the radiation dose by up to fivefold compared with a circular one.[126] Radiographic equipment should provide rectangular collimation for exposure of periapical and bitewing radiographs.

Evaluation and differential diagnosis

Diagnosis includes evaluating pertinent history of the current problem, clinical examination, pulpal testing, periodontal probe charting, and recorded radiographic results. If therapy is indicated, the reasons should be discussed with the patient in an organized way. When other factors affect the prognosis (e.g., strategic importance, restorability of the tooth, or proximity to vital structures), the clinician should consider further consultation with the referring clinician or specialists, including a prosthodontist, periodontist, or radiologist with CBCT imaging, before initiating endodontic treatment.

Diagnosis is essential in treatment decisions. Recommending extractions for undiagnosed pain etiology is generally contraindicated. Chronic idiopathic orofacial pain warrants referrals and consultation with other health care professionals, lest wholesale extractions worsen the patient's dental state and not relieve pain (see Chapter 4).[7] For instance, in a long-term follow-up study of patients with chronic facial pain and without a recognizable

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odontologic pathosis, some patients were subsequently diagnosed with maladies ranging from cracked-tooth syndrome to medication-mediated illnesses.[7] Accordingly, referral to other specialists is essential, lest a patient be misdiagnosed with idiopathic or atypical facial pain when instead the pain etiology was misdiagnosed.[238,255]

About 5.3% of patients have postendodontic pain 6 months or more after nonsurgical and surgical endodontic treatment. Nonodontogenic pain may account for about 57% to 64% of the total causes of postendodontic pain that persists after 6 months. Therefore of patients being treated with endodontic procedures, about 3.4% (64% of 5.3%) will have posttreatment pain that arises from a nonodontogenic cause.[41,237]

Fig. 27.4, *A* and *B*, demonstrates the advantage of CBCT 3D imaging compared to 2D imaging. In *A*, the nail appears inside the cranium, contrasted with *B*, which shows the nail taped outside the skull.

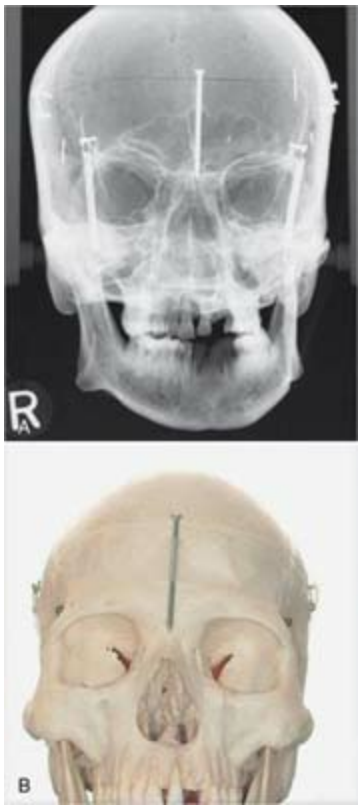

FIG. 27.4 **A,** Nail inside cranium? **B,** Nail in same patient as in **(A)**, showing nail taped to outside of skull. Alas, poor Yorick!

A) Scan of front view of skull shows a nail placed vertically at the center of

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forehead.

B) 3D image shows the skull with the same nail.

Diagnostic tests

Sound endodontics begins with a proper diagnosis. Otherwise, unnecessary or risky treatment with a compromised prognosis follows. Generally, as described in greater detail in Chapter 1, the following endodontic tests should be performed to arrive at a correct and accurate endodontic diagnosis, including adjacent teeth for comparison:

1. Percussion
2. Thermal testing
3. Electrical testing (optional)
4. Palpation
5. Mobility
6. Periodontal assessment (pockets, mobility, furcations, gingival recession)

Both positive and negative pulpal testing results should be recorded. Juries, peer-review committees, and insurance consultants often disbelieve unrecorded test results. Uncharted test findings may be regarded as never having been done. Consequently, reasonable clinicians should record all testing results, both positive and negative. Evaluation of the temporomandibular apparatus is necessary to diagnose or rule out pain producing temporomandibular disorders (TMDs).

Treatment plan

Treatment records should contain a written plan that considers all aspects of the patient's oral health. Treatment plans should be coordinated, preferably in writing, with other concurrent or jointly treating clinicians. For instance, endodontic treatment will probably fail if underlying periodontal pathosis is ignored or untreated. Thus the clinician should assess the patient's entire dentition and not just a single root canal system. The clinician should also recommend a periodontal consultation to both the patient and the referring

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clinician if periodontal therapy is required. The patient should approve the treatment plan. Remember the maxim: If you fail to plan, plan to fail.

Examination

After gathering the dental and medical history, findings obtained from the various phases of the clinical and radiographic examinations are recorded as shown in Fig. 27.5. Lists in each category afford the clinician a systematic format for recording details pertinent to an accurate diagnosis. Appropriate descriptions are circled, followed by the necessary notations in the accompanying spaces. Tabular arrangement allows easier recording and comparison of diagnostic test data acquired from one tooth on different dates or from different teeth on the same visit. A pain intensity index (i.e., 0 to 10) or pain classification (i.e., mild +, moderate ++, severe +++) should be used to differentiate diagnostic test results.

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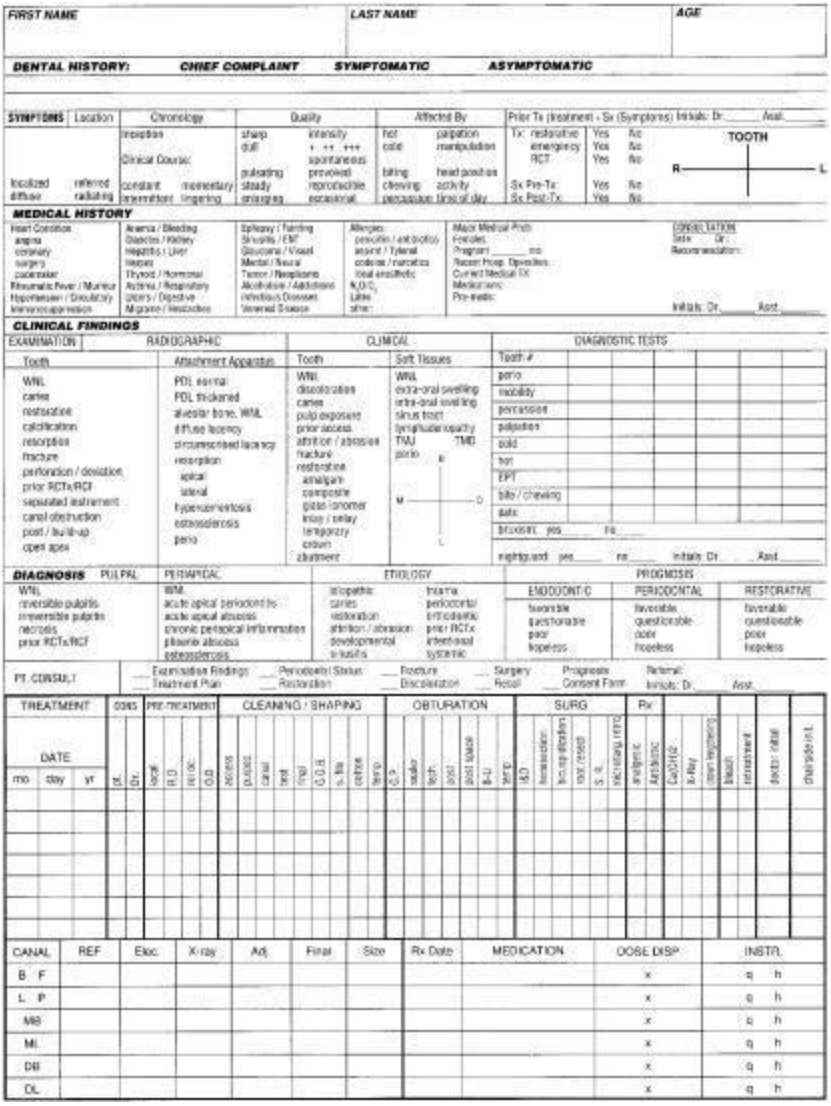

FIG. 27.5 Diagnosis and treatment form.

Diagnosis and treatment form shows spaces for first name, last name, and age, followed by spaces for chief complaint, symptomatic, and asymptomatic options under dental history. Next to it, symptoms panel shows sub-options under location, chronology, quality, affected by, prior treatment plus symptoms, initials: doctor and assistant. It is followed by medical history and clinical findings with sub-options under examination, radiographic, clinical, and diagnostic tests column headers. Further, the next panel shows diagnosis with sub-options under pulpal, periapical, etiology, prognosis (endodontic, periodontal, and restorative) column headers. Next panel shows data for treatment, consultation, pre-treatment, cleaning or shaping, obturation, surgery, and Rx. The bottom panel shows data for canal, ref. elec, X-ray, adj, final, size, Rx date, medication, dose tip, and instrument.

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Diagnosis

Careful analysis of accumulated examination data should result in the determination of an accurate pulpal and periapical diagnosis. Clinical conditions and the probable etiologic factors for the presenting problem are circled. Alternative modalities of therapy are considered and analyzed. The recommended treatment plan is circled, followed by a prognostic assessment of the intended therapeutic course.

Patient consultation

Patients should be advised of each diagnosis and should consent to the treatment plan before therapy is instituted. Consultation should include an explanation of reasonable alternative treatment approaches and rationales and treatment consequences of each alternative therapy, including risks from nontreatment or delayed treatment that may affect the outcome of intended therapy. This discussion is documented by completing the checklist items.

Treatment

All treatment provided on a given date is documented by placing a checkmark (✓) within the designated procedural category. Only the most frequent retreatment procedures are included for tabulation. Descriptions of occasional procedures or explanatory treatment remarks should be entered in writing. A separate dated entry should be made for each patient visit, phone call, e-mail message, and fax communication (e.g., consultations with the patient or other clinicians) or correspondence (e.g., biopsy report, treatment completion letters).

Individual root canal lengths are recorded by (1) circling the corresponding anatomic designation and the method of length determination (e.g., radiograph or electronic measuring device), (2) writing the measurement (in millimeters), and (3) indicating the reference point. For any medication prescribed, refilled, or dispensed, the treatment record should show the date and type of drug (including dose, quantity, and instructions for use) in the treatment table under the heading “Rx.” Fig. 27.6 also shows a sample medication log. Periodic recall intervals, dates, and findings are entered in the spaces provided.

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MEDICATION LOG: P. Ulp (Patient name) (415) 391-5353 (Phone number)

Medication allergies Salfa drugs

Date	Medication	No. Tabs/ Caps/ Gel	Dosage	Refill? (Y/N)	Rx Written-W Dispen.-D Phone-P	Phoned Rx? (pharmacy & phone #)	Staff Init	Dr. Init
6/19/20	Augmentin	10	1 gm	N	W			EZ
6/29/20	Acetaminophen	12	1000 mg	Y	W			MZ
6/29/20	Ibuprofen	12	400 mg	Y	W			MZ

FIG. 27.6 Medication log.

Template of medication log is as follows:

It begins with patient name, followed by phone number, and medication allergies.

Further, the template has ten rows and nine columns with the following column headers: date; medication; number of tabs/ caps/ gel, dosage/quantity; refill question mark (yes or no); R sub x written-W, Dispen-D, and phone-P; phoned R sub x question mark (pharmacy and phone hash); staff initial, and Doctor initial.

If the scope of the examination or treatment is intentionally limited, such as a screening examination or emergency endodontic therapy, the limited scope of the visit should be recorded. Otherwise, the chart appears as if the

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examination were superficial and treatment incomplete. If a suspicious apical lesion requires subsequent reevaluation, the clinician should record the future reevaluation date and differential diagnosis—for example, “Small apical lesion on #28. Reevaluate for changes in 2 months. Also, check for any root fractures.” If this is not done, it appears (on the chart) as if the clinician ignored potential pathosis, such as suspected root fracture. A soft-tissue examination (including cancer check) is a standard part of any complete dental examination. E-mail, prescriptions, and correspondence should be documented with hard copies in the chart unless virtually all treatment records are stored in electronic format.[191,307]

Informed consent form

After endodontic diagnosis, the benefits, risks, treatment plan, and alternatives to endodontic treatment—including any patient refusal of recommended treatment and consequences of refused treatment—should be presented to the patient or the patient’s guardian. This will document acceptance or rejection of treatment recommendations. The patient or guardian, along with a witness (who can be a staff member), should sign and date the consent form. Any informed-consent video should similarly be noted, if viewed. Subsequent changes in the proposed treatment plan should also be discussed and initialed by the patient to indicate continued acceptance and acknowledge the patient’s understanding of any newly disclosed risks, alternatives, or referrals.

Despite a patient’s confirmed signature on an informed consent form, a jury is free to decide that the patient never read the informed consent form’s content before signing. For instance, the patient may claim it was impossible to have read the consent form, because he or she did not have reading glasses when signing and no clinician ever explained the form contents. Another frequent factual scenario is when the patient claims the staff stated it was a standard consent form that was a mere procedural formality requiring a patient signature, rather than a detailed informed consent form. Consequently, numerous legal cases have been lost at trial on the issue of inadequate informed consent despite a signed patient consent form.

To obviate a patient’s claim that no explanation ever occurred, a patient questionnaire can additionally be used. Patients can be instructed that unless

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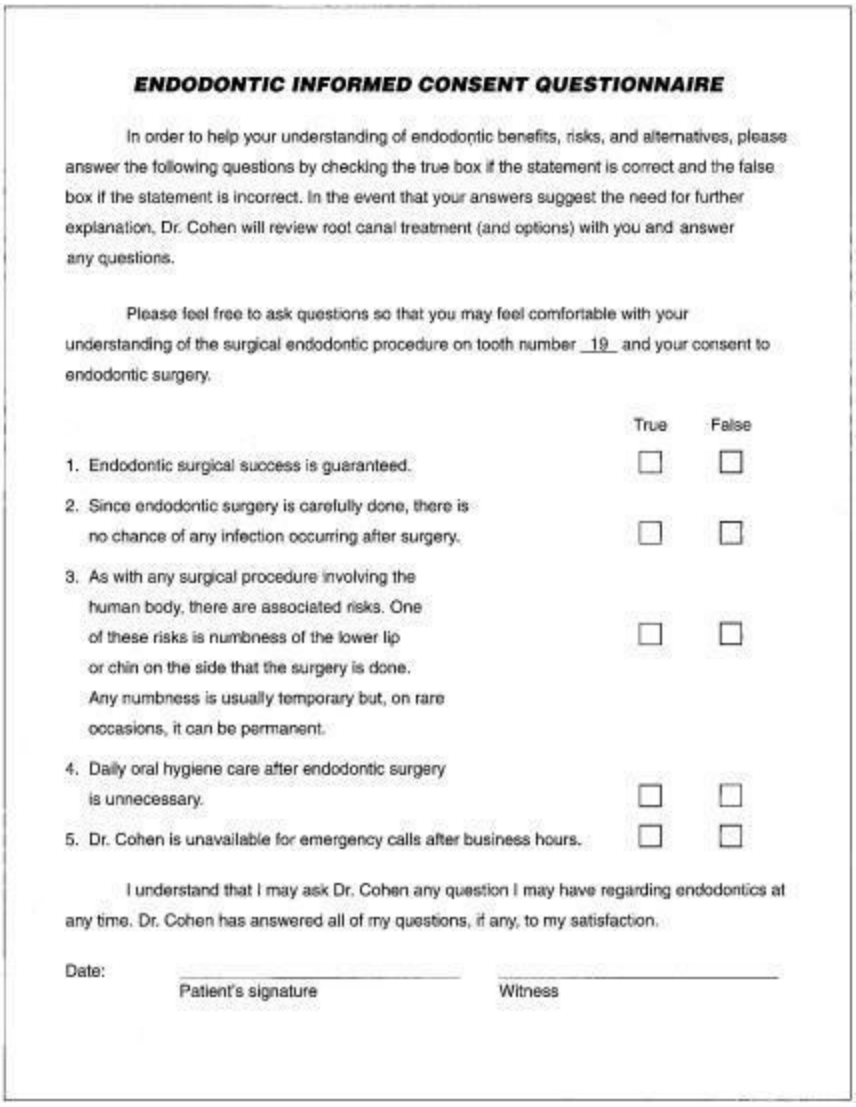
they score 100%, proposed procedures will not be done (because patient understanding is imperative and essential for cooperation, including postoperative care). To be effective, the patient questionnaire should be relatively short and simple (Fig. 27.7). Also, the patient should be given an opportunity to correct any incorrect answers after the clinician reviews with the patient the reasons why the patient answered incorrectly. Note that the majority of answers are false. This precludes a patient from guessing the correct answer by assuming that all test questions are necessarily true.



ENDODONTIC INFORMED CONSENT QUESTIONNAIRE

In order to help your understanding of endodontic benefits, risks, and alternatives, please answer the following questions by checking the true box if the statement is correct and the false box if the statement is incorrect. In the event that your answers suggest the need for further explanation, Dr. Cohen will review root canal treatment (and options) with you and answer any questions.

Please feel free to ask questions so that you may feel comfortable with your understanding of the surgical endodontic procedure on tooth number 19 and your consent to endodontic surgery.

	True	False
1. Endodontic surgical success is guaranteed.	☐	☐
2. Since endodontic surgery is carefully done, there is no chance of any infection occurring after surgery.	☐	☐
3. As with any surgical procedure involving the human body, there are associated risks. One of these risks is numbness of the lower lip or chin on the side that the surgery is done. Any numbness is usually temporary but, on rare occasions, it can be permanent.	☐	☐
4. Daily oral hygiene care after endodontic surgery is unnecessary.	☐	☐
5. Dr. Cohen is unavailable for emergency calls after business hours.	☐	☐

I understand that I may ask Dr. Cohen any question I may have regarding endodontics at any time. Dr. Cohen has answered all of my questions, if any, to my satisfaction.

Date: _____ Patient's signature _____ Witness

FIG. 27.7 True/false endodontic informed consent form.

The form titled, "Endodontic informed consent questionnaire" is as follows:

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In order to help your understanding of endodontic benefits, risks, and alternatives, please answer the following questions by checking the true box if the statement is correct and the false box if the statement is incorrect. In the event that your answers suggest the need for further explanation, Dr. Cohen will review root canal treatment (and options) with you and answer any questions.

Please feel free to ask questions so that you may feel comfortable with your understanding of the surgical endodontic procedure on tooth number 19 and your consent to endodontic surgery.

1. Endodontic surgical success is guaranteed. (True or False)

2. Since endodontic surgery is carefully done, there is no chance of any Infection occurring after surgery. (True or False)

3. As with any surgical procedure involving the human body, there are associated risks. One of these risks is numbness of the lower lip or chin on the side that the surgery is done. Any numbness is usually temporary but, on rare occasions, it can be permanent. (True or False)

4. Daily oral hygiene care after endodontic surgery is unnecessary. (True or False)

5. Dr. Cohen is unavailable for emergency calls after business hours. (True or False)

I understand that I may ask Dr. Cohen any question I may have regarding endodontics at any time. Dr. Cohen has answered all of my questions, if any, to my satisfaction.

At the base, date and blank spaces for Patient's signature as well as witness are given.

Treatment record: Endodontic chart

A suggested chart is presented in Fig. 27.8 to facilitate recording of information pertinent to the diagnosis, recommendations, and treatment of the endodontic patient. Systematic acquisition and arrangement of data from the patient questionnaire, along with clinical and radiographic examinations and careful recording of treatment performed, expedites accurate diagnosis and maximizes clinician efficiency. Suggested chart format and use are described in the sections that follow.

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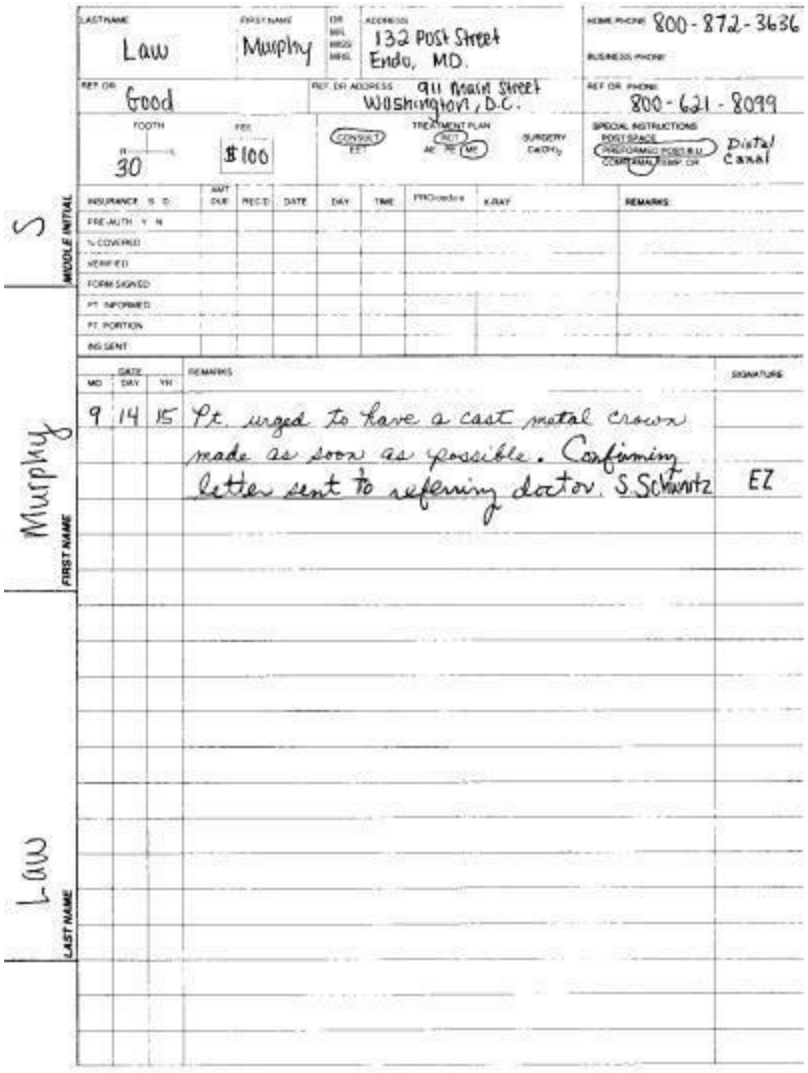

LAST NAME: Law
FIRST NAME: Murphy
MIDDLE INITIAL: S
ADDRESS: 132 Post Street, Endo, MD.
HOME PHONE: 800-872-3636
REF DR: Good
REF DR ADDRESS: 911 Main Street, Washington, D.C.
REF DR PHONE: 800-621-8099
TOOTH: 30
FEE: $100
SPECIAL INSTRUCTIONS: Distal Canal

INSURANCE I. D.	AMT DUE	REC'D	DATE	DAY	TIME	PROcedure	X-RAY	REMARKS
PRE-AUTH Y N								
% COVERED								
VERIFIED								
FORM SIGNED								
PT. INFORMED								
PT. PORTION								
INS SENT								

DATE MO	DAY	YR	REMARKS	SIGNATURE
9	14	15	Pt. urged to have a cast metal crown made as soon as possible. Confirming letter sent to referring doctor. S. Schmitz	EZ

A

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B

FIG. 27.8 **A,** Progress notes. **B,** Diagnosis and treatment form with notes.

It begins with last name, first name, address, home phone, and business phone. The next row shows information about ref Doctor, ref doctor address, ref doctor phone, followed by tooth location and number with fee, treatment plan, and special instructions in the next row.

Next section shows arrays with row headings as follows: insurance, pre-auth, percent covered, verified, form signed, pt. informed, pt. portion, and ins sent. The columns are as follows: amount due, rec'd, date, day, time, pro-cedure, x-ray, and remarks.

Next section has data as follows: date (month, day, year), remarks, and signature.

Filled diagnosis and treatment form is shown. First name, last name, and age of the patient, followed by chief complaint, symptomatic, or asymptomatic

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dental history. Next to it, symptoms panel shows sub-options under location, chronology, quality, affected by, prior treatment plus symptoms, initials: doctor and assistant. It is followed by medical history and clinical findings with sub-options under examination, radiographic, clinical, and diagnostic tests column headers. Further, the next panel shows diagnosis with sub-options under pulpal, periapical, etiology, prognosis (endodontic, periodontal, and restorative) column headers. Next panel shows data for treatment, consultation, pre-treatment, cleaning or shaping, obturation, surgery, and Rx. The bottom panel shows data for canal, ref. elec, X-ray, adj, final, size, Rx date, medication, dose tip, and instrument.

General patient data

Patient name, address, phone numbers, referring clinician, and chief complaint are printed or typed in the corresponding space at the patient's initial office visit. The patient's appointment and financial record are divided into two parts as follows:

1. The treating clinician or staff completes the first portion after the diagnosis and treatment plan have been formulated and presented to the patient. Tooth number and quoted fee are posted. The treatment plan is recorded by circling the appropriate description. Under "Special Instructions," specific treatment requests by the referring clinician are circled. Details of planned adjunctive procedures (e.g., hemisection, root resection) may be written in the adjacent space, along with information from the patient data section. The clinician can then use this information for general reference during future treatment. The secretary or treatment coordinator can also use this information when scheduling appointments and establishing financial arrangements.
2. Business personnel complete the second portion. Financial agreements, third-party coverage, account status, and appointment data (including the day, date, and scheduled procedure) are recorded.

Either the clinician or the assistant may complete portions of the diagnosis and treatment sections, but *the clinician should review and approve all entries*.

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Dental history

The chief complaint should include whether the patient is symptomatic at the time of examination or in the recent past and what medications the patient is currently taking for pain relief. Narrative facts regarding the presenting problem are then recorded. Additional details of the chief complaint obtained during successive questioning are recorded by circling the applicable word within each symptom parameter. The pain intensity index (i.e., 0 to 10) or pain classification (i.e., mild +, moderate ++, severe +++) should be registered alongside the appropriate description. For accurate assessment of the effects of prior dental treatment pertaining to the examination site, a summarized account of these procedures should be documented. In addition, all pretreatment signs and symptoms should be described. It is essential that the patient comprehend all communications with the clinician and staff. If there is any language comprehension difficulty, the clinician should ask the patient whether he or she understands completely the language the clinician is speaking. To verify patient comprehension, the clinician should ask the patient to repeat important information that has been conveyed. A clinician- or patient-provided interpreter may be necessary if language problems exist.

Medical history

More medical history information is obtained from a patient-administered questionnaire than from the clinician obtaining an oral history solely from the patient.[43,286] Maximum information is obtained if the clinician reviews with the patient the written health history form the patient has completed.

Information (e.g., personal physician's name, address, and phone number; patient's age; date of last medical examination) is recorded for reference. The clinician can obtain a detailed medical history by completing a survey of common diseases and disorders along with a comprehensive review of corresponding organ systems and related pathosis. Specific entities that have affected the patient are circled. Essential remarks regarding these entries (e.g., details of consultations with the patient's physician) should be documented on an attached blank sheet with dated treatment notes attached to the back of the chart (see Fig. 27.2). A review of the patient's medical status (including recent or current conditions, treatments, and medications) completes the medical history. Updated medical histories that the patient

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signs or initials should be done at least annually and at reevaluation visits.

Excessive alcohol consumption can contribute to a higher incidence of restoration corrosion from chronic regurgitation and vomiting from gastritis associated with alcohol abuse.[139] Corrosion can lead to coronal marginal leakage predisposing to endodontic failure.[305] Accordingly, medical histories should include histories of gastric reflux disease, bulimia with associated habitual vomiting, gastritis, and excessive alcohol intake.

Periodontal disease

Nearly half of American adults older than age 30 years have periodontal disease.[241] Periodontal disease was higher in men than in women (56.4% vs. 38.4%) and was highest among Mexican Americans (66.7%).

Periodontal examination

Competent endodontic treatment begins with adequate diagnostic procedures. An adequate periodontal evaluation must accompany each endodontic diagnosis, which requires a diagnostic radiograph, clinical visualization, mobility recordings, evaluation of the periodontal tissues, and probing for periodontal pockets with a calibrated periodontal probe, particularly in furcation areas.[9,117a,211]

Periodontal chart documentation should include pocket recordings, extent of furcation, bleeding on probing, mobility, areas of gingival recession, and any inadequate attached gingiva.[93,218,241]

Although endodontic treatment may be successful, tooth loss may result from progression of any residual, untreated periodontitis. Consequently, periodontal evaluation and prognosis are mandatory so that the patient and clinician can make an informed and intelligent choice about whether to proceed with endodontics, a combination of periodontal and endodontic treatment, or extraction.

Each tooth undergoing endodontic therapy (and adjacent teeth) should be probed with a calibrated periodontal instrument to obtain six measurements per tooth. Pockets of 4 mm or greater should be recorded on the patient's chart. If no pockets exist, WNL (within normal limits) or a similar abbreviation should be noted. Mobility should also be charted and classified class I, II, or III. Gingival recession, furcations, and mucogingival

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deficiencies should also be recorded.[9,117a]

A clinician who treats with endodontic success but ignores loss of periodontal attachment may misdiagnose or fail to appreciate the risk of failure because of poor periodontal prognosis. It bears repeating that the endodontic treating clinician should not assume an adequate periodontal evaluation has been performed by another clinician or the referring clinician. Instead, an independent periodontal evaluation should be done with a calibrated periodontal probe and results recorded before initiating endodontics, including noting the absence of pockets or within normal limits.

If clinically significant periodontal disease is present, the endodontic treating clinician should consult with the restorative clinician to determine whether the periodontal disease will be properly treated or referred to a periodontist in conjunction with endodontic treatment. A patient should be advised of any compromise of the endodontically treated tooth's periodontal status to comply with required informed consent disclosure.

Drug history

A current drug history, both written and oral, is necessary to alert the clinician to any potential for interaction between any newly prescribed drugs and any other medication, supplements, or over-the-counter medication the patient is already taking.

In patients aged 57 through 85, 40% are potentially at risk of a major drug reaction—half involving various prescription medications and the other half involving anticoagulant drug reactions. About half of prescription users also concurrently take over-the-counter medications plus dietary supplements.[190,235]

Risks of drug interactions or side effects incidence may not be discovered or appreciated in premarket research-controlled drug trials for newly marketed drugs, despite FDA approval. For example, rofecoxib (Vioxx) was withdrawn from the market in September 2004 after increased incidences of myocardial infarcts and strokes manifested in a postmarket research study of 2600 patients. Merck's voluntary recall followed an FDA researcher's claim that the FDA tried to ostracize and intimidate him after he raised safety concerns over Vioxx.[33] Currently, pharmaceutical manufacturers budget more for marketing than research and development.[121] Some drug

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manufacturers have suppressed publication of adverse research.[55,208,337] The Patient Protection and Affordable Care Act of 2010 (Section 6002) requires drug companies to disclose payments to medical/dental professionals who tout their drugs in seminars or publications and receive compensation greater than $500.[245]

For newly marketed drugs, the small number of premarket patients studied may lack sufficient statistical power to expose serious side effects that occur infrequently but are nonetheless life threatening.[337] For instance, mibefradil (Posicor) was withdrawn from the market after 1 year because it increased plasma concentrations of 25 other co-administered drugs, including erythromycin.[235,239] In another example, the popular prescription nighttime heartburn drug Propulsid (cisapride) was linked to 70 deaths and more than 270 significant negative reactions.[328] Other examples include the risk of esophageal cancer with bisphosphonate[337,341] and bisphosphonate-associated osteonecrosis.[92,287] In 2008, the FDA added a black box warning label for Levaquin-associated risks of tendonitis (www.levaquin.com/levaquin/medication).

Preoperative and postoperative radiographs

Pretreatment, midtreatment, and posttreatment radiographs or digital images are essential for endodontic diagnosis and treatment:

1. A current, pretreatment, interpretable periapical radiograph of adequate diagnostic quality is mandatory.
2. Measurement films or digital images are necessary to verify canal length (which can be supplemented with an electronic canal-measuring device) and the apical extent of the gutta-percha fill to the radiographic apex.
3. Posttreatment radiographs are essential for determining the adequacy of the endodontic seal or if further treatment is necessary (see Chapter 10).

Digital radiography

Digital radiographic endodontic applications are ever increasing in

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accuracy.[142] The standard of care does not currently require digital imaging, because traditional silver halide radiographic film is a reasonable alternative. When there is more than one reasonably acceptable practice modality, a clinician who chooses either modality meets the standard of care. Fig. 27.9, *A*, represents a distal open margin on tooth #30 (shown digitally) that is not evident in part *B* with plain-film radiography.

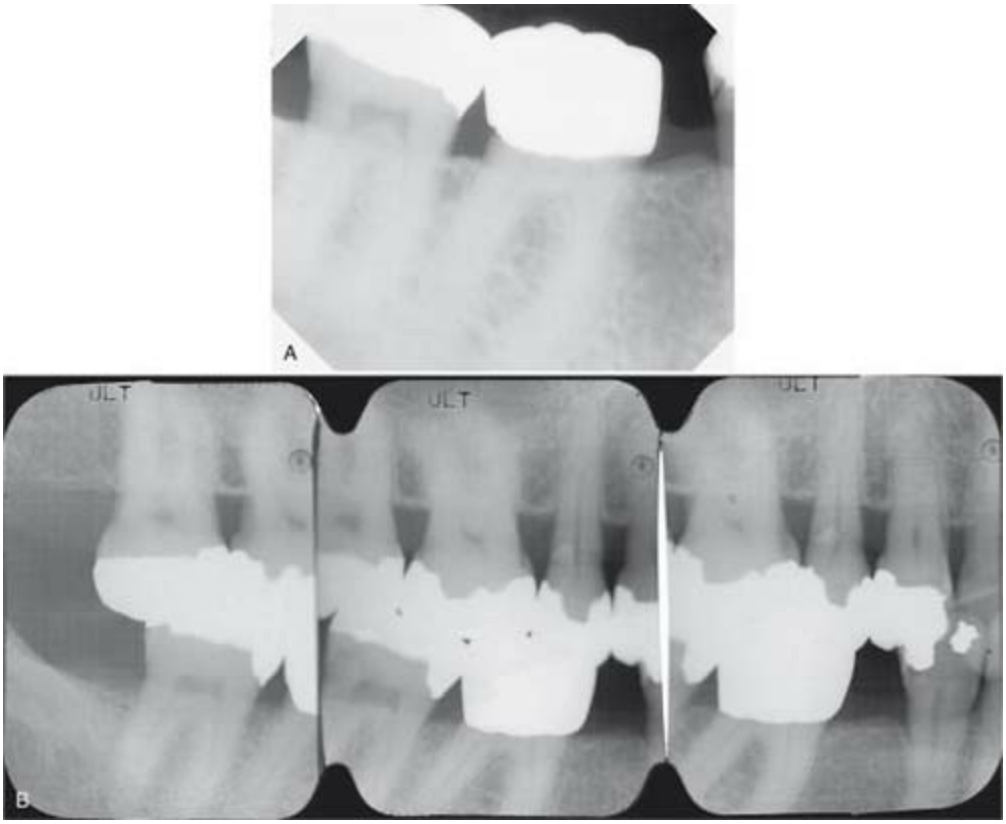

FIG. 27.9 **A,** Distal open margin on tooth #30 (shown digitally). **B,** Distal open margin on tooth #30 not evident with plain-film radiography.

Set of four radiographs marked A through B evaluates the distal open margin of tooth 30 using digital and plain-film radiography.

Advertised claims of 80% reduction in radiation with direct digital radiography (rather than film) assume the following[53,142]:

1. Ultraspeed D-speed film is used. (Ektaspeed E-speed film and the newer F-speed film reduce radiation 50% and 60%, respectively, while producing images of comparable quality to D speed.)[4]

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2. No rectangular collimation is used, which reduces exposure another 30%.
3. No extra radiographs are taken to compensate for a smaller area. For instance, a 2 CCD sensor has an active area smaller than size 1 film and may require an extra image with additional radiation exposure.

A digital x-ray image should never be modified to “enhance” the radiographic appearance of the original image. X-ray modification can be detected. In litigation, it can prove devastating to the clinician’s defense because the clinician’s credibility of events or recorded entries are not believable, particularly if the radiograph is Photoshopped.

Periapical (PA) radiography is limited to only two dimensions. Newer 3D imaging systems for dental radiology use x-ray beams that are cone shaped. CBCT can provide a more nearly accurate diagnosis.[75,122,157] In Fig. 27.10, *A–C*, the PA does not show the transported canal and extent of short fill, which is more evident in the CBCT. Each CBCT scan produces three image views: axial, coronal, and sagittal.

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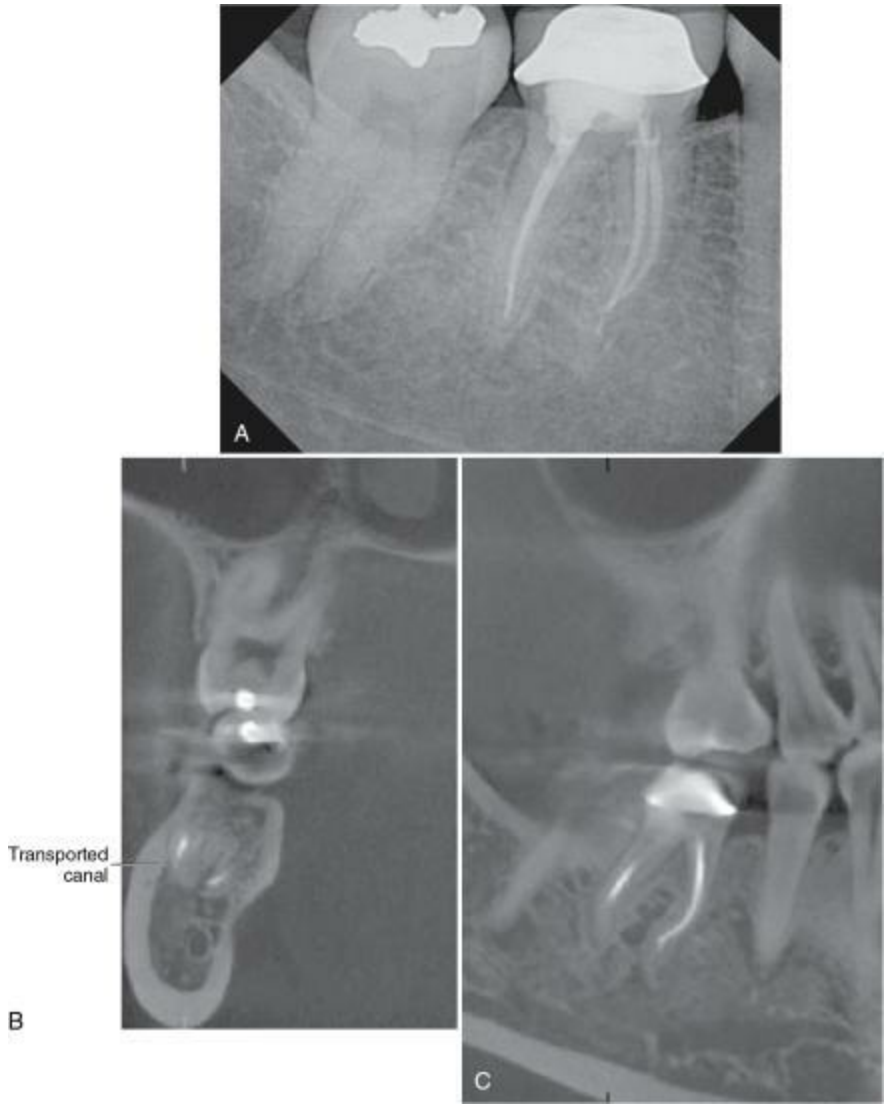

FIG. 27.10 **A,** Periapical (PA) view, which does not show full extent of transported and short-filled canal. Both deficiencies evident in **B** and **C** with cone-beam computed tomography (CBCT). **B,** CBCT Accuitomo of transported canal. **C,** CBCT Accuitomo of short-filled canal.

A) Periapical radiograph shows filled root canals.

B) CBCT image shows transported canal in tooth.

C) CBCT image shows right canal one-fourth filled at the center and left canal completely filled in the tooth.

Radiation safety

The American Dental Association (ADA) Council on Scientific Affairs

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recommends the dentist recognize the importance of keeping radiation exposure as low as reasonably achievable (ALARA). Accordingly, CBCT imaging should not be performed for screening purposes but rather for specific diagnostic and treatment usage.[20]

The Food and Drug Administration provides recommendations for dental CBCT.[99] The American Academy of Oral and Maxillofacial Radiology provides indicated use of the CBCT for implant placement.[317] In the future, magnetic resonance imaging (MRI) may supplant or augment CBCT because its imaging quality equals and in certain instances surpasses that of CBCT.[189]

Children have greater radiosensitivity because of developing cells in their young bodies. Thus CBCT imaging radiation dosage should be considered for young patients in assessing the need for a CBCT.

Computed tomography (CT) scans enable evaluation of the true extent of lesions and their spatial relationship to important anatomic landmarks.[97] Fig. 27.11, *A–C*, shows a bifid inferior alveolar nerve canal (IANC) that is not apparent on Panorexes. CT scans may provide information important for retreatment decisions, especially apical surgery. CBCT[75,201,204] showed significantly more lesions than PA radiography, including expansion of lesions into the maxillary sinus, sinus membrane thickening, and missed canals; PA radiography missed 34% of these lesions.[201,204] Artificial bone defects in the antral surface were not detected with PA radiography. Because of overlapping roots, only 1 out of 14 furcation defects in upper molars were seen on PA radiography, but CT scans were able to identify all furcal defects. Second molars proved to be the most difficult for detecting lesions with PA radiography alone. Twenty-three teeth with lesions expanding into the maxillary sinus were detected by CBCT, of which only two teeth were seen with PA radiography. Thirty-five teeth with membrane thickening were identified with CBCT, of which 16 teeth were also detected with PA radiography. Fifteen teeth with missing canals were detected with CBCT, of which only four teeth were identified with PA radiography. In a study that compared the efficacy of PA radiography and CBCT in detecting periapical lesions in maxillary premolars and molars for apical surgery,[204] Lofthag-Hansen showed that 38% of maxillary posterior teeth lesions were undetected by PA radiography, despite the fact that an additional different angulated PA radiograph was taken.[201] Lesions associated with apices near the sinus floor

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had a higher probability of being missed with PA than lesions associated with apices located away from or overlapping the sinus floor. Expansion of the lesion into the maxillary sinus, thickening of the sinus membrane, missed canals, and presence of apicomarginal communications were more frequently detected with CBCT than with PA radiography.[201]

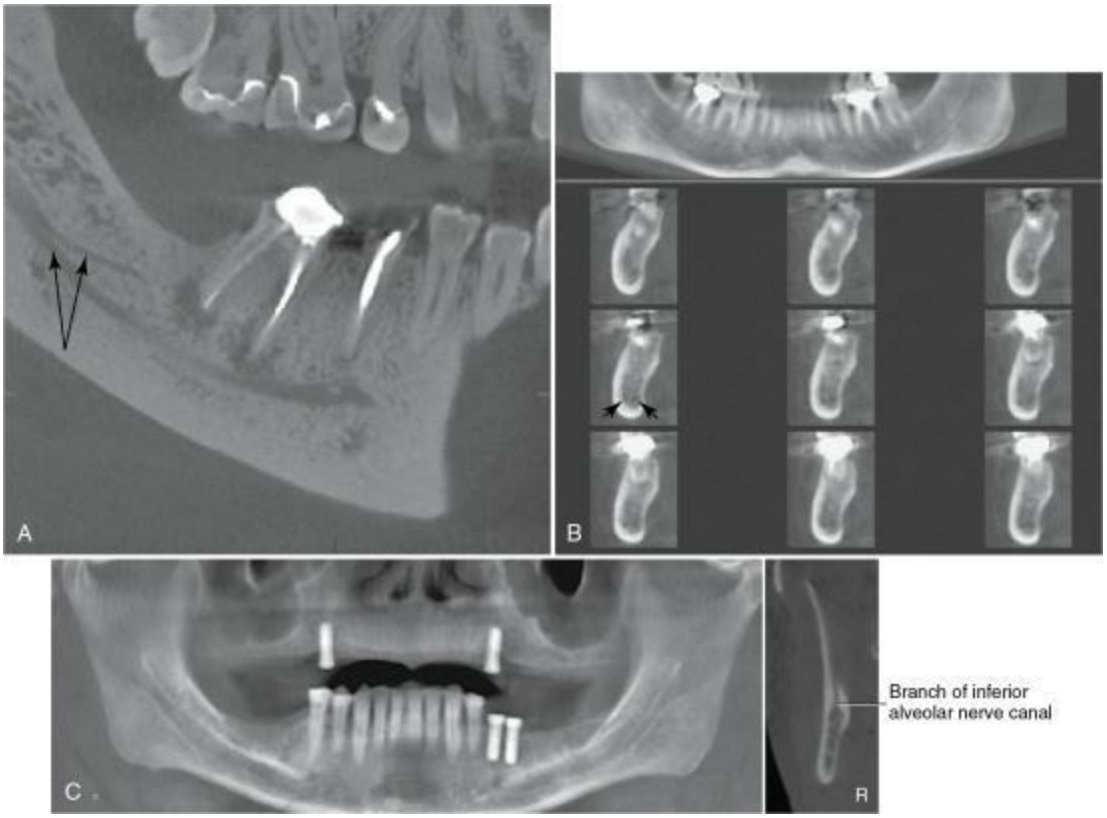

FIG. 27.11 **A,** Bifid inferior alveolar nerve canal (IANC) visible with cone-beam computed tomography (CBCT). **B,** Bifid canal visible with CBCT but not shown on Panorex. **C,** Branch of IANC apparent on CBCT but not shown on Panorex.

A) Radiograph shows IANC on right side of lower jaw.

B) Panoramic view of lower jaw is followed by nine coronal sections. In one coronal section, bifid IANC is marked.

C) Panoramic view of both jaws is shown. Branch of inferior alveolar nerve canal is marked separately.

Source: (Courtesy Gurminder Sidhu, DDS, MS.)

Apicomarginal communication is an important predictor for the success

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rate of apical surgery, including evidence of undetected vertical fractures necessitating extraction; 83% of apicomarginal communications were not seen with PA radiography in the Lofthag-Hansen study.

If the radiograph shows a small distance between the periapical lesion and the sinus floor when the bony wall is thin, there is a high probability that an oral antral communication (OAC) can result, unless skillful care avoids sinus penetration during apical surgery. CBCT also allows the clinician to study the topography of the bony defect and assess whether the use of guided tissue membranes would be beneficial. Precise morphometric assessment of osseous relationships to the sinus is often inadequate with periapical radiography. Thus CBCT is invaluable in treatment planning for apical surgery, as 70% of cases studied revealed clinically relevant information not found in PA radiography.[204]

The probability of detecting lesions with PA alone was limited for teeth with apices in close contact with the floor of the maxillary sinus, for molars (in particular second molars), and when bone thickness between lesion and sinus was less than or equal to 1 mm. Findings of lesion expansion into the sinus, sinus membrane thickening, missed canals, and presence of apicomarginal defects are more frequently diagnosed with CBCT than PA.[75,204]

Endodontics and heart disease

Associations between chronic infections such as periodontal diseases and coronary heart disease (CHD) have been suggested.[280] Conversely, supporting evidence linking root canal–filled teeth or teeth with periapical disease to CHD is lacking.[119]

If a patient's medical history shows valvular heart defects with regurgitation, the clinician should consult with the patient's physician to determine the necessity of antibiotic prophylaxis against infective endocarditis in accordance with current American Heart Association (AHA) guidelines.[334] If the physician does not appear knowledgeable about those guidelines, the clinician should provide a copy of the guidelines to the physician. If the physician advises deviation from AHA guidelines, the clinician should inquire why the physician feels that this is appropriate. The

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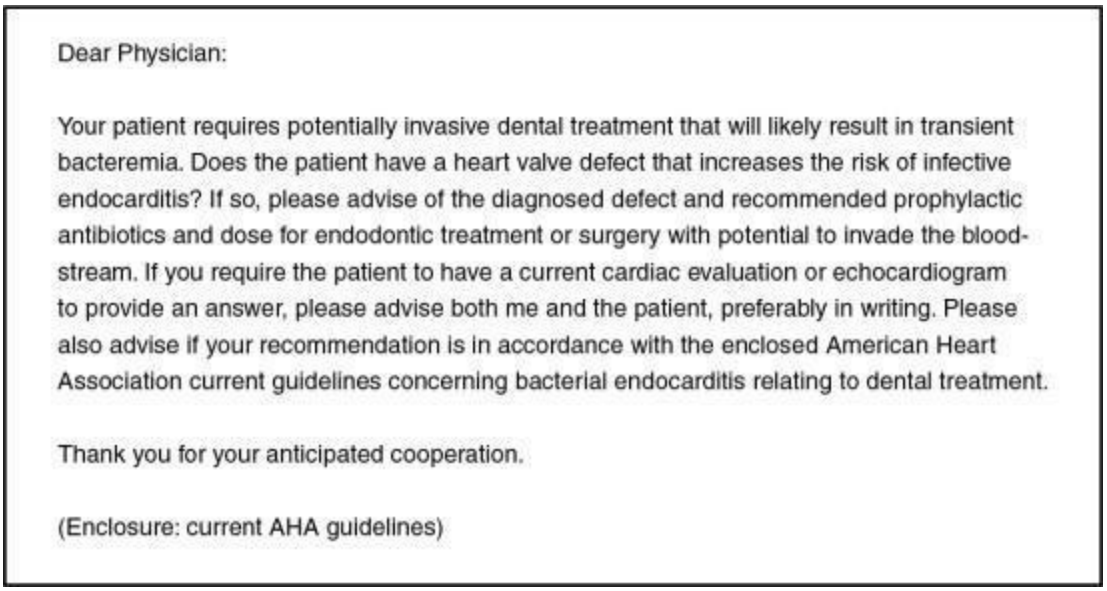
clinician should also record any discussions with the physician in the patient's chart and confirm the discussion and the physician's recommendation in writing (by fax and letter to the physician).

Communications with the physician should be specific, because dental treatment risks, such as overextension of files or filling material sealants that occur nonsurgically and may enter the bloodstream, may not be fully understood or appreciated. A sample format is shown in Fig. 27.12.



Dear Physician:

Your patient requires potentially invasive dental treatment that will likely result in transient bacteremia. Does the patient have a heart valve defect that increases the risk of infective endocarditis? If so, please advise of the diagnosed defect and recommended prophylactic antibiotics and dose for endodontic treatment or surgery with potential to invade the blood-stream. If you require the patient to have a current cardiac evaluation or echocardiogram to provide an answer, please advise both me and the patient, preferably in writing. Please also advise if your recommendation is in accordance with the enclosed American Heart Association current guidelines concerning bacterial endocarditis relating to dental treatment.

Thank you for your anticipated cooperation.

(Enclosure: current AHA guidelines)

FIG. 27.12 Sample of written physician recommendations.

Sample of written physician recommendations is as follows:

Dear Physician:

Your patient requires potentially invasive dental treatment that will likely result in transient bacteremia. Does the patient have a heart valve defect that increases the risk of infective endocarditis? If so, please advise of the diagnosed defect and recommended prophylactic antibiotics and dose for endodontic treatment or surgery with potential to invade the blood-stream. If you require the patient to have a current cardiac evaluation or echocardiogram to provide an answer, please advise both me and the patient, preferably in writing. Please also advise if your recommendation is in accordance with the enclosed American Heart Association current guidelines concerning bacterial endocarditis relating to dental treatment.

Thank you for your anticipated cooperation.

(Enclosure: current AHA guidelines)

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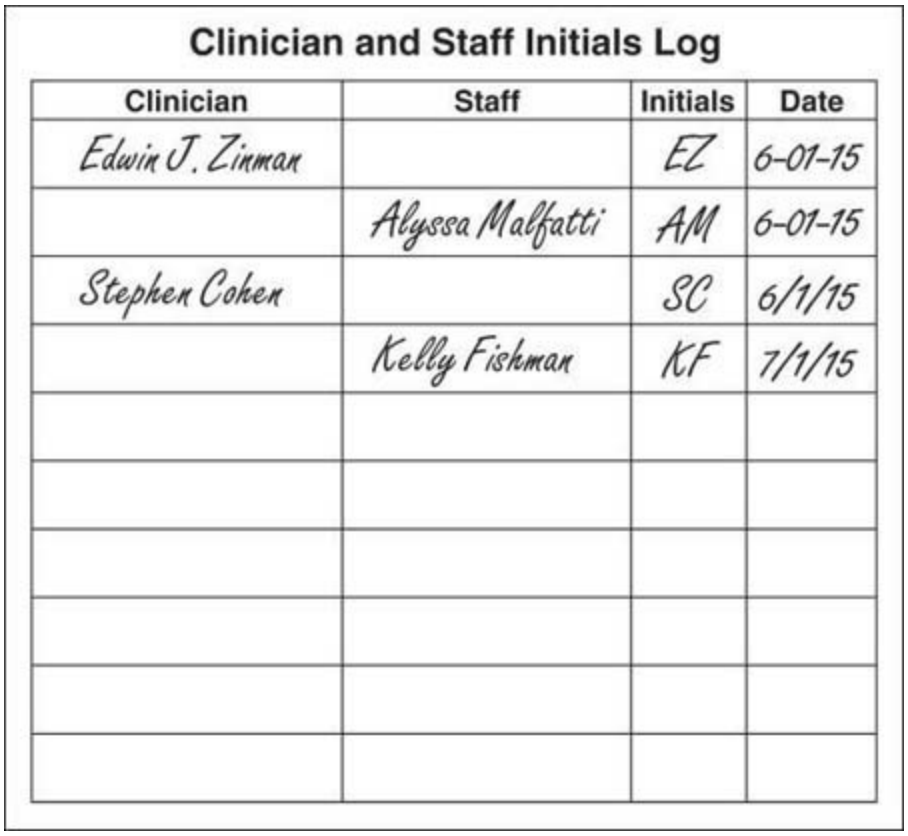
Abbreviations

Abbreviated records can be frustrating if the clinician is unable to decipher his or her own or other clinicians' handwritten entries, so the clinician should use standard or easily understood abbreviations. Pencil entries are legally valid, but ink entries are less vulnerable to a plaintiff's claim of erasure or alteration. A short pencil is better than a long memory; records remember, but patients and clinicians may forget.

A sample of a completed endodontic chart (Fig. 27.13) illustrates its proper use. Box 27.1 lists a key to the abbreviations.



Clinician and Staff Initials Log

Clinician	Staff	Initials	Date
Edwin J. Zinman		EZ	6-01-15
	Alyssa Malfatti	AM	6-01-15
Stephen Cohen		SC	6/1/15
	Kelly Fishman	KF	7/1/15

FIG. 27.13 Clinician and staff initials log.

Template of clinician and staff initials log has four column headers as follows: clinician, staff, initials, and date.

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Box 27.1

Standard Abbreviation Key

Ab	=	Antibiotic
ABS	=	Abscess
Access	=	Access cavity
AG or AmAL	=	Amalgam
AE	=	Air embolus
analg.	=	Analgesic
Apico	=	Apicoectomy
B-U	=	Buildup of tooth
Bisp	=	Bisphosphonate
BW	=	Bitewing
Canal	=	Identify canal that has been cleaned and shaped
CBCT	=	Cone-beam computed tomography
Comp	=	Composite
Cotton	=	Placed in pulp chamber between treatments
CRN	=	Crown
Dx	=	Diagnosis
ENDO	=	Endodontics
EPT	=	Electric pulp test
Epin	=	Epinephrine
Final	=	Final file
Fx	=	Fracture
G.G.B.	=	Gates-Glidden bur
G.P.	=	Gutta-percha
I & D	=	Incision and drainage
I.P.	=	Irreversible pulpitis
L.A. or local	=	Local anesthetic
Mm	=	Millimeters
O+D	=	Open and drain
P.A.	=	Periapical
Perio	=	Periodontal
Post	=	Preformed, custom, or transilluminated post
Pt	=	Patient
Pulpec	=	Pulpectomy
R.D.	=	Rubber dam
Rel occ	=	Relieved occlusion

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resorp.	=	Resorption
Retro	=	Retrograde procedure
S/R	=	Suture removal
s.file	=	Serial filing
S/D	=	Single insurance or dual coverage
Sealer	=	Sealer type used
Sepr	=	Separated
Sutr	=	Suture
SX	=	Symptom
tech.	=	Technique for canal obturation
Temp	=	Temporary restoration
Test	=	Test file
Tx	=	Treatment
WNL	=	Within normal limits

Computerized treatment records

Clinicians are increasingly using computerized record storage as described in this chapter.[282] To avoid a claim of record falsification, whatever computer system is used should be able to demonstrate that records indicating earlier treatment were not recently falsified. Software technology, such as the “write only read memory” (WORM) system, used to identify computer data tampering, is not foolproof because it cannot detect tampering where an entire disk of recent origin has been substituted for an earlier version. Periodically, a hard copy of computer data should be printed out, hand initialed, dated in ink, and stored in the chart to provide written verification of the computer records if the office is a virtual paperless office.[307]

Health information technology for economic and clinical health act pitfalls

Affected patients must be notified if there is a breach in the electronic record storage system.[148] Electronic records have the following pitfalls:

- Data entry in the wrong patient record
- Incorrect data entry in the patient record

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Incorrectly entered information or information that was not entered at all was reported as the cause of the majority of errors. The absence of an undecipherable scrawl is designed to reduce errors. Whether the result of fat fingers or providers pushed for time, mistakes will still occasionally occur. Some of the things that make paperless electronic records desirable—transparency, the ability of more people to view the same information, and more immediate accessibility—may cause any electronic record mistakes risk to be amplified.

Health Insurance Portability and Accountability Act

The Department of Health and Human Services' Security and Electronic Signature Standards rule is part of the HIPAA of 1996. This act emphasizes the safeguarding of clinicians' health care information and permits a patient to obtain a copy of the requesting patient's records. Most states have authorized digital electronic signatures as binding for most contracts and orders. Digital signatures with electronic transactions are authorized by the Third Millennium Electronic Commerce Act (also called the Electronic Signature in Global and National Commerce Act).[323]

Pertinent HIPAA requirements follow:

1. Original records and radiographs should not be released but only copies provided to the patient. A reasonable copying charge may be requested before or after release of the copies.
2. A patient may amend health information in the chart that the patient believes to be incorrect or incomplete. The clinician may respond to the patient's proposed chart amendments.
3. Notice of office privacy practices should be provided to the patient, and the patient should acknowledge receipt in writing.
4. If privacy rights are violated, the patient can file a grievance with the secretary of the Department of Health and Human Services. Patients cannot be penalized for complaining.
5. Confidential dental and medical information may be shared with other clinicians, based on a need-to-know.
6. All e-mail communications between clinician and patient must use a secure network and be stored on a secure server.[282] A patient may

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waive this requirement and request communications and copies of records be sent via unencrypted email.

Record size

Although there is little harm in recording too much information, there is great danger in recording too little. Standard 8½ × 11 inch or larger clinical records possess the advantage of providing the treating clinician adequate space for clinical notes.

Identity of entry author

Either a clinician or an auxiliary can chart clinical entries unless state law indicates otherwise. Some states require clinicians and assistants to sign or initial each treatment entry.[58] What is important is that the correct clinical information is recorded. Each person who makes a clinical chart entry should record the date and initial the entry. Otherwise, the author's identity may be forgotten if the individual who recorded the entry is needed in a legal proceeding. For instance, initialing the entry makes it easier to identify the particular clinician or auxiliary who, after the original recording, is now employed elsewhere. A separate log of clinician and staff initials should be created and stored for later comparison to identify the entrant's initials (see Fig. 27.13).

Patient record request

Patient requests for record transfers or copies must be honored. It is unethical to refuse to transfer patient records, on the patient's request, to another treating clinician.[18] Moreover, refusing to provide patient records is illegal in some states, subjecting the clinician to discipline and fines should the records not be provided to the patient upon written request, even if the balance is outstanding.[62]

Patient education materials

Patient education pamphlets may be used in litigation as evidence that a patient was properly informed and given endodontic alternatives but instead chose extraction. Such pamphlets include the ADA's *Your Teeth Can Be*

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Saved by Endodontic Root Canal Treatment and the American Association of Endodontists' (AAE's) *Your Guide to Endodontic Treatment, Your Guide to Endodontic Retreatment,* and *Your Guide to Endodontic Surgery*. The clinician should indicate in the patient's chart which particular pamphlets the patient was provided or which video was shown, because this may prove useful should the patient later deny being informed of endodontic alternatives.

The use of educational materials may improve health behavior, enhance patient satisfaction, and facilitate the consultation processes. Consequently, the patient is better informed if patient questions are answered after prompting with informative educational materials.

Postoperative instructions

It is unlikely a patient will remember oral postoperative instructions unless accompanied with written instructions. After endodontic procedures, the patient may be sedated or affected by analgesic drugs. Accordingly, written postoperative instructions are beneficial. Emergency phone numbers to contact the treating clinician should be included on the form. Written instructions reduce postoperative morbidity and pain and improve patient compliance.[6] Document that both written and oral postoperative instructions were provided. If treating in close proximity to the IANC, place a precaution, such as seen in Fig. 27.14, in the postoperative instruction sheet.

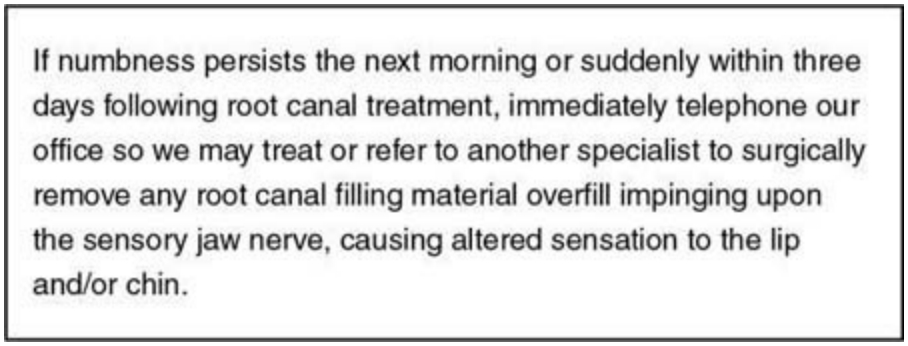

FIG. 27.14 Postoperative instruction sheet.

The text reads:

If numbness persists the next morning or suddenly within three days following root canal treatment, immediately telephone our office sol we may treat or

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refer to another specialist to surgically remove any root canal filling material overfill impinging upon the sensory jaw nerve, causing altered sensation to the lip and/or chin.

Recording referrals

Every clinician, including the endodontic specialist, has a duty to refer under appropriate circumstances. If consultations with additional experts or specialists become necessary, referrals should be recorded lest they be forgotten or refused. Carbonless, two-part referral cards allow the clinician to provide an original referral slip to the patient while retaining a copy for the patient's chart. The clinician or staff member should document the fact that the original referral card was given or mailed to the patient and record the name of the person who provided the referral card and the date on which it was provided. A copy of the referral card should also be sent to the referred doctor. If the patient fails to keep the referral appointment, this copy will provide proof that a referral was made. The clinician should request that the patient and the referred clinician report back if the referral appointment is canceled. Staff should calendar to verify that the referral consultation occurred.

Dental fees related to quality care

The link between health care fees and the quality of health care remains unclear.[156]

Record correction

Records must be complete, accurate, legible, and dated. All diagnoses, treatments, and referrals should be recorded. Chart additions may expand, correct, define, modify, or clarify (as long as they are currently dated to indicate a belated entry).

To correct an entry, the clinician should draw a line through (but not erase or obscure) the erroneous entry. The correction should then be written on the next available line and dated. Handwriting and ink experts use ink chemical tags, age dating, and infrared technology to prove falsified additions, deletions, or substitutions. If records are proved to be falsified, the clinician

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may be subject to punitive damages in civil litigation. In addition, the clinician may be subject to license revocation for intentional misconduct.[58] Professional liability insurance policies may defend but will usually not indemnify punitive damages if the clinician is found to have committed fraud or deceit.[56,57,61,77] In addition, insurance carriers may deny renewal of professional liability coverage to a clinician who fraudulently alters dental records.

If an erroneous entry occurs, the clinician should add another entry dated as a late entry to demonstrate later corrected information. Fig. 27.15 gives an example of the proper method of belated record correction.

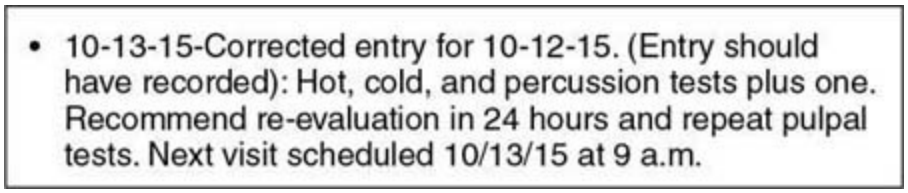

FIG. 27.15 Belated record correction.

The record correction is as follows:

• 10-13-15-Corrected entry for 10-12-15. (Entry should have recorded): Hot, cold, and percussion tests plus one. Recommend re-evaluation in 24 hours and repeat pulpal tests. Next visit scheduled 10/13/15 at 9 a.m.

When patient records have been requested or subpoenaed, it is wise to refrain from examining in detail prior to copying to avoid the temptation to clarify or belatedly add an entry. Alteration of clinical records is a cause of large settlements because the clinician's credibility is suspect. Also the jury may be similarly instructed. For instance, California Jury Instruction 204 regarding "Willful Suppression of Evidence" states as follows:

You may consider whether one party intentionally concealed or destroyed evidence. If you decide that a party did so, you may decide that the evidence would have been unfavorable to that party.

Spoliation

Spoliation is the destruction or significant alteration of evidence, or the failure to preserve property for another's use as evidence, in pending or

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reasonably foreseeable litigation under Fed. R. Civ. P. 37(b)(2)(C).[103]

Spoliation occurs when the wrongdoer alters, changes, or substitutes dental records in an attempt to defeat a civil lawsuit.[64,322] It is far easier to defend a dental negligence lawsuit with poor records compared to attempting to justifiably defend altered (falsified) records. A jury and/or a judge will likely conclude the clinician acted with consciousness of guilt for falsifying records compared with excusing staff clerical oversight if records are poorly maintained. Dental records, like all business records, deserve accuracy and completeness.

Questioned document experts utilize ink age dating, examine watermarks, or apply infrared techniques to ascertain substituted pages and additions or deletions in records, including prior erasures, entries underneath whiteouts, and indentations made when one page is overwritten on another page. Infrared technique analyzes these patterns of indentations underneath the altered pages that detect belated second entries. In one case, the defendant clinician claimed a written referral was made to a specialist in 1998, but the patient refused the referral. During litigation, the patient denied the referral and subpoenaed records of the print shop where the specialist's referral forms were printed. These subpoenaed records proved the carbon copy of the form used for the purported 1998 referral was instead first printed in 2000, which was 2 years after the alleged specialist referral was made (Fig. 27.16, *A* and *B*).

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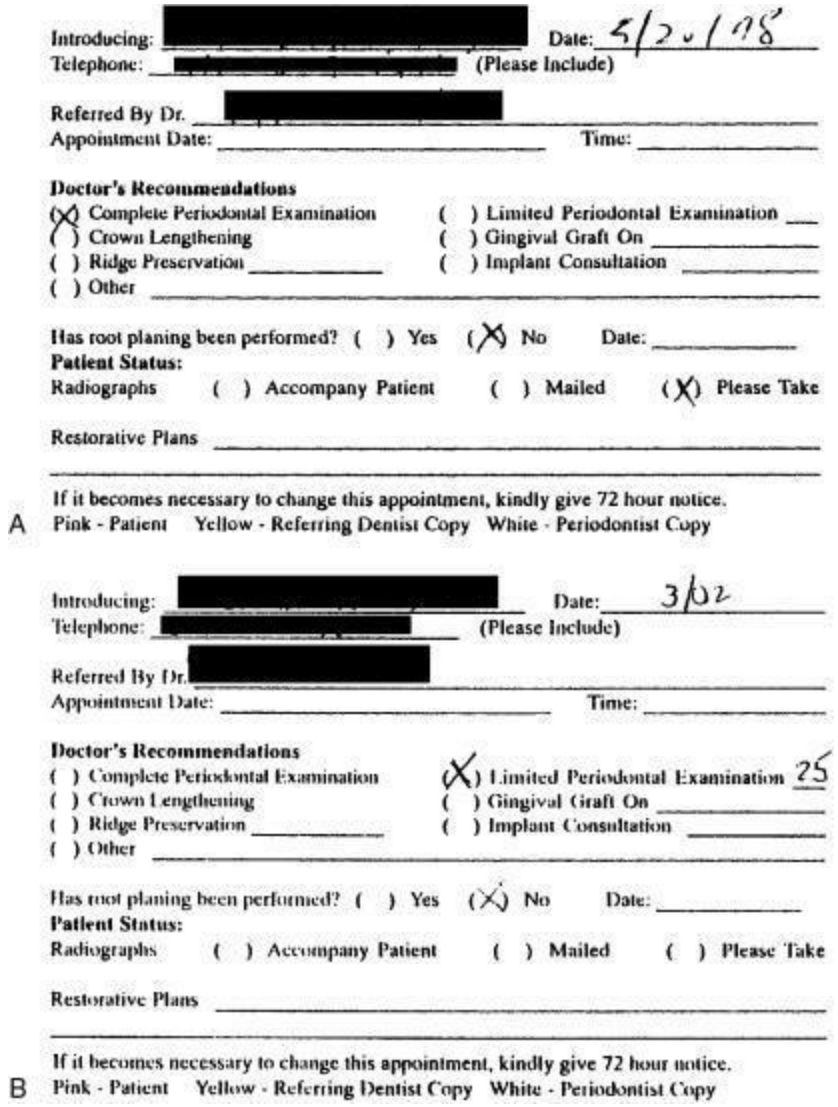

Introducing: [redacted] Date: 5/20/78
Telephone: [redacted] (Please Include)

Referred By Dr. [redacted]
Appointment Date: _____ Time: _____

Doctor's Recommendations
(X) Complete Periodontal Examination ( ) Limited Periodontal Examination ___
( ) Crown Lengthening ( ) Gingival Graft On _____
( ) Ridge Preservation _____ ( ) Implant Consultation _____
( ) Other _____

Has root planing been performed? ( ) Yes (X) No Date: _____
Patient Status:
Radiographs ( ) Accompany Patient ( ) Mailed (X) Please Take

Restorative Plans _____

If it becomes necessary to change this appointment, kindly give 72 hour notice.
Pink - Patient Yellow - Referring Dentist Copy White - Periodontist Copy

A

Introducing: [redacted] Date: 3/02
Telephone: [redacted] (Please Include)

Referred By Dr. [redacted]
Appointment Date: _____ Time: _____

Doctor's Recommendations
( ) Complete Periodontal Examination (X) Limited Periodontal Examination 25
( ) Crown Lengthening ( ) Gingival Graft On _____
( ) Ridge Preservation _____ ( ) Implant Consultation _____
( ) Other _____

Has root planing been performed? ( ) Yes (X) No Date: _____
Patient Status:
Radiographs ( ) Accompany Patient ( ) Mailed ( ) Please Take

Restorative Plans _____

If it becomes necessary to change this appointment, kindly give 72 hour notice.
Pink - Patient Yellow - Referring Dentist Copy White - Periodontist Copy

B

FIG. 27.16 **A,** Falsified records with belated entry of year of referral. **B,** Falsified records.

A) Falsified records with belated entry are as follows: introducing (black bar), date (5/20/78), telephone (black bar) (please include), referred by doctor (black bar), appointment date, time.

Doctor's recommendations:

Complete periodontal examination (cross mark), crown lengthening, ridge preservation, other, limited periodontal examination, gingival graft on, implant consultation.

Has root planning been performed? Yes, No (cross mark), date.

Patient status: radiographs, accompany patient, mailed, please take (cross mark). Restorative plans. If it becomes necessary to change this appointment, kindly give 72 hours notice. Pink-patient, yellow-referring dentist copy, white-

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periodontist copy.

B) Falsified record are as follows: introducing (black bar), date (3/02), telephone (black bar) (please include), referred by doctor (black bar), appointment date, time.

Doctor's recommendations:

Complete periodontal examination crown lengthening, ridge preservation, other, limited periodontal examination (25) (crossed mark), gingival graft on, implant consultation.

Has root planning been performed? Yes, No (cross mark), date.

Patient status: radiographs, accompany patient, mailed, please take (cross mark). Restorative plans. If it becomes necessary to change this appointment, kindly give 72 hours notice. Pink-patient, yellow-referring dentist copy, white-periodontist copy.

In another example, altered records were detected in nine different entries in one medical negligence case involving an undiagnosed malignancy. The physician's carrier subsequently settled for $1 million. Also, the defendant personally paid a separate fine to the court. As part of the settlement, the defendant authored an article for the Washington Medical Association detailing why records falsification is wrong.[29]

Records are subjected to (1) audits by insurance carriers for documentation that treatment was performed, (2) review by peer review committees, and (3) subpoena by state licensing boards or agencies for disciplinary proceedings. Accordingly, falsified records expose the clinician not only to civil liability for professional negligence but also to criminal penalties for criminal offenses, such as insurance fraud.[64,313,318,322,323]

Deliberate record alteration with intent to deceive may also subject the clinician to licensing[58] and ethical discipline as well as punitive damages for deceitful misconduct. Insurance carriers may defend but not indemnify a verdict for intentional material alteration of dental records done with intent to deceive.

Digital radiography may have dental advantages, but because the digital images originally may be computer manipulated, they may be legally suspect as altered.[168,313] Therefore hard copies of the digital images may also be printed and dated in ink to show the informational baseline upon which the

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clinician based diagnostic or therapeutic decisions. This also protects against computer glitches, such as disk drive crashes, electrical power surges, computer viruses, and operator delete errors.

In one instance, a judgment of $641,541.65 was awarded for negligently designed and prepared veneers and crowns with resulting endodontics from some teepee appearing restorations. The dentist claimed she spilled Diet Coke on her records, which compromised inspection by a questioned document expert. Her defense that the flood of muddy water caused the dentist to discard plaintiff's study models was refuted by demonstrating that the models could have been easily washed and preserved.[324] Subsequently the dentist surrendered her dental license with pending disciplinary allegations concerning the patient who obtained the judgment along with six other patients of which some were alleged to be have had unnecessary dental restorations necessitating root canals and restoration remake.

False claims

Performing or billing for unnecessary endodontic therapy, such as prophylactic endodontic therapy with every crown preparation, subjects the clinician to fraud. In non–pulpal exposure crown preparation, the likely incidence of subsequent endodontic therapy is approximately 3%. Therefore performing prophylactic endodontics on the other 97% of patients represents unnecessary and therefore fraudulent treatment. Another example of doing unnecessary endodontic therapy is treating incipient caries of a deciduous molar with pulpotomy rather than a filling or monitoring for caries progression until the tooth exfoliates. Excessive unnecessary treatment also ethically violates the Hippocratic oath of "primum non nocere" ("first, do no harm"). The ADA Code of Professional Conduct, Section 5—Principle: Veracity ("truthfulness") in 5.A requires "dentists shall not represent the care being rendered to their patients in a false or misleading manner."

The Federal False Claims Act carries both civil and general penalties of treble damages, fines, and attorneys' fees for fraudulently billing government programs, such as the Civilian Health and Medical Program of the Uniformed Services (CHAMPUS) or Medicare. Fines between $5000 and $10,000 per claim form apply if the U.S. mail was used for a false-claim submission. If any portion of the claimed treatment was fraudulently misrepresented, even if

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a small minority, a violation results.[322] Fraudulent intent need not be conclusively proved. Reckless disregard for the accuracy of submitted data is all that is necessary to obtain a federal criminal conviction or prove civil wrongdoing.[314]

Legal responsibilities

Malpractice claims

More than 42% of physicians have been sued over the course of their careers. Before reaching the age of 40, more than half of obstetricians/gynecologists have already been sued. About 90% of general surgeons aged 55 and over have been sued.[22]

Incidence of negligence

Medical errors or adverse events are estimated to occur in almost one out of four patient visits to a family practitioner.[270] A U.S. government study of the Institute of Medicine found that 152,000 avoidable deaths occur annually because of medical negligence.[118,120] In this government study, injury from medical care occurred in 3.7% of hospital admissions. Some 58% of these injuries were concluded to be preventable, and 13.6% were fatal injuries. The actual fatality numbers may be underestimated, because calculations were based solely on hospital chart review.

Malpractice prophylaxis: Importance of records

Good clinicians keep good records. Records represent the single most critical evidence a clinician can present in court as confirmation that an accurate diagnosis, properly planned treatment, and informed consent were provided.

Prevention is the goal of modern dental care. Competent endodontic treatment performed within the requisite standard of care not only saves endodontically treated teeth but also helps prevent a lawsuit for professional negligence. Thus sound and carefully applied endodontic principles protect both patient and clinician. Prudent practices reduce avoidable and unreasonable risks associated with imprudent or careless endodontic diagnosis or therapy.

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Not all examples of negligent endodontic treatment are included in this chapter, because the myriad of malpractice incidents far exceeds its scope and length. Rather, only select examples are elucidated for educational purposes.

Standard of care

Good endodontic practice, as defined by the courts, is the standard of reasonable care that reasonably careful clinicians legally are required to comply. The standard of care does not require perfection. Instead, the legal standard is the reasonable degree of skill, knowledge, and care ordinarily possessed and exercised by reasonably careful clinicians under the same or similar circumstances.[172]

Although the standard of care is flexible to accommodate individual variations in treatment, it is objectively tested based on what a reasonable clinician would do under the same or similar circumstances. Reasonable conduct represents a minimum standard required for legal due care. Reasonable care is care based on reasoning supported by sound science such as evidence-based, peer-reviewed literature. Additional precautionary steps that rise above this minimal floor of reasonableness and ascend to achieve ideal care are laudable but not legally mandated. Again, the standard of care does not require perfection; nor does it require ideal endodontics. Nevertheless, prudent clinicians should always strive to achieve a higher level of care than barely or minimally adequate care. Excellent clinicians always strive to do their best and practice endodontics at the highest level. Some have criticized this concept by contending that average care is below average, because 49% of clinicians represent a minority compared to the majority of clinicians. Careful clinicians should strive to avoid the mediocrity of minimally acceptable care and instead zealously pursue a goal of endodontic excellence. Endodontists set the standard of care in endodontics. Accordingly, a general dentist and endodontist must each meet the same standard of care in rendering endodontic therapy.

Negligent errors are unintended acts, either of omission or commission. Some errors do not lead to patient injury if intercepted. Failure to mitigate factors that contribute to errors or near misses may combine with human fallibilities of mood, inattention, carelessness, and forgetfulness to cause error repetition or a future more serious injury.

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The written policies and procedures of a clinician, institution, or organization may be used as evidence of standard of care. An expert may consider these the appropriate standard of care in testifying about alleged breaches of standard of care on that issue. For instance, an expert may rely upon AAE's 1998 position paper on paraformaldehyde-containing root canal filling materials as substandard practice, as well as the fact that all U.S. dental schools teach that toxic paraformaldehyde substances should not be used for adult patients as a root canal obturant.

Standards of care: Generalist versus endodontist

A general practitioner performing treatment ordinarily performed exclusively by specialists—such as apical endodontic surgery, periodontal surgical grafting, or full bony impaction surgery—will be held to the specialist's standard of care.[131,132] To avoid performing treatment that is below the specialist's standard, a generalist should refer to a specialist rather than perform procedures that are beyond the general practitioner's training or competency. The three levels of clinical skill are (1) competency or beginning level, (2) proficiency, and (3) mastery. To err is human. Even specialists may not always know what they believe they know.[98,179] Approximately 80% of general practitioners in the United States provide some endodontic therapy. Endodontic expansion into the realm of the generalist can be linked to (1) refinements in root canal preparation and improved obturation (i.e., packing) techniques currently taught in dental schools, (2) continuing education courses, and (3) significant improvements in the armamentarium of instruments, equipment, and materials available to all clinicians to optimize efficiencies in endodontics.

The AAE's Guidelines Case Difficulty Assessment Form and Guidelines provide a road map for when a generalist should treat or refer to an endodontist. Levels of difficulty range from minimal to moderate and high difficulty (Fig. 27.17). General dentists are competent to treat cases of minimal difficulty. Experienced general dentists may treat the moderate-difficulty cases but should refer high-difficulty patients.

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AAE Endodontic Case Difficulty Assessment Form and Guidelines

PATIENT INFORMATION

Name_____

Address_____

City/State/Zip_____

Phone_____

DISPOSITION

Treat in Office: Yes ☐ No ☐

Refer Patient to:

_____

Date_____

Guidelines for Using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

LEVELS OF DIFFICULTY

MINIMAL DIFFICULTY	Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the MINIMAL DIFFICULTY category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.
MODERATE DIFFICULTY	Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the MODERATE DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.
HIGH DIFFICULTY	Preoperative condition is exceptionally complicated, exhibiting several factors listed in the MODERATE DIFFICULTY category or at least one in the HIGH DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

The contribution of the Canadian Academy of Endodontics and others to the development of this form is gratefully acknowledged.

The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way.

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AAE Endodontic Case Difficulty Assessment Form

Criteria and Subcriteria	Minimal Difficulty	Moderate Difficulty	High Difficulty
A. PATIENT CONSIDERATIONS			
Medical history	☐ No medical problem (ASA Class 1*)	☐ One or more medical problems (ASA Class 2*)	☐ Complex medical history/serious illness/disability (ASA Classes 3-5*)
Anesthesia	☐ No history of anesthesia problems	☐ Vasoconstrictor intolerance	☐ Difficulty achieving anesthesia
Patient disposition	☐ Cooperative and compliant	☐ Anxious but cooperative	☐ Uncooperative
Ability to open mouth	☐ No limitation	☐ Slight limitation in opening	☐ Significant limitation in opening
Gag reflex	☐ None	☐ Gags occasionally with radiographs/treatment	☐ Extreme gag reflex which has compromised past dental care
Emergency condition	☐ Minimum pain or swelling	☐ Moderate pain or swelling	☐ Severe pain or swelling
B. DIAGNOSTIC AND TREATMENT CONSIDERATIONS			
Diagnosis	☐ Signs and symptoms consistent with recognized pulpal and periapical conditions	☐ Extensive differential diagnosis of usual signs and symptoms required	☐ Confusing and complex signs and symptoms; difficult diagnosis ☐ History of chronic orofacial pain
Radiographic difficulties	☐ Minimal difficulty obtaining/interpreting radiographs	☐ Moderate difficulty obtaining/interpreting radiographs (e.g., high floor of mouth, narrow or low palatal vault, presence of tori)	☐ Extreme difficulty obtaining/interpreting radiographs (e.g., superimposed anatomical structures)
Position in the arch	☐ Anterior/premolar ☐ Slight inclination (<10°) ☐ Slight rotation (<10°)	☐ 1st molar ☐ Moderate inclination (10-30°) ☐ Moderate rotation (10-30°)	☐ 2nd or 3rd molar ☐ Extreme inclination (>30°) ☐ Extreme rotation (>30°)
Tooth isolation	☐ Routine rubber dam placement	☐ Simple pretreatment modification required for rubber dam isolation	☐ Extensive pretreatment modification required for rubber dam isolation
Crown morphology	☐ Normal original crown morphology	☐ Full coverage restoration ☐ Porcelain restoration ☐ Bridge abutment ☐ Moderate deviation from normal tooth/root form (e.g., taurodontism, microdens) ☐ Teeth with extensive coronal destruction	☐ Restoration does not reflect original anatomy/alignment ☐ Significant deviation from normal tooth/root form (e.g., fusion, dens in dente)
Canal and root morphology	☐ Slight or no curvature (<10°) ☐ Closed apex (<1 mm in diameter)	☐ Moderate curvature (10-30°) ☐ Crown axis differs moderately from root axis. Apical opening 1-1.5 mm in diameter	☐ Extreme curvature (>30°) or S-shaped curve ☐ Mandibular premolar or anterior with 2 roots ☐ Maxillary premolar with 3 roots ☐ Canal divides in the middle or apical third ☐ Very long tooth (>25 mm) ☐ Open apex (>1.5 mm in diameter)
Radiographic appearance of canal(s)	☐ Canal(s) visible and not reduced in size	☐ Canal(s) and chamber visible but reduced in size ☐ Pulp stones	☐ Indistinct canal path ☐ Canal(s) not visible
Resorption	☐ No resorption evident	☐ Minimal apical resorption	☐ Extensive apical resorption ☐ Internal resorption ☐ External resorption
C. ADDITIONAL CONSIDERATIONS			
Trauma history	☐ Uncomplicated crown fracture of mature or immature teeth	☐ Complicated crown fracture of mature teeth ☐ Subluxation	☐ Complicated crown fracture of immature teeth ☐ Horizontal root fracture ☐ Alveolar fracture ☐ Intrusive, extrusive or lateral luxation ☐ Avulsion
Endodontic treatment history	☐ No previous treatment	☐ Previous access without complications	☐ Previous access with complications (e.g., perforation, non-negotiated canal, ledge, separated instrument) ☐ Previous surgical or nonsurgical endodontic treatment completed
Periodontal-endodontic condition	☐ None or mild periodontal disease	☐ Concurrent moderate periodontal disease	☐ Concurrent severe periodontal disease ☐ Cracked teeth with periodontal complications ☐ Combined endodontic/periodontic lesion ☐ Root amputation prior to endodontic treatment

*American Society of Anesthesiologists (ASA) Classification System

Class 1: No systemic illness. Patient healthy.
Class 2: Patient with mild degree of systemic illness, but without functional restriction, e.g., well-controlled hypertension.
Class 3: Patient with severe degree of systemic illness which limits activities, but does not incapacitate the patient.
Class 4: Patient with severe systemic illness that immobilizes and is sometimes life threatening.
Class 5: Patient will not survive more than 24 hours whether or not surgical intervention takes place.

www.aaehq.org/clinical/physicalstatus.html

FIG. 27.17 American Association of Endodontists (AAE) Endodontic Case Difficulty Assessment Form and Guidelines.

AAE Endodontic Case Difficulty Assessment Form and Guidelines are shown along with AAE logo at the upper left corner.

The first panel shows patient information including its name, address, city/state/zip, and phone. To the right, the information under the heading disposition is as follows: treat in office (Yes or no each with blank check box), refer patient to, date.

The second panel shows guidelines for using the AAE Endodontic Case Difficulty Assessment Form

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record

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keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

Levels of difficulty are as follows:

Minimal difficulty: Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the minimal difficulty category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.

Moderate difficulty: Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the moderate difficulty category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.

High difficulty: Preoperative condition is exceptionally complicated, exhibiting several factors listed in the moderate difficulty category or at least one in the high difficulty category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

Text at the bottom reads, The contribution of the Canadian academy of endodontics and others to the development of this form is gratefully acknowledged.

The AAE Endodontic Case Difficulty Assessment Form is designed to aid the practitioner in determining appropriate case disposition. The American Association of Endodontists neither expressly nor implicitly warrants any positive results associated with the use of this form. This form may be reproduced but may not be amended or altered in any way. It is followed by the address of AAE.

The continuation of AAE Endodontic Case Difficulty Assessment Form shows three sections as follows: A) Patient considerations, B) Diagnostic and treatment considerations, C) Additional considerations. Each section has list of queries under criteria and subcriteria, minimal difficulty, moderate difficulty,

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and high difficulty columns.

Ethical guidelines

The ADA's Code of Ethics guides conduct that distinguishes the dental profession from a trade by placing patient service paramount and profit secondary.[122] Section 5 of the ADA Code of Ethics regarding veracity ("truthfulness") states, "Professionals have a duty to be honest and trustworthy in their dealings with people." Accordingly, both ethically and legally,[159] clinicians are obligated to communicate truthfully and without deception to patients. Announcements to the public must be truthful so that the average person is not materially misled. Truth in dentistry is the rule and not the exception.[122]

Section 5.I of the ADA Code of Ethics provides the following:

> *General Practitioner Announcement... general clinicians who wish to announce the services available in their practices are permitted to announce the availability of those services so long as they avoid any communications that express or imply specialization.*

Thus clinicians should not misrepresent or overstate the facts of their training and competence in any way that would be false or misleading in any material or significant manner to the public or their peers. General clinicians should avoid false and misleading self-representation regarding endodontic "superiority." Implying credentials that the generalist lacks misrepresents a general clinician's endodontic skill and knowledge as equivalent to a board-eligible or board-certified endodontist. A generalist's 30-year experience may be little more than 1 year of experience repeated 30 times. Every state has specific laws governing advertising. Consult your state's law, because each state deals differently with specialty announcements.

Telephone directory listings are organized into categories based on types of procedure, such as endodontics. Generalists must also state in such ads that endodontic services are being provided by general clinicians. The public is entitled to be informed of the distinction between an endodontist specialist

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and a generalist, although each provides endodontics. Only an ADA-recognized board-eligible or board-certified clinician is entitled to represent him or herself as an endodontist. With this distinction in mind, the ADA-recognized endodontic specialty organization is titled the American Association of Endodontists, not the American Association of Endodontics.

Standard of care for endodontics

Endodontists set the standard for routine endodontics. Therefore if the endodontist's standard of care cannot be met, such as the need for microscopy or apical surgery, the generalist should refer the patient to an endodontist.[76,131,132,183]

Endodontists should not forget their general dentist training. Even though a patient may be referred for a specific procedure or undertaking, the endodontist should not overlook sound biologic principles inherent in the overall treatment. A specialist may also be held liable for relying solely on the information referral card or radiographs of the referring clinician if the diagnosis or therapeutic recommendations prove incorrect, unnecessary, or the referral card lists the wrong tooth for treatment. Therefore conduct a "time-out" with patient and staff to verify which tooth is to be treated.[167]

Fig. 27.18 demonstrates a general clinician's radiograph showing apparent complete endodontic fill within the canal space, whereas Fig. 27.19 demonstrates an endodontist's radiograph showing a transported canal. This difference is explained by radiographic quality and angulation. Taking radiographs at different angles improves radiographic accuracy.[201,204]

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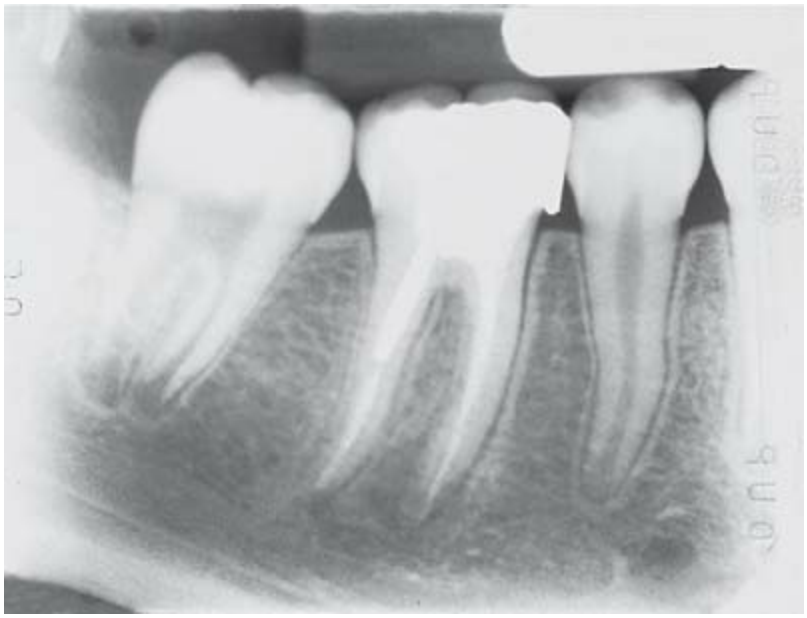

FIG. 27.18 A general clinician's radiograph that ostensibly shows a complete endodontic fill within the root canal space.

Radiograph shows three teeth where the central tooth has filled root canals and the adjacent teeth on both sides each have unfilled root canals. The tooth on the right is extremely radiopaque. The surrounding tissue of all teeth has less bone density.

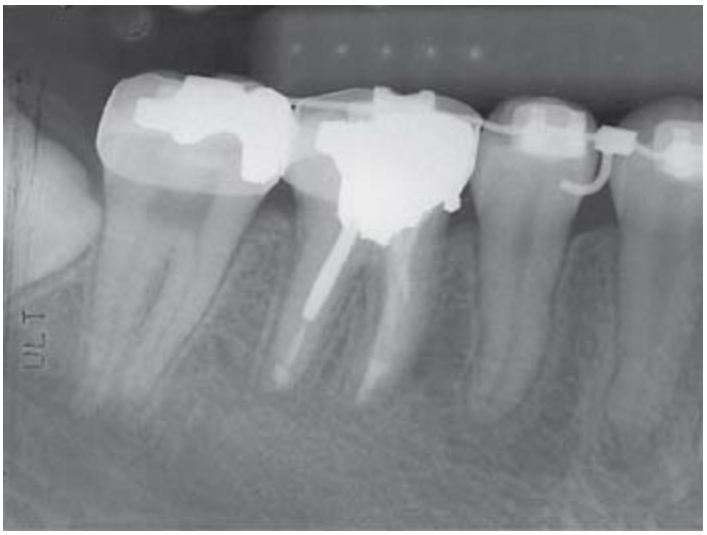

FIG. 27.19 Endodontist's radiograph demonstrating a transported canal.

Radiograph shows transported canal passing horizontally above the crowns of teeth. All teeth except second have unfilled root canals.

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Endodontists should not provide rubber-stamp treatment to whatever the clinician refers or recommends. Without performing an independent examination, the endodontist risks misdiagnosis and resulting incorrect treatment. Prevention of misdiagnosis or incorrect treatment requires an accurate medical and dental history preceding a clinical examination (not only of the specific tooth or teeth involved but also of the general oral condition). Obvious problems such as oral lesions, periodontitis, or gross decay should be noted in the chart and the patient advised regarding a referral for further examination, testing, or another specialist's consultation.

Radiographs from the referring clinician should be reviewed for completeness, clarity, and diagnostic accuracy. An endodontist should expose a new radiograph to verify current status before treatment and only use the referring clinician's radiograph for historical comparison. Unfortunately, the referring clinician may surreptitiously forward a pretreatment film with the referred patient, rather than the referring clinician's own posttreatment film depicting a perforation or broken file that necessitated the referral. This may occur whenever an errant clinician attempts to fraudulently conceal negligently performed endodontic treatment in an attempt to shift blame for bungled treatment to the endodontist. In such cases, a valuable lesson is learned when the endodontist exposes independent pretreatment radiographs, rather than relying exclusively on the referring clinician's radiographs to determine current status of endodontic treatment.

Poor oral hygiene may contribute to periodontal disease. In such cases, endodontic treatment may be compromised unless the associated periodontal condition, along with the tooth being treated endodontically, is brought under periodontal disease control, including any crown-lengthening needs (see Chapter 25 for more information). Referral to a periodontist may be necessary before or contemporaneous with completion of endodontic treatment.

In summary, it is necessary for the endodontist to do the following:

1. Be alert to any contributory medical or dental condition within the operative area of endodontic treatment that can affect treatment.
2. Undertake an independent diagnostic and radiographic examination of the treatment area and treatment plan rather than relying solely on

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the referring clinician.
3. Perform a general dental examination (at least a screening) to diagnose any hard- and soft-tissue pathosis.
4. Evaluate status and prognosis of adjacent and opposing teeth.
5. Advise the referring clinician and patient of pertinent findings.

Ordinary care equals prudent care

Ordinary is commonly understood (outside its legal context) to mean "lacking in excellence" or "being of poor or mediocre quality."[217] As expressed in the context of actions for negligence, however, ordinary care has assumed a technical legal definition that differs somewhat from its common meaning. The eighth edition of *Black's Law Dictionary* describes ordinary care as "that degree which persons of ordinary care and prudence are accustomed to use or employ... that is, reasonable care."[46]

In adopting this distinction, the courts have defined *ordinary care* as "that degree of care which people ordinarily prudent could be reasonably expected to exercise under circumstances of a given case."[108] It has been equated with the reasonable care and prudence exercised by ordinarily prudent clinicians under similar circumstances.[136,265] It is not extraordinary or ideal care. Thus ordinary care is not average care but instead equates with prudently careful care. Stated otherwise, average care may be below the average of what all reasonable clinicians should practice.

Although the standard required of a professional cannot be only that of the most highly skilled practitioner, neither can it be limited to the arithmetic-average member of the profession, because those who possess somewhat less than median skill may still be competent and qualified to treat.[265] By such an illogical definition of average care, half of all clinicians would automatically fall short of the mark and be negligent as a matter of law. As one court explained, "We are not permitted to aggregate into a common class the quacks, the young men who have not practiced, the old ones who have dropped out of practice, the good, and the very best, and then strike an average between them."[281] Thus the standard of care equates with reasonable care and not average care.

Customary practice versus negligence

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Customary practice may constitute evidence of the standard of care, but it is not the only determinant. Moreover, if the customary practice constitutes negligence, it is not considered reasonable (although or even if customarily practiced by a majority of clinicians).[38] Rather, the reasonably careful clinician is the standard of care and not an average or mediocre practitioner. For instance, a majority of clinicians did not perform biologic testing of the dental unit waterlines despite ADA recommendations to do so.[220,222] However, careful clinicians did follow the ADA's recommendation (Box 27.2 and Fig. 27.20).

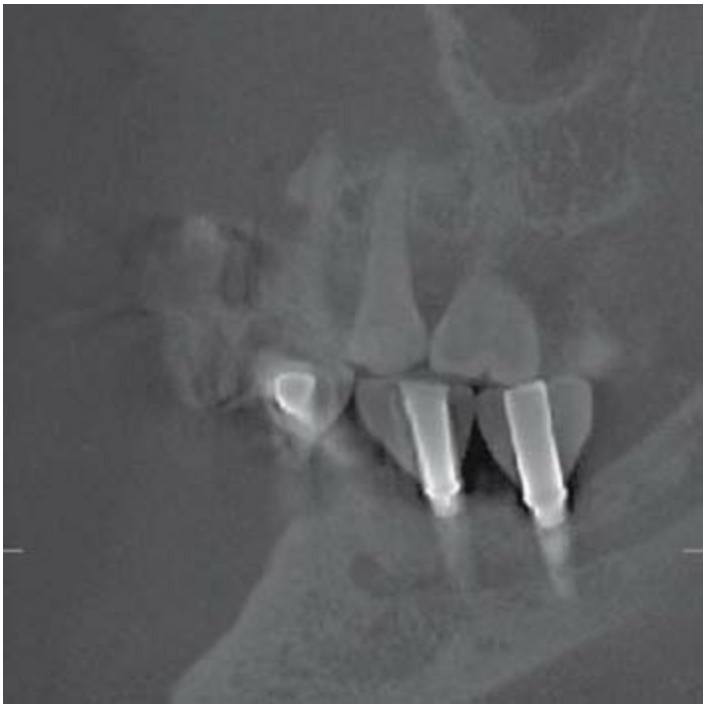

FIG. 27.20 Implant "harpooned" inferior alveolar nerve canal.

Radiograph shows a teeth quadrant where harpoons are implanted in three teeth of lower jaw.

Box 27.2

Typical Examples of Negligent Customs

- Failing to probe and record periodontal pockets

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- Failing to take diagnostic-quality radiographs
- Failing to refer patients for complicated procedures
- Failing to use aseptic practices such as gloves, facemasks, and sterile fluids for surgery
- Failing to use rubber dams for root canal therapy
- Failing to install or periodically check valves in dental units to prevent water retraction, suck-back, and cross-contamination
- Failing to do thermal pulp testing as an aid to diagnosing pulpal disease (see Chapter 1)
- Failing to discontinue quaternary ammonium chloride–based products for precleaning or disinfection of environmental surfaces
- Failing to use a microscope for fractured file retrieval and PRS
- Failure to include CAMBRA in a comprehensive new patient exam
- Failure to use CBCT technology to guide implant placement when placement is in close proximity to the IANC (see Fig. 27.21 for implant "harpooning" of IANC)
- Not informing CT radiation risks to patient [171]

CAMBRA, Caries management by risk assessment; CBCT, cone-beam computed tomography; IANC, inferior alveolar nerve canal.

Data from American Dental Association Council on Scientific Affairs: The use of prophylactic antibiotics prior to dental procedures in patients with prosthetic joints. J Am Dent Assoc 4:11, 2015; ADA Caries Risk assessment forms. Assertion additional resources, J Am Dent Assoc Jan 12, 2009, update; Cheung G: Endodontic failures, Int Dent J 146:131, 1996; Cotton TP, Geisler TM, Holden DT, et al: Endodontic applications of cone-beam volumetric tomography, J Endod 33:1121, 2007; Fontana M, Zero D: Assessing patients' caries risk, J Am Dent Assoc 137:1231, 2006; Kersten DD, Mines P, Sweet M: Use of the microscope in endodontics: results of a questionnaire, J Endod 34:804, 2008; Kutsch V, Milicich G, Domb W, et al: How to integrate CAMBRA into private practice, J Calif Dent Assoc 2007; Lyon TC: Quaternary ammonia compounds: should they be used for disinfection in the dental office? J Dist Columbia Dent Soc 48:10, 1973; Occupational Safety and Health Administration (OSHA): Occupational

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Safety and Health Administration Directive, Washington DC, 1999. Available at: www.OSHA.90V; Rosenbaum S, Frankford DM, Moore B: Who should determine medical care necessity? N Engl J Med 340:229, 1999; Simonsen R: Greed and gravy train: is this success? J Esthet Dent 11:287, 1999; Young D, Featherstone J, Roth J, et al: Consensus statement caries management by risk assessment: implementation guidelines to support oral health, J Calif Dent Assoc 35:799, 2007; Zeider S, Ruttimann U, Webber R: Efficacy in the assessment of intraosseous lesions of the face and jaws in asymptomatic patients, Radiology 162:691, 1987.

Merely because a majority of clinicians in a community practice a particular method does not establish it as the standard of care if the practice is unreasonable or imprudent.[38] Ultimately, the courts determine what constitutes reasonable practice by considering available dental knowledge and evaluating the relative risks versus benefits of a particular procedure.

Again, the law does not require dental perfection.[311] Instead, the legal yardstick by which prudent conduct is measured is what a reasonably prudent clinician should do under the same or similar circumstances, regardless of how many or how few clinicians conform to this standard.

In a seminal case, it was not customary practice in the state of Washington for ophthalmologists to test patients under 40 years old for glaucoma, because the incidence was only 1 in 25,000 patients. Nevertheless, the Supreme Court of Washington State held that the defendant ophthalmologist was negligent as a matter of law, irrespective of customary medical practice, as a negligent customary practice is no defense.[150,213]

Little excuse exists for failing to routinely probe and chart periodontal pockets before rendering endodontic therapy, no matter how many other clinicians in the community may fail to do so. The benefit of probing for periodontal disease substantially outweighs the virtually nonexistent risk of conducting this valuable diagnostic procedure. A legal defense likely to invoke a jury's wrath is to claim that a necessary diagnostic or prophylactic procedure is "too time consuming," when dental and medical patient health are placed at risk if not done. If the benefit outweighs the risk, a reasonable clinician complying with the standard of care should opt for the beneficial diagnostic test or treatment. For instance, the Accuitomo and Kodak 9000

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CBCT may prove an invaluable benefit for diagnosing the etiology and source of endodontic pain when compared to the relatively low radiation risk. Thus the CBCT diagnostic benefit outweighs the radiation risk if a differential diagnosis cannot accurately rule in or rule out a final diagnosis.[203]

Another example of negligent customary practice includes a majority of general dentists not using a rubber dam for all root canal therapy despite virtually all endodontists who do so. The AAE's 2010 Position Statement mandates that "tooth isolation using the dental dam is the standard of care; it is integral and essential for any nonsurgical endodontic treatment."

A further instance of a customary but nevertheless negligent practice concerns the majority of dentists who did not adhere to the 2003 CDC Guidelines for Infection Control in Dental Health-Care Settings.[70]

Health maintenance organization care versus standard of care

Reasonably careful or prudent practitioners (not insurance carriers) set the standard of care. Third-party payers may limit reimbursement but should not limit access to quality care. Clinicians have an affirmative duty on behalf of patients to appeal insurance carrier care denial decisions and, in some states, are protected against retaliation.[332] The clinician who complies (without protest) with restrictive limitations or denials imposed by a third-party payer when sound judgment dictates otherwise cannot avoid ultimate responsibility for the patient's care.[332]

Although dental insurance companies do not set the standard of care, insurance carriers may contractually limit dental benefits. Therefore a clinician is obligated to inform patients of their dental needs, regardless of carrier reimbursement. Patients may then elect to pay out of pocket for noncovered treatment or decline uninsured services. Informed choice is uninformed if the clinician fails to provide patients with all reasonable options and alternatives.[209] Follow the adage to not x-ray the patient's pocket book or wallet to determine what options the patient should be presented. Instead offer the patient all reasonable treatment options.

Notwithstanding that an insurance carrier denies endodontic therapy or limits endodontics to only certain clinical conditions, a prudent clinician must nevertheless, both legally and ethically, provide an informed consent option to the patient that a tooth may be endodontically treated and retained rather

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than extracted.[71,220,288,303,346] The California Dental Association's Dental Patient Bill of Rights advises patients: "You have the right to ask your clinician to explain all treatment options regardless of coverage or cost."[60]

Clinicians may agree to a discounted fee with a health maintenance organization (HMO) carrier but must never discount the quality of care provided. Peer reviews and the courts recognize only one standard of care. A lower, double, or different standard of care for reduced-fee HMO plans is not legally recognized. Expediency should not be exercised at the expense of quality patient care. Section 3 of the ADA Code of Ethics affirms, "Managed care contract obligations do not excuse clinicians from their ethical duty to put the patient's welfare first."

Plans that delay treatment approval, resulting in endodontic complications or tooth nontreatability, may be subject to liability.[233,303]

Capitation systems have a built-in incentive to undertreat, delay, or discourage treatment and access to care. For the HMO-paid clinician, the patient may be perceived as a threat to profits. Thus capitation creates incentives that can transform clinicians from the patient's advocate to the patient's adversary. Conversely, the federal Patient Protection and Affordable Care Act compensates based on the quality of care rather than the quantity.[244]

Although clinicians seem like double agents serving two masters (i.e., managed care carriers and patients), the law is clear that the clinician must always act in the patient's best interest, because the clinician owes a fiduciary obligation to the patient. Should the carrier deny requested care, the clinician is legally obligated to appeal the decision to protect the patient's dental health.[332] A patient's best interest is always paramount, both legally and ethically, to a clinician's financial interest.[122,333]

Although profitable to insurance carriers, the success of denying or limiting benefits has created an era of rightfully indignant patients and frustrated clinicians. Consequently, the clinician must guard against patient disappointment with limited insurance benefits by explaining that although insurance carriers determine coverage under their patients' insurance policy, carriers do not set the standard of care. Regardless of how much or how little is covered by the patient's insurance carrier, prudent and reasonably careful clinicians set the standard of care. The legal doctrine of informed consent mandates the patient be informed of alternative therapy choices that the

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patient may pay privately even though the HMO denies approval for alternative therapies. Follow the maxim: Do not x-ray a patient's pocketbook. Reasonable informed consent is premised on patient choice and not cost alone.

Employment law

An employer may fire an employee for a bad reason or no reason at all, as long as termination was not done for discriminatory reasons.[236,326]

Dental negligence defined

Dental negligence is defined as a violation of the standard of care (i.e., an act or omission that a reasonably careful or prudent clinician under similar circumstances would not have done).[172] *Negligence* is equated with carelessness or inattentiveness.[179] *Malpractice* is a lay term for such professional negligence. Dental negligence occurs for two reasons:

1. A clinician does not possess a reasonable degree of education and training to act prudently.
2. Despite reasonable schooling, additional training, and continuing education, the clinician acts with unreasonable carelessness or imprudently fails to act as a reasonably careful clinician would act.

One simple test to determine if an adverse outcome results from negligence is to ask the following question: Was the treatment result reasonably avoidable? If the answer is yes, then it is probably malpractice. If the answer is no, then it is instead an unfortunate maloccurrence that resulted despite reasonable care attempts.

Locality rule

The locality rule, which provides for a different standard of care in different communities, is rapidly becoming outdated. Originating in the 19th century, the rule was designed to acknowledge differences in facilities, training, and equipment between rural and urban communities.[198,347]

The trend across the United States is to move from a locally based standard

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to a statewide standard, at least for generalists. For endodontists, a national standard of care is applied, as the AAE Board is national in scope.[290] Because of nationally published endodontic literature, advances in Internet communication, continuing education courses, and reasonably available transportation for patients, no disparity generally exists between rural and urban endodontic standards. A current exception may be the limited geographic availability of specific CBCT technology such as the Accuitomo or Kodak 9000. Their higher resolution provides superior radiographic endodontic diagnosis compared with 2D imaging and even other CBCT devices. As CBCT machine pricing is lowered and availability increases, the locality rule may be broadened beyond a local community for CBCT technology. On the other hand, time is critical during the first 48 hours of symptomatic numbness if an endodontic overfill enters the IANC.[254] In such instances, a referral to a distant CBCT facility may be required to assess the need for immediate microsurgical removal or instead obtain, at the very least, a medical CT in the local geographic area if a CBCT facility is unavailable.[75,97,157,169,201,204]

A clinician should provide reasonably careful endodontic care to a patient regardless of treatment locality. Rather than focusing on different standards for different communities, more important considerations include knowledge of endodontic advances in the field gained with continuing education to utilize improved diagnostics, instrumentation, and therapeutic interventions.

The locality rule has two major drawbacks. In areas with small populations, clinicians may be reluctant to testify as expert witnesses against other local clinicians. Also, the locality rule allows a small group of clinicians in an area to establish a local standard of care inferior to what the law requires of larger urban areas. Peer-reviewed publications are available to all clinicians in print and online. Clinicians can easily travel great distances to attend continuing education courses or attend webcasts in their own office. Blaming technologic ignorance on a clinician's rural location is inexcusable with modern media, computer technology, continuing education courses, and travel ease.

Continuing education

Continuing education should not add new skills at the expense of

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compromising or eliminating core clinical values. A prudent practitioner should be cautious adding a new methodology that is counterintuitive to basic biomechanical principles.[224] As a general maxim, don't be the first or the last to adopt new technology.

A clinician is legally obligated to maintain current knowledge in the field of endodontics. If not, the clinician may have only 1 year of knowledge (repeated 30 times) during the span of a 30-year career. By not maintaining continuing education knowledge and updating clinical skills, a clinician may unreasonably condemn otherwise salvageable teeth because of inadequate diagnosis or treatment.

State dental board relicensing continuing education requirements range from 0 in Colorado to 100 continuing education units in South Dakota.

Evidence-based endodontics

Although much emphasis is currently placed on evidence-based medicine and dentistry, most clinical practices are not based on data derived from randomized clinical trials.[240] It is virtually impossible to conduct a trial to test the validity of every possible patient-management option. Many therapeutic choices are so compelling that a test would be unethical; others are so trivial that a test might not be worth the time or effort. Clinicians do not always practice in conformity to evidence-based research, even when evidence is available from randomized clinical trials. This is because knowing the right answer is only the first step in the process of adopting a new treatment methodology. Many clinicians will not use a new drug simply because supporting research data suggest its efficacy. Instead, identification of the probable mechanism of action is often a prerequisite before adopting a new treatment. Moreover, the test of time may eventually reveal an adverse effect of a particular drug or device (e.g., defects that were not identified before marketing). Even if the new product's research proves beneficial, comparative effectiveness studies with other time-proven and tested products are needed to determine a new product's relative worthiness.[137,196,253,295]

Clinicians should not avoid using certain drugs if the data supporting their use are overwhelming. When the results of a clinical trial conflict with a widely held mechanistic model, many clinicians doubt research-based evidence.[226] However, when the results from well-researched clinical trials

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become so convincing that the evidence can no longer be ignored, a prudent clinician embraces the new paradigm and discards previously held concepts. Endodontic science advances as disproved older concepts retreat. For example, delaying extractions until after several days of antibiotic use to reduce infection is no longer the accepted practice. Rather, immediate extraction, along with any necessary antibiotics, is the preferred current therapy.[6] Another example is one-visit endodontics, which has been proved to be as effective as two-visit endodontics.[109,225,250]

Holistic dentistry

Evidence-based dentistry is reached when there is a consensus of the majority of practitioners relying upon peer-reviewed research published in respectable dental journals with adequate statistical power that has been replicated. In contrast, pseudoscientific research and/or publications are typically espoused by a very small minority of dentists belonging to international dental or medical organizations that number less than 1000 practitioners. By contrast, the ADA membership is 170,000.

Examples of holistic teachings and preaching to patients include that root canals are toxic foci of infections, which cause systemic maladies. Thus perfectly healthy root-canaled teeth are unnecessarily extracted and the underlying alveolar bone excessively removed by cavitation therapy. More than 70 years ago, the ADA published refutation of such holistic hokum in its *Journal of the American Dental Association*.[b] AAE offers scientific refutation of holistic therapies for extracting healthy root-canaled teeth.[c] Moreover, state dental boards have disciplined dentists for promoting in advertisements and/or performing unnecessary holistic therapies.[d]

A dentist is not necessarily negligent just because he or she chooses one dentally accepted method of treatment or diagnosis and it turns out that another dentally accepted method would have been a better choice.[e] This defense jury instruction for acceptable alternative treatment method does not excuse extraction of healthy root-canaled teeth. Such treatment is an **unacceptable,** negligent choice of treatment. Nor is it acceptable within the standard of care to ask a patient to consent to negligent care.

Ozone therapy

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Ozone's antibacterial efficacy is not comparable with that of NaOCl.[f] Treatment of apical periodontitis involves elimination of root canal infection by a combination of mechanical and chemical means. Mechanical instrumentation alone does not completely eliminate bacteria from the root canal system because not all of the root canal surfaces are planed by the action of files. Consequently, antibacterial irrigants are employed to penetrate the noninstrumented surfaces. Sodium hypochlorite (NaOCl) is the current irrigant of choice due to its antibacterial and tissue-dissolving effects.

Food and Drug Administration Department of Health and Human Services subchapter H—Medical Devices, Section 801.415 Maximum acceptable level of ozone states:

> *(a) Ozone is a toxic gas with no known useful medical application in specific, adjunctive, or preventive therapy. In order for ozone to be effective as a germicide, it must be present in a concentration far greater than that which can be safely tolerated by man and animals.*
>
> *(b) Although undesirable physiological effects on the central nervous system, heart, and vision have been reported, the predominant physiological effect of ozone is primary irritation of the mucous membranes. Inhalation of ozone can cause sufficient irritation to the lungs to result in pulmonary edema. The onset of pulmonary edema is usually delayed for some hours after exposure; thus, symptomatic response is not a reliable warning of exposure to toxic concentrations of ozone. Since olfactory fatigue develops readily, the odor of ozone is not a reliable index of atmospheric ozone concentration.*

New products

New devices that lack definitive research studies should be used cautiously. For instance, high-intensity, wireless, fast-curing lights may generate heat at the wand tip and cause pulpal pathosis. First-generation halogen bulbs require longer composite curing time to achieve polymerization but generate only 400 to 800 mW/cm. Plasma arc bulbs reduce curing time but generate 2000

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mW/cm.

Hydron was marketed without long-term clinical testing. Its purported biocompatibility and reduced inflammation portended an improved endodontic drug for obturation. Postmarketing failures resulted from inadequate premarket testing that lacked long-term research. Thus Hydron's inability to obturate canals with a durable filling material resulted in both clinicians and patients being harmed as postmarket guinea pigs. Hydron's in vitro research did not match in vivo patients' experiences.

Other examples include Endocal 10 (formerly Biocalex 6/9), which proved ineffective. Arsenic to promote devitalization of the pulp resulted in local osteonecrosis. Thermafil metal carriers proved difficult to remove and were replaced with plastic carriers. Advance cement was touted as an improved luting cement for retaining permanent restorations. The high incidence of marginal leakage with Advance resulted in its withdrawal from the market in 2000. A multimillion-dollar class action settlement resulted against the manufacturer on behalf of California clinicians with unsealed margins of crowns cemented with the Advance luting agent.[128]

Older devices, over time and with sufficient patient experience, may manifest adverse events. Overheating with ultrasonic tips for post removal has resulted in teeth and tissue loss (Fig. 27.21). Accordingly, despite insufficient label warnings on some ultrasonic devices, copious water coolant, rest periods, and avoidance of prolonged ultrasonic device use are needed for safe post removal.[90,95,130]

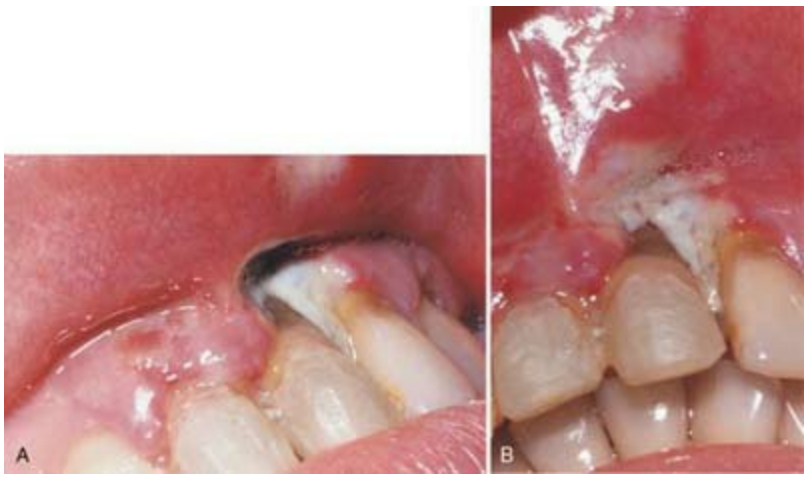

FIG. 27.21 Results of overheating a post with an ultrasonic device

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during post removal.

A) Close-up shows top view of yellow stained teeth with whitish tissue on the black gum line.

B) Close-up view shows pale teeth with greater loss of tissue compared to A at gum line. The black line is diminished.

Today's clinician who is exploring ways to improve the quality and success of endodontic therapy is constantly presented with new dental products and techniques. For prescribing and using drugs or other agents, the ADA's Principles of Ethics, Section 5D, provide this guideline[18]:

> *Except for formal investigative studies, dentists shall be obliged to prescribe, dispense, or promote only those devices, drugs and other agents whose complete formulae are available to the dental profession. Dentists shall have the further obligation of not holding out as exclusive any device, agent, method or technique if that representation would be false or misleading in any material respect.*

Ethical clinicians should not indiscriminately adopt every new product. Instead, the supporting research should be reviewed, rather than risk the patient's welfare with inadequately tested products or researched products only tested in vitro. Again, do not be the first or the last to adopt new technology.

U.S. food and drug administration approval

Despite FDA approval, some drugs are marketed with fraudulent concealment of the number and nature of adverse event incidents.[117a]

U.S. food and drug administration's drug approval

Until 1962, drug companies were allowed to promote their products for any use so long as they were shown to be "safe" for one use. Manufacturers were marketing drugs with serious side effects for minor conditions and to vulnerable populations, resulting in many injuries and deaths. Ineffective drugs were the rule rather than the exception. When a retrospective review of

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all drugs was conducted after 1962, the National Academy of Sciences found that fully 80% of the uses for which drugs were being promoted could not be shown to be effective. Since 1962, the FDA has required prescription drug manufacturers to demonstrate safety and efficacy with animal and randomly controlled research trials conducted with FDA-approved protocols. FDA "approval" to market new drugs for specific uses is termed *premarket approval* (PMA).

In a new drug application (NDA) to the FDA, findings from many clinical studies are used to assess a new drug's efficacy and safety and to gain the FDA's PMA to market the drug. An NDA judges whether the proposed new drug (1) causes more good than harm (benefit outweighs the risk), (2) is comparatively more effective than already existing marketed drugs, or (3) has unreasonably frequent or severe side effects (risk outweighs the benefit).[35,321]

The Pharmaceutical Research and Manufacturers Association of America has created an online database that summarizes clinical study results involving hundreds of prescription medicines. Notwithstanding, the sheer volume of clinical testing has overwhelmed the FDA's ability to independently assess commercial trials and make all test results public. Deceptive marketing, unreported side effects, and hidden payments to medical/dental researchers highlight the gap between the number of clinical trials conducted and the number published.[27,181,196,216,256,269,274,284,295,316,337]

U.S. food and drug administration clearance

The applicant must receive FDA approval of its PMA application prior to marketing the device as FDA approved. PMA approval is based on an FDA determination that the PMA contains sufficient valid scientific evidence to assure that the device is safe and effective for its intended uses.

The FDA lists certain products that are exempt from premarket notification requirements under the Food and Drug Administration Modernization Act of 1997 (the Modernization Act),[116] which include the following endodontic products:

- Root canal posts
- Gutta-percha

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Adverse drug events

Adverse drug events (ADEs) include "injury due to medications."[39] In 1999, the Institute of Medicine identified medication errors as a significant and preventable source of ADEs. Warning labels represent a quick reminder to highlight the most important instructions for the safe and effective use of prescribed medications. In one study, 50% of participants looked directly at the warning labels; 22% did not look at any. The recommendation was to move all of the warnings from the colored stickers to the main, white label.[304] More than 30% of those 65 and older take 10 medications daily.[304]

Primary medication nonadherence: Unfilled prescriptions

Primary medication nonadherence occurs when a noncompliant patient fails to fill a prescription. Up to 25% of children discharged from a hospital with a prescription do not have those prescriptions filled.[348] Other data from emergency departments suggest that the frequency of unfilled prescriptions is between 7% and 35%.[348] Electronic prescriptions are more likely to be filled. When a prescription is electronically prescribed, the patient does not have an opportunity to lose or misplace it. Data suggest that clinicians should explore whether a patient actually filled the medication prescribed or was taking it properly when the patient does not appear to be responding adequately to prescribed treatment.[199]

U.S. food and drug administration medwatch

Newly marketed drugs may not list all potential adverse drug reactions or events because of the limited number of patients in research trials before marketing. Therefore the FDA's MedWatch program encourages clinicians to report known or suspected drug reactions. MedWatch encourages reporting of serious unexpected adverse drug reactions whose nature and severity are inconsistent with or absent from the drug's labeled warnings.[111–113,115,235] Clinician reporting to the FDA is confidential and voluntary. The patient's identity need not be disclosed. Clinicians should contact the FDA by telephone at (800) FDA-1088 or fax at (800) FDA-0178 to obtain the FDA Medical Products Reporting Program (MedWatch) form (FDA form #3500). The back portion of the *Physicians' Desk Reference* (PDR) contains a

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MedWatch form. Clinicians can also download the MedWatch reporting form at www.fda.gov/medwatch/getforms.htm; request the form by mail via MedWatch, 5600 Fishers Lane, Rockville, Maryland 20852-9787; and report suspected drug interactions, reactions, or adverse product events at www.fda.gov/medwatch/report.htm. Other electronic drug databases are available to report and access important drug interaction information.[340] For FDA-approved safety-related drug labeling online, see www.fda.gov/MedWatch.

Adverse drug reactions are injuries occurring when drugs are administered at usual doses. These reactions represent the primary focus of regulatory agencies and postmarketing surveillance. An adverse drug event suggests medication prescribing or dispensing errors that compromise patient safety. Unfortunately, clinicians report only between 1% and 5% of adverse drug incidents to the FDA, because clinician reporting is voluntary.[184] On the other hand, drug manufacturers must report known adverse drug incidents to the FDA.[113]

Clinicians should document adverse drug reactions not only in the progress notes but also in the allergy section of the chart in order to assess whether future use of the same drug is contraindicated and to prevent a harmful recurrence. Predictable adverse drug reactions that manifest should also be recorded as consideration for future disuse of the involved particular drug.

The Weber effect is a known phenomenon in the epidemiology literature in which the number of spontaneously reported adverse events for a drug tends to peak around the second year after a drug's initial marketing.[80] The influence of the Weber effect suggests that an increase in adverse events may be attributed to epidemiologic reporting bias, which may not reflect a true difference in adverse events.[80]

Compounding pharmacies

More than 500 cases of fungal meningitis, including 44 fatalities, were attributed to injectable steroid solutions prepared by the New England Compounding Center (NECC), which subsequently filed for bankruptcy.[297]

Contamination is only one of five categories of risk associated with compounding pharmacies. The other four are subpotency, superpotency, overmedication, and medication replacement.[47]

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Many patients received sterile injections for back and joint pain for procedures that lack high-quality evidence-based efficacy.[299] FDA-approved methylprednisolone acetate is sold by Pfizer and two generics companies. NECC's version did not contain preservatives, which avoided the FDA's regulatory process.

Compounding involves the preparation of medications on prescription to meet unique patient health care needs that cannot be met with commercially manufactured and marketed drug products. Compounding includes providing different strengths of a drug with different nonactive excipients for allergy patients or creating dosage forms that are more palatable for a patient. Compounding pharmacies and pharmacists work directly with the prescribing dentist to create customized personalized medication solutions for patients in doses and formulations that the pharmaceutical industry does not manufacture.

The Food and Drug Administration Modernization Act (FDAMA), enacted November 21, 1997, amended the Federal Food, Drug, and Cosmetic Act relating to the regulation of food, drugs, devices, and biologic products. FDAMA exempted individualized compounded drug products from the rigid federal current goods manufacturing practices. Under federal law, drugs that are not manufactured in conformity with federal current goods manufacturing practices are deemed to be "adulterated" under 21 U.S.C. 351(a)(2)(B) and subject to federal sanction. FDAMA, 21 U.S.C. 351(a)(2)(B) does not apply to a drug product compounded by a licensed pharmacist in a state-licensed pharmacy for an identified individual patient with a valid prescription order —or in limited quantities before receipt of the valid prescription order.[299]

Federal law prohibits compounding of drugs previously removed from the market because such drug products, or one or more of their components, have been found to be unsafe or ineffective. Compounding drugs that are copies of a commercially available drug product are also prohibited.

After the 1937 sulfanilamide disaster that killed more than 100 people, Congress passed the Food, Drug, and Cosmetic Act (FDCA), requiring drugs to be safe and properly labeled. In 1962, a requirement was introduced for proof of drug efficacy through "adequate and well-controlled investigations," in response to thalidomide's fatal side effects causing impaired limb development.

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Traditional compounding pharmacies are not registered with the FDA as drug manufacturers. The FDA does not approve compounding pharmacy prescriptions before marketing. Nor does the FDA require compounding pharmacies to report adverse events. Instead, state law generally controls recordkeeping, certifications, and licensing for compounding pharmacies.

Dietary supplements

The Dietary Supplements Health and Education (DSHE) Act allows dietary supplements to be sold directly to consumers without any oversight or FDA regulation. In response to intense lobbying by the multibillion-dollar dietary supplement industry, Congress in 1994 exempted these products from FDA regulation.[190,205,342] Products may contain amounts claimed, but they need not, and nothing can prevent their sale if they do not. For example, an analysis of ginseng[205] products showed no ginseng at all in some products. The DSHE Act places the burden of proof for dietary supplements' safety on the FDA and not the manufacturer. As a result, consumers of herbal supplements must depend on self-regulation within the industry for assurance of product quality, consistency, potency, and purity.[164,342] An understaffed and overworked FDA can hardly be expected to be a vigorous enforcer.

An herbal product label can state the way the product is intended to affect "the structure or function" of the body but cannot claim its use for a specific disease. Manufacturers thus use creative language that complies technically with the statute but generally is confusing or deceptive.[205]

A current medication history form should include over-the-counter, herbal, and illicit drugs, many of which have the potential for synergistic or antagonistic interaction with clinician-prescribed drugs.[342]

Negligence per se

Violation of a health safety statute constitutes negligence per se if the patient was in the class of persons the statute was designed to protect.[292] Compliance with a health safety statute does not conclusively establish due care, because regulations require only minimal care and not (necessarily) prudent care or what the law regards as due care.[79,308]

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Prescription drugs

Clinicians should exercise extreme caution when administering or prescribing dangerous drugs. For sedative or narcotic drugs, cautionary directions should be written on prescriptions, and the pharmacist should place these directions on the prescription container as a patient reminder. For example, for the appropriate drugs, the clinician should prepare a prescription rubber stamp or obtain preprinted prescriptions that state the following:

> *Do not drive or operate dangerous machinery after taking medication because drowsiness is likely to occur. Alcohol, sedative, or tranquilizing drugs will cause drowsiness if taken in combination with this prescribed drug.*

The ADA and American Medical Association (AMA) provide prescription drug warning pads. Clinicians should document each drug information form provided with the prescription in the patient's chart.

The overuse of antibiotics risks resistant-strain development and side effects. Studies demonstrate generally no increased therapeutic efficiency of endodontic therapy with antibiotics when performed in the absence of facial swelling.[327] Unless persistent infections occur or compelling systemic reasons exist (e.g., uncontrolled diabetes, antibiotic prophylaxis necessary because of mitral valve regurgitation), antibiotics should not be prescribed prophylactically.[163,227] Neither pain nor localized swelling alone justifies antibiotics. However, extraoral swelling, cellulitis, or lymphadenopathy may require surgical drainage, antibiotics, or both. The U.S. Centers for Disease Control and Prevention (CDC) estimates about one third of all antibiotic outpatient prescriptions are unnecessary.[12]

Ability to foresee unreasonable risk

Each endodontic procedure has a variable degree of inherent risk. The standard of care requires that the clinician avoid unreasonable risks that may harm the patient (Fig. 27.22). Treatment is deemed negligent when a reasonably careful clinician would have foreseen some unreasonable risk of harm to the patient. Failure to follow the dictates of sound endodontic

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practice increases the risk of negligently induced deleterious results. Accordingly, prophylactic endodontic practice is designed to prevent foreseeable or reasonably avoidable injury risks. Foreseeability connotes predictability.

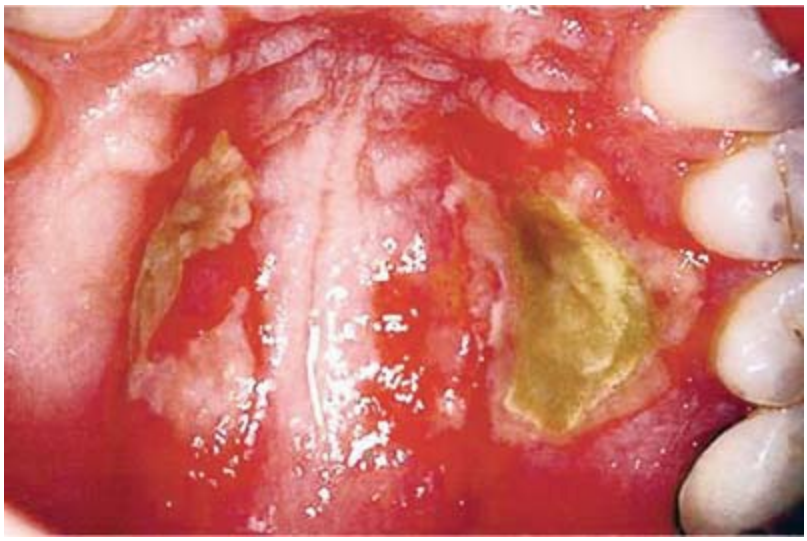

FIG. 27.22 Excessive local anesthetic resulting in palatal tissue sloughing with anterior middle superior alveolar (AMSA) (wand) injection.

Close-up view of interior of teeth shows greenish patches on palatal region little above the gums.

It is not necessary that the exact injuries that occur be foreseeable. Nor is it necessary to foresee the precise manner or circumstances under which injuries are inflicted. It is enough that a reasonably prudent clinician would foresee that injuries of the same general type would likely result in the absence of adequate safeguards.[265]

Consent

Simple consent occurs when the clinician makes the treatment decision unilaterally and obtains the patient's consent without offering the patient a choice. Conversely, informed consent is a joint decisional process whether to proceed with treatment or not after the dentist informs the patient of reasonable alternatives, benefits, and potential complications. Unconsented treatment constitutes a battery in legal parlance.

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Emergency endodontic treatment often requires that the patient be provided pain relief. Acute pain clouds the patient's decisional process; palliative treatment provides immediate pain relief so that on a return visit, the patient can rationally decide among treatment choices. With ample time, the patient can rationally decide and compare choices such as completion of endodontics and a crown versus extraction followed by an implant with crown. Also, an extraction that may result in progressive alveolar ridge bone loss and the need for bone grafting are considerations the clinician should explain to the patient.

Patient choices may be influenced by cost, convenience, time off work, travel needs, esthetics, or a myriad of other reasons. The clinician plants the seeds of the decisional tree, but the patient decides which branch of the tree should be followed.

Informed consent principles

The legal doctrine of informed consent requires that the patient be advised of reasonably foreseeable material risks of endodontic therapy, the nature of the treatment, reasonable alternatives, and the consequences of nontreatment.[71,173,221] This doctrine is based on the legal principle that, as individuals, patients have the right to do with their own bodies as they see fit, including the right to prematurely lose teeth regardless of recommended dental treatment. Once the clinician has informed the patient of the diagnosis, treatment risks, prognosis, and nontreatment risks, as well as recommendations for corrective treatment or alternative therapy, the patient has the right to decide how, when, or if to proceed. An adult of sound mind is entitled to elect to do nothing about existing endodontic disease. Rather than elect corrective treatment, the patient may elect to suffer nontreatment consequences, including present or future tooth loss or apical abscess.

To be legally effective, a patient's consent to treatment involving potentially serious injury complications must include informed consent. Accordingly, a clinician has a fiduciary duty to disclose all material or essential information necessary for the patient to make a decision.[71,173,221] The scope of a clinician's duty to disclose information is measured by the amount of knowledge a reasonable patient requires to make an informed choice. Material information is the disclosure a clinician knows (or should

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know) would be regarded as significant by a reasonable person in the patient's position who must decide whether to accept or reject a recommended endodontic procedure.

If a clinician fails to reasonably disclose information that would make a reasonable person in the patient's position decline the procedure, the clinician may be liable should an undisclosed risk manifest. Beyond the foregoing minimal disclosure, a clinician must also reveal such additional information as a skilled practitioner of good standing would provide under similar circumstances.

Informed consent

Adequate informed consent requires that a patient be informed of the ABC's —the alternatives, benefits, and complications—of a proposed treatment, along with the pros and cons of the alternative therapies or options.[71,173,221] A patient has a right to be informed of these ABC's from the treating clinician and not solely from an informed consent form or by a front office staff person who lacks a dental license. Informed consent must be presented in layperson's language, so that the patient can appreciate and understand what the treatment risks are.

A patient cannot legally consent to negligent care.[315] Accordingly, informed consent applies only to treatment that complies within the standard of care. If the clinician is reasonably careful during treatment but an untoward result occurs, the clinician is not liable so long as the patient was forewarned of the potentially unavoidable risk prior to treatment.

A reasonably unavoidable risk that manifests during treatment represents a maloccurrence and not malpractice. For example, a small percentage of endodontic failures occur for nonnegligent reasons. The clinician should be sensitive to the patient's feelings of disappointment. When endodontics fails, regardless of cause, it is a 100% failure rate for the patient. On the other hand, if the risk was reasonably avoidable, then informed consent does not apply to reasonably avoidable risks.

Patient treatment refusals should be documented. Some states require informed refusal disclosures advising the patient about the consequences of refusing treatment or diagnostic modalities.

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Informed consent application

Informed consent is a flexible standard that considers reasonably foreseeable consequences, depending on the clinical situation present both before and during treatment. For instance, a fractured or separated endodontic instrument left in the root canal creates the possibility of root canal failure or impaired success (depending on whether the fracture occurred in the coronal, middle [least favorable], or apical third of the root canal). The clinician must advise the patient of the relative risk of future failure and suggest treatment alternatives to correct the problem. An adequately informed patient can better make an intelligent choice among apicoectomy, referral to an endodontist utilizing microscopy for attempted retrieval, or periodic radiographic observation at recall visits.[76,183,197] In Fig. 27.23, the clinician overinstrumented and perforated the inferior alveolar nerve (IAN), causing both a permanent dysesthesia and paresthesia. The clinician should have immediately referred the patient to an endodontist for attempted gutta-percha retrieval before the endodontic sealant containing eugenol set, which caused more deleterious chemical injury to the IAN. If retrieval was unsuccessful, the patient should have then been referred to a microsurgeon within 48 hours from occurrence for optimal opportunity to prevent chemical cytotoxic injury to the IAN.[262]

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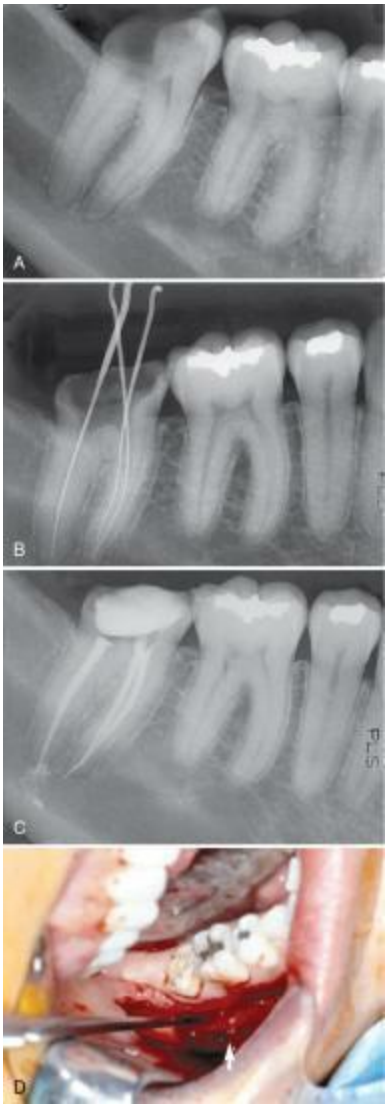

FIG. 27.23 **A,** Preoperative radiograph. **B,** Measuring film without a rubber dam. **C,** Postoperative radiograph. **D,** Decompression microsurgery showing protruding “dagger” of bone to right of retractor at inferior border of inferior alveolar nerve, resulting from prior perforation of the mandibular canal. This is probably due to file overinstrumentation.

A) Radiograph shows right tooth with radiolucent patch on the crown and unfilled root canals.

B) Radiograph shows three canals in front of the right tooth.

C) Radiograph shows right tooth with filled root canals and crown.

D) Close-up view shows blood oozing out below the teeth.

Source: (Courtesy Anthony Pogrel, DDS, MD.)

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Adequate disclosure includes clinical judgment and experience, which assesses current research and applies it to the clinical needs of each patient. Today's advance may be tomorrow's retreat if materials, devices, or instruments lacking adequate long-term study of safety and efficacy predictably fail. For instance, the one-component bonding agent is more technique sensitive than its two-component predecessor. This is because the one-component agent contains acetone that desiccates the tooth surface and requires a moist surface to avoid postoperative sensitivity. Long-term testing before marketing would have revealed that the two-step system is more reliable and forgiving.[312] Reasonable clinicians do not sacrifice patient safety for speed and profit and then compensate with desensitizing agents. Clinicians should be sensitive to limited research on new products or devices lest the patient be the injured guinea pig patient with products that were inadequately tested before marketing.

Material disclosure concerns whether the patient was provided sufficient information for a reasonable patient to achieve a general understanding of the proposed treatment or procedure. This disclosure includes information concerning any dentally acceptable alternatives, any predictable risks of serious injury, and any likely consequences should the patient refuse the proposed therapy. The standard to be applied is whether a reasonable person in the patient's position would have consented to the procedure or treatment in question if adequately informed of all significant perils.[66] Informed consent applies only to inherent risks of nonnegligent treatment, because a patient's consent to negligent treatment is voidable as being contrary to public policy.[315] Accordingly, a patient who refuses necessary diagnostic radiographs should be refused treatment.

If warnings are limited to some risks but not others, some courts have held that the patient can recover for all surgical injuries including warned risks.[160]

Endodontic informed consent

If the practitioner's own statistical experience varies significantly from national statistics, using statistics presented in national literature regarding success rates for endodontic procedures is considered insufficient disclosure and does not fulfill the legal requirements of informed consent (Fig. 27.24).[145,291]

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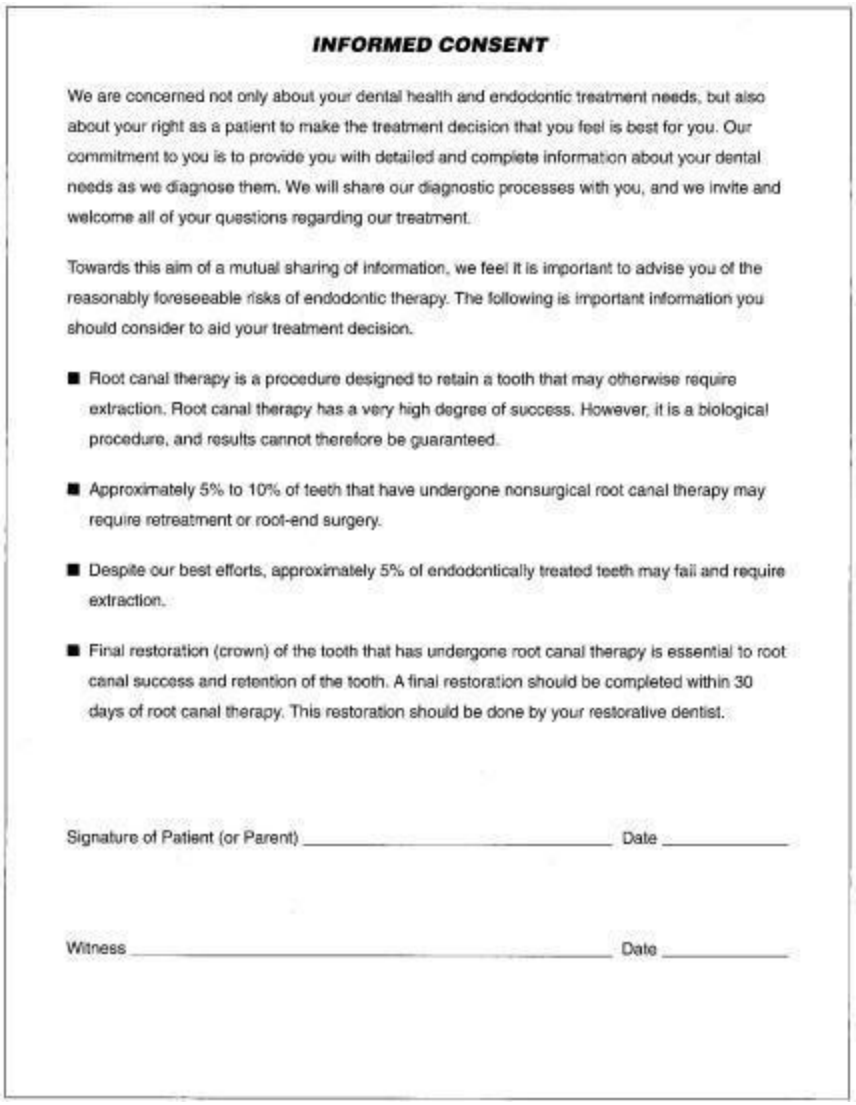

INFORMED CONSENT

We are concerned not only about your dental health and endodontic treatment needs, but also about your right as a patient to make the treatment decision that you feel is best for you. Our commitment to you is to provide you with detailed and complete information about your dental needs as we diagnose them. We will share our diagnostic processes with you, and we invite and welcome all of your questions regarding our treatment.

Towards this aim of a mutual sharing of information, we feel it is important to advise you of the reasonably foreseeable risks of endodontic therapy. The following is important information you should consider to aid your treatment decision.

- Root canal therapy is a procedure designed to retain a tooth that may otherwise require extraction. Root canal therapy has a very high degree of success. However, it is a biological procedure, and results cannot therefore be guaranteed.
- Approximately 5% to 10% of teeth that have undergone nonsurgical root canal therapy may require retreatment or root-end surgery.
- Despite our best efforts, approximately 5% of endodontically treated teeth may fail and require extraction.
- Final restoration (crown) of the tooth that has undergone root canal therapy is essential to root canal success and retention of the tooth. A final restoration should be completed within 30 days of root canal therapy. This restoration should be done by your restorative dentist.

Signature of Patient (or Parent) _____ Date _____

Witness _____ Date _____

FIG. 27.24 Informed consent form.

Form of informed consent is as follows:

We are concerned not only about your dental health and endodontic treatment needs, but also about your right as a patient to make the treatment decision that you feel is best for you. Our commitment to you is to provide you with detailed and complete information about your dental needs as we diagnose them. We will share our diagnostic processes with you, and we invite and welcome all of your questions regarding our treatment.

Towards this aim of a mutual sharing of information, we feel it is important to advise you of the reasonably foreseeable risks of endodontic therapy. The following is important information you should consider to aid your treatment decision.

1) Root canal therapy is a procedure designed to retain a tooth that may

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otherwise require extraction. Root canal therapy has a very high degree of success. However, it is a biological procedure, and results cannot therefore be guaranteed.

2) Approximately 5 percent to 10 percent of teeth that have undergone nonsurgical root canal therapy may require retreatment or root-end surgery.

3) Despite our best efforts, approximately 5 percent of endodontically treated teeth may fail and require extraction.

4) Final restoration (crown) of the tooth that has undergone root canal therapy is essential to root canal success and retention of the tooth. A final restoration should be completed within 30 days of root canal therapy. This restoration should be done by your restorative dentist.

At the bottom, the blank spaces for signature of patient (or parent), date, witness, and date are given.

Among specialists, the reported incidence of treatment complications in endodontics is relatively low. Based on a Southwest Endodontic Society retrospective study, a reasonable endodontist or a practitioner with similar abilities should disclose the following facts to patients[288]:

1. Endodontic therapy cannot be guaranteed.
2. Although endodontic therapy is usually successful, a small percentage of teeth are lost due to complications or treatment failure, despite competent endodontic care.
3. Slight overextending or underfilling of root canals occurs in 2% to 4% of cases, which may contribute to treatment failure.
4. Slight to moderate transient postoperative pain may occur. Severe postoperative pain rarely occurs.
5. Irreparable damage to the existing crown or restoration secondary to endodontic treatment is uncommon after the access opening is restored.

Video-informed consent.

Animated video-informed consent shown to the patient is a dynamic method of providing informed consent. Because the video-informed consent is considered part of the clinician’s records, in the event the patient disputes having ever been advised of (1) the nature of endodontic disease, (2) the

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availability of endodontic specialists, or (3) the relative indications for nonsurgical versus surgical endodontic care, the videotape can be played back to the jury as proof that the patient was informed. It is doubtful that a jury would believe a forgetful patient who admits having previously viewed the videotape, because the videotape refreshes the stream of memories that may have otherwise faded into the unconscious.

A patient viewing the videotape is more likely to understand the informed consent disclosure of a technical procedure, particularly if the video contains illustrated animation. After viewing the videotape and discussing its contents with the clinician, the patient should sign a video consent form to verify that the video was viewed and all the patient's questions were answered.

Alternative technique choices

Currently taught methodologies include filling the root canal with the lateral compaction method or using vertical warm gutta-percha carrier delivery systems, such as heat-transfer units for warm gutta-percha.[197] Each technique has zealous advocates. Both methods are reasonable and acceptable choices, and each method conforms to the standard of care. The choice of techniques changes as new products are introduced and scientific research is conducted. The vertical warm gutta-percha technique promises to deliver improved flow to fill lateral canals and is likely to predominate in the 21st century as the preferred methodology. Nonetheless, lateral compaction has a long-term proven track record, particularly with less-experienced practitioners, and is less technique sensitive. Regenerative endodontics will have greater utilization as this scientific application increases.[16]

Different schools of thought.

After the clinician advises treatment, if other reasonable clinicians would disagree or if there are other respectable schools of thought on the correct treatment, this material information should be disclosed to the patient.[325,346] For example, there may be two schools of thought concerning the optimal treatment for retrofilling and apicoectomy. One school posits that a retrograde with mineral trioxide aggregate (MTA) or Biodentine is the appropriate procedure, whereas another school asserts that retrograde with Super EBA is a reasonable choice.[343] One can assume further that an

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explanation of these two different methods of treatment constitutes material information for the purposes of informed consent. The mere fact that there is a disagreement within the relevant endodontic community does not establish that the selection of one procedure as opposed to the other constitutes negligent endodontic therapy. Because competent endodontists regularly use both procedures, a patient would have a difficult time proving dental negligence (i.e., that the endodontist failed "to have the knowledge and skill ordinarily possessed, and to use the care and skill ordinarily used, by reputable specialists practicing in the same field and in the same or a similar locality and under similar circumstances"). Moreover, neither school of thought possesses long-term clinical research results to prognosticate apical sealant insolubility rates in an 18-year-old patient with an additional 60-year life expectancy.[293] Long-term durability depends on an insoluble seal to reduce the risk of bacterial leakage.[66,228] Fig. 27.25 represents a preoperative and postoperative radiograph of a successful apicoectomy and retrograde in a 14-year-old.

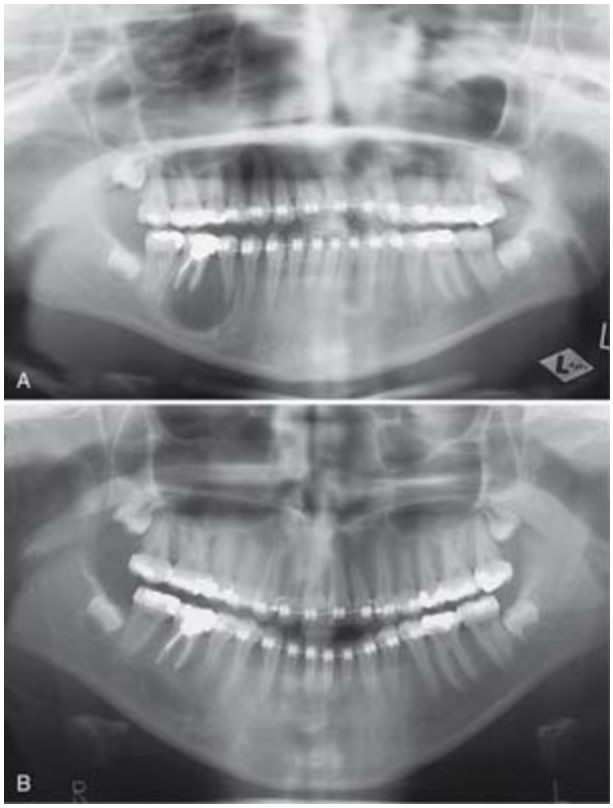

FIG. 27.25 **A,** Preoperative panoramic radiograph showing a

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successful apicoectomy and retrograde seal. **B,** Postoperative panoramic radiograph showing a successful apicoectomy and retrograde seal.

A) Radiograph shows an ovoid chamber below the right molar and premolar in lower jaw. The teeth have metal braces on them.

B) Radiograph shows high-density tissue in place of the ovoid chamber. The braces are slightly curved at the center of teeth.

Source: (Courtesy Edmond Bedrossian, DDS.)

On the other hand, the specialist would have a duty under the previous hypothetical circumstances to disclose the two recognized schools of treatment so that the patient could be sufficiently informed to make the final personal decision. An endodontist, being the expert, appreciates the risks inherent in the procedure prescribed, the risks of a decision not to undergo the treatment, and the probability of a successful outcome of the treatment. Once this information has been disclosed, this aspect of the endodontist's expert function has been performed. The weighing of these risks against the individual subjective fears and hopes of the patient is not an expert skill. This evaluation and decision constitute a nondental judgment reserved for the patient alone.[71,173] In this hypothetical situation, failure to disclose such material information would deprive the patient of the opportunity to weigh the risks versus the benefit. Consequently, the clinician would have failed in the duty of disclosure, which the doctrine of informed consent requires.

When improved technology offers clearly superior results, it no longer becomes a patient choice issue but rather a requisite requirement to fulfill the standard of care. Apical microsurgery with ultrasonic tips for retrofilling exemplifies improved technology and the current standard of practice. Likewise, apical retrogrades should be done with MTA and not with amalgam.[66] Finally, microscopic endodontics represents the current standard of care for apical surgery or fractured instrument retrieval.[76,86,166,183,197]

Avoiding patient claims.

If a clinician fails to obtain adequate informed consent, a plaintiff can recover damages (even in the absence of any negligent treatment) should the patient testify that performed treatment would have been refused if the clinician had

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provided information concerning possible risks. Therefore discussions of treatment risks with the patient should be documented. Informed consent forms are very helpful, although not legally mandated. A jury may believe that the patient was informed orally although the patient's attorney may argue that if it is not documented it did not happen. Equally, if not more important than consent forms, is a chart notation indicating that the clinician discussed informed-consent risks and alternatives (and that the patient understood and accepted this information).

Patients may mentally block out frightening information. Trauma and a potent anesthetic agent can create retrograde amnesia. Therefore clinicians must document (in the patient's chart) any risks, benefits, alternatives, and consequences of nontreatment provided to the alert patient before sedation.

Clinicians should follow only the patient-authorized and consented treatment plan. If an emergency precludes advising treatment risks to the minor patient, lack of informed consent may be defensible as implied consent (because no reasonable parent would refuse necessary emergency treatment).

The following example demonstrates how a clinician should record any recommended treatment the patient has refused and the reason for refusal:

> *Patient refused endodontic referral for consultation with endodontist (Dr. Goodguy) because husband was laid off work last month and cannot afford treatment. After Dr. G. provided explanation, patient states she understands detrimental delay risks but is willing to assume these risks.*

Patients may initial any referral refusal on the chart. Although patient signature is not mandatory, it will enhance the clinician's credibility should the patient later dispute a referral was made.

Reasonable familiarity with a new product or technique is required before it is used. In addition, a patient is entitled to know the clinician's personal experience with a particular modality because the patient has a right to choose between reasonable alternatives or to seek care from a clinician who has more extensive experience with a particular modality or new product. A clinician who fails to obtain informed consent may be liable for injury caused by a product or instrument. The fact that the clinician followed the

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manufacturer's instructions is no defense if the clinician did not provide adequate information concerning a product or instrument risk (to permit the patient to intelligently weigh the disclosed information and choose among treatment options).

Referrals to other specialists

Every clinician, including specialists, will at some time need to refer a patient to another specialist for treatment to comply with the standard of care required of a reasonably careful clinician.[123]

Generally, if the referral takes place within the same dental practice, the legal doctrine of respondeat superior ("let the master answer") may be applicable. Under this doctrine, a clinician is liable for the dental negligence of a person acting as his or her agent, employee, or partner.[264] Liability is determined by whether the principal clinician controls the agent clinician's activities or methodology, regardless of whether such control is actually exercised.

If the referral is made (even within the same physical environment) to an "independent contractor" endodontist who does not diagnose or treat under the direction or supervision of the referring clinician, and that referring clinician has no right of control as to the mode of performing the treatment, the principle of agency and responsibility for the acts of another does not apply.[223] The legal test for agency is the principal's right to control, regardless of whether the control is exercised over the agent. However, if the independent contractor or referring clinician in the same practice does not provide adequate notice of this independent relationship to the patient, the independent contractor will still be liable under the legal doctrine of ostensible agency. Thus despite an independent contractor agreement, a patient can legally infer the so-called independent clinician is instead an employee, because neither clinician in the group practice disclosed an independent contractor relationship to the patient.

To ensure that the referred clinician is not considered the agent of the referring clinician within the same facility, fees for the referred clinician should not be set by the referring clinician nor should the fees be divided equally or shared based on some other arrangement. Also, the referred clinician should bill separately and exercise independent diagnostic and

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therapeutic judgment. The patient should be advised that the referred clinician is independent of the referring clinician. Otherwise, the referred clinician or specialist may be regarded or inferred to be the ostensible agent of the referring clinician in the same office although not intended. The legal test for agency is how the facts or circumstances appear to a reasonable patient, regardless of the clinician's understanding or written contract intent that the other treating clinician is to be considered an independent contractor. Thus agency may be actual in fact or, alternatively, ostensible (i.e., implied by circumstantial conduct).[174]

Surgical versus nonsurgical endodontics

Litigation to determine whether nonsurgical or surgical endodontics was the proper treatment choice will not be decided by any one clinician, the ADA, the AAE, or the ablest of judges. Rather, after considering all the evidence and the opinions of experts, a jury of the patient's peers will decide the disputed matter. Depending on the individual case, the jury may decide that either a combination of nonsurgical and surgical endodontic therapy, rather than one method exclusively, should have been attempted. The jury may also decide that the patient should have been advised of the availability of such alternative therapy. Similarly, the jury may determine that apical surgery should have been done microscopically rather than macroscopically, depending on expert testimony regarding the relative advantages of each method or upon presenting clinical circumstances (e.g., suspected calcification, viewing a separated instrument).

Microscopic endodontics

Microscopic endodontics improves posture and reduces neck and back fatigue.[25] Identification of microfractures in teeth[76,183,197,230] and removal of diseased dental tissue are easier and more accurate when done microscopically.

> *The operating microscope is an indispensable tool for state-of-the-art endodontic treatment. The specialty practice should not be without a microscope; this instrument is useful in all phases of endodontic*

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treatment from diagnosis to placement of the final restoration.[166]

Microscope

A 2007 survey of 1091 endodontists indicated that 90% of endodontists have access to and use the OM in their practice, a dramatic increase from 52% in use in 1999.[15] The 2012 AAE Position Statement is that the microscope is an integral and important part of the performance of modern endodontic techniques.[15]

According to the AAE, microscope benefits are as follows:

- Locating hidden canals obstructed by calcifications or reduced in size
- Removing materials such as solid obturation materials (silver points and carrier-based materials), posts, or separated files
- Removing canal obstructions
- Assisting in access preparation to avoid unnecessary destruction of mineralized tissue
- Repairing perforations
- Locating cracks and fractures that not clinically visible or palpable with an endodontic explorer
- Facilitating all aspects of endodontic surgery, particularly in root-end resection and placement of retrofilling material
- Photographic documentation enhancement
- Ergonomic benefit[15]

Magnification.

The modern approach to periradicular surgery (PRS) is through magnification, illumination, and microsurgery. Prognosis is greatly improved when these techniques are employed. Although loupes are helpful, improved illumination and magnification of the surgical operating microscope are essential for PRS. Microscopy provides a more detailed examination of the root apex anatomic features such as isthmuses, accessory canals, fracture cracks, and crazing. Failure to shape, clean, and fill the root canal system is acknowledged as the main reason for failure of root canal treatment. The

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same principle applies to apical surgery. A disadvantage of MTA for retrograde sealant is handling and placement. MTA is hydrophilic and requires moisture to set with up to 3 hours of setting time. Newer agents to replace MTA include Biodentine, containing tricalcium silicate.

Root canal systems have much greater complexity at the level of root resection than previously recognized. At least 3 mm of the root apex should be removed to ensure successful retrograde obturation of all portals of exit. Angle of resection should be close to horizontal.

The floor of the sinus may be only 1 or 2 mm above root apices of the posterior dentition. Accordingly, the risk of sinus perforation increases when apical pathosis is present. Care must be taken to prevent debris from entering the sinus during maxillary posterior root canal therapy.

Separated instruments

The risk of broken files can be reduced if all hand and rotary nickel and titanium (NiTi) cleaning and shaping files are not resterilized and reused but are discarded after single-tooth use. Efficiency also increases, because approximately 50% of cutting efficiency is lost after initial use. Episodes of broken instruments will be dramatically reduced and nearly eliminated when files are used correctly and discarded after single-tooth use. Breakage increases sharply when hand or rotary shaping files are reused. Therefore the clinician should discard rotary instruments after a single-use visit. Chair and staff time efficiency, along with improved safety, dictates single-visit use of files.[228]

The clinician should save broken or defective instruments (e.g., a file that was broken inside a canal) (Fig. 27.26). The instrument manufacturer may be liable because the product was defective, rather than the clinician being liable for dental negligence.[266] Electron microscopy spectrographic analysis can determine if manufacturing defects with contaminants caused the break, rather than the clinician excessively stressing the instrument.

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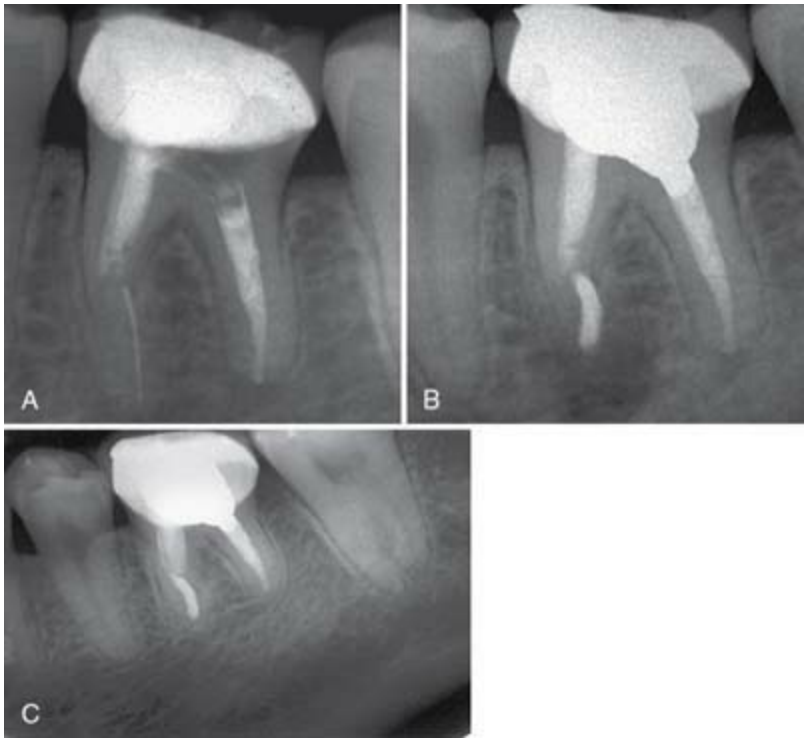

FIG. 27.26 **A,** Nickel-titanium file broken inside the mesiobuccal canal of the mandibular first molar. **B,** Due to patient discomfort, the segment was surgically removed and mineral trioxide aggregate (MTA) was used as root-filling material. **C,** Periapical radiograph 2½ years posttreatment shows complete healing.

Three radiographs are marked A through C.

A) Tooth has radiopaque crown. The filling in root canals is faded at the lower part of the first root canal and upper part of the second root canal.

B) Tooth has filled root canals but there is a radiolucent patch at the root apex of first canal.

C) Tooth has filled root canals and high-density tissue in place of the radiolucent patch.

Source: (From Torabinejad M, Walton RE: *Endodontics: principles and practice,* ed 4, St. Louis, 2009, Saunders/Elsevier.)

Equipment and supplies

Equipment should be kept in good repair by checking and following the manufacturer's recommended maintenance schedule. The clinician should carefully read the manufacturer's instruction warnings on instruments and

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inform staff of any important points. Infection control in operating dental equipment is mandatory, such as updating and maintaining dental units with check valves to prevent water retraction or suck-back.[70] The clinician should inspect check valves monthly and change clogged valves immediately.[287] Water retraction testers, at no charge to the clinician, are available from some manufacturers (A-dec [Newberg, OR], for instance).

The clinician or a staff member should also disassemble the unit's handpiece, run water through the line for a few seconds, and then stop. If a bubble of water is visible at the end of the water hose holes, the check valve is operating properly. If a bubble of water is not visible, water may be sucked back because of an absent or clogged check valve. An absent or clogged check valve is a source of cross-contamination, so it is important for the clinician or staff to perform weekly spore testing of the autoclave and monthly bacteriologic testing of waterlines.[17] The clinician or staff should consider using filters and should flush dental-unit waterlines between patients and also daily with FDA-approved chemical disinfectants to reduce water tubing biofilm. Some chemical disinfectants claim improved cost effectiveness by increasing handpiece and bur longevity concomitant with waterline purging or with continual use during treatment. Currently available waterline delivery systems, such as AquaSept, provide for safe water delivery or coolant to the handpiece or sonic scaler with autoclavable tubing.

Waterlines

Dental unit waterlines, typically, are constructed from a polymer or silicone rubber tubing, to provide water for irrigation, cooling, and flushing during dental procedures. Dental units are class I, FDA-regulated medical devices, which require FDA premarket clearance (510[k]).

Water stagnates in dental unit waterlines. Stagnant water contributes to biofilm buildup in the dental unit tubing, which can cause cross-contamination between patients. Dental unit waterlines connected to municipal water sources or closed-bottle systems typically cannot be sterilized. Without disinfection, waterborne microorganisms can collect in the dental unit waterline and form a biofilm. A layer of microorganisms adherent to the dental unit waterline tubing can also become biofilm, then dislodged and enabled to enter the output water stream to the handpiece and water

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syringe. Contaminated dental unit waterlines pose a risk of infection to the patient by direct exposure of waterborne pathogens and to dental professionals due to inhalation of aerosols.

The ADA[g] recommends routine monitoring of the water to demonstrate bacteria count of less than or equal to 500 colony forming units (CFU)/mL of heterotrophic bacteria. Sampling locations include the connection to the water source, dental handpiece connection, and air/water syringe. Organization for Safety Asepsis and Prevention (OSAP) recommends quarterly bacteriological test monitoring.

Flushing the waterline between patients as well as at the end of the day and before each day's first dental patient alone does not eliminate all biofilm. Therefore chemical disinfection of the waterlines is required to achieve water of potable drinking water quantity of no more than 500 CFU/mL. Germicidals that are automatically applied daily appear to be the most efficacious means of maintaining potable water quality. The CDC recommends that the dentist check with the dental unit manufacturer for recommendations for achieving and maintaining waterline quality to prevent contaminated dental water emanating from the dental drill and/or air/water syringe.[h]

Dental staff should not attach dental handpieces or dental instruments to dental unit waterlines that have not been cleaned or disinfected.

Dentists should establish written standard operating procedures to guide dental personnel in performing infection control procedures for dental unit waterlines.

To verify that the dental office complies with waterline quality maintenance, an infection control log should be kept.

Since 2003, the CDC in its Guidelines for Infection Control in Dental Health-Care Settings has recommended that the dentist test and monitor waterline quality with periodic bacteriological testing.[i]

Pulpotomy

California Dental Board requires that the dentist who performs procedures on exposed dental pulp, water, or other methods used for irrigation must be "sterile or contain recognized disinfecting or antibacterial properties."[j]

Examples of infection outbreaks from inadequately disinfected waterlines

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during pediatric pulpotomies include Georgia[k] in 2012 and Anaheim, California[l] in 2016. In each instance, dozens of children in the same dental clinic office suffered from mycobacterium abscessus infections. Consequently, dozens of young children who had not yet become immunocompetent lost significant portions of their maxilla or mandible and were hospitalized, administered IV PIC lines for 6 weeks, and suffered psychological damages including being fearful of future medical and dental care.

For surgical endodontic procedures, sterile water is required in accordance with CDC Guidelines for Infection Prevention.[m] Ultrasonic unit waterlines contained in the inner mechanisms of the device cannot be sterilized. Unless the device has autoclavable or disposable tubing rather than inaccessible inner waterline tubings, such devices can only deliver disinfected coolants and not sterile water. Consequently, such devices are contraindicated for surgical endodontic procedures. Thus during surgical procedures, use only sterile solutions as a coolant/irrigant using an appropriate delivery device, such as a sterile bulb syringe, sterile tubing that bypasses dental unit waterlines, or sterile single-use devices.

Fig. 27.27 shows a student who was burned by an inadequately maintained handpiece that overheated. This case settled for $280,000.

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FIG. 27.27 **A,** Before injury. **B–D,** A clarinetist music student was burned by an inadequately maintained handpiece that overheated. This lawsuit was settled for $280,000.

A) Front view of patient's face without any injury.

B) Patient with closed lips shows a wound on the lower lip that has yellowish tissue with blood clots inside.

C) Patient with open lips shows the increase in the wound size.

D) Patient's tongue has white tissue along with the wound on lower lip.

Clinician's liability for staff's acts or omissions

A clinician is liable for the acts or omissions of the clinician's staff under the doctrine of respondeat superior ("let the master answer"). This is termed *vicarious liability*, which means that the clinician is responsible not because

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of personal wrongdoing but because the clinician assumes legal responsibility for the conduct of employees and agents who act in the course and scope of their employment.

The clinician should instruct the staff in advising patients regarding posttreatment complaints. For example, if the staff ignores signs of infection, such as difficulty in swallowing or breathing or elevated temperature, and dismisses the patient's complaints as normal postoperative swelling, the clinician may be held liable for injury to the patient, such as delayed cellulitis, diagnosis or treatment of Ludwig angina, brain abscess, or other serious complications.

A clinician must be cautious when delegating responsibilities and give clear instructions to ensure that staff members properly represent the clinician and the chosen practice methods. Auxiliaries should not be allowed to practice beyond their competency level or license. For example, in states where assistant-placed restorations are legally permissible, the clinician should check any assistant-placed restoration before patient dismissal. Staff members should not make final diagnoses or handle patient clinical complaints without the clinician's involvement and consultation. Staff should be instructed to ask appropriate questions and relay the patient's answers to the clinician so the clinician can adequately determine what should be done.

Abandonment

Once endodontic treatment is initiated, the clinician is legally obligated to complete the treatment regardless of the patient's payment of any outstanding balance. This requirement is posited on the legal premise that any person who attempts to rescue another from harm must reasonably complete the rescue with beneficial intervention unless another rescuer (i.e., clinician) is willing to assume the undertaking.[69,195] Another view is that should a patient be placed in a position of danger unless further treatment is performed, the clinician must institute reasonable therapeutic measures to ensure that adverse consequences do not result.[212,296]

A clinician performing endodontic therapy should have reasonable means of communicating with patients after regular office hours to avoid a claim for abandonment. A recorded message is inadequate if the clinician fails to check for recorded messages frequently. Therefore answering services, pagers, or

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cell phones are required by the standard of care.

If the clinician providing endodontic therapy is away from the office for an extended period, a substitute on-call clinician should be available for any endodontic emergency and to answer patients' emergency calls. The endodontic treating clinician should arrange in advance for emergency service with a covering clinician. Leaving a name on the office answering machine or with the answering service without first determining the availability of the covering clinician is a mistake.

To avoid an abandonment claim, several prophylactic measures apply:

1. No legal duty requires a clinician to accept all patients for treatment. A private practice clinician may legally refuse to treat a new patient, despite severe pain or infection, except on the grounds of race or disability.[49,329] If treatment is limited to emergency measures only, the clinician must advise the patient that only temporary emergency endodontic therapy is being provided and that endodontic treatment is incomplete. The clinician should record this information in the patient's chart. For example:

Emergency palliative treatment only. Patient advised endodontic treatment of tooth #8 needs to be completed, either here or with another clinician. Explained complications likely if not soon completed, including infection recurrence, tooth loss, or both.

The patient should also be asked to acknowledge that treatment is limited to the existing emergency by endorsing an informed consent to emergency endodontics statement as follows:

I agree to emergency endodontic treatment of my tooth #8 and have been advised that (1) emergency treatment is for temporary relief of pain and (2) further root canal treatment of tooth #8 after emergency treatment is necessary to avoid further complications, including, but not limited to, pain, infection, swelling, tooth fracture, abscess, or tooth loss.

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2. No legal duty requires a clinician to continue treating former patients on recalls or subsequent emergency care once treatment is complete. Thus completion of endodontic treatment for tooth #19 does not legally obligate the clinician to initiate endodontic therapy for tooth #3 if endodontic disease of #3 began months after the clinician completed treatment of tooth #19.
3. Any patient may be discharged from a practice for any arbitrary reason, except on the grounds of race or disability, so long as all initiated treatment is completed. Accordingly, a former patient who evokes memories of a "frictional" relationship, who is financially irresponsible, or who arrives at the office after an absence of several years with an acute apical abscess in a site where previous care was not rendered may legally be refused treatment.
4. It is not considered abandonment if a patient is given reasonable notice to seek endodontic treatment with another clinician, and the patient is willing to seek endodontic services elsewhere.[229] Thus if rapport with the patient dissolves, the clinician should not hesitate to suggest that the patient would be better served if any remaining endodontic treatment were performed by a different clinician.

The clinician may discontinue treatment, provided it is not done at a time when the patient's dental health will be jeopardized (e.g., in the middle of treatment). To discontinue treatment, the clinician should do the following:

1. Notify the patient of the plan to discontinue treatment after a certain date.
2. Allow enough time for the patient to obtain care with another clinician, usually 30 days.
3. Offer to make emergency service available during the interim 30 days until a new clinician is obtained.
4. Provide diagnostic-quality records, copies of radiographs, and other pertinent clinical information in transfer records to the new treating clinician.
5. Allow the patient to select a new clinician or suggest referral by the local dental society if a referral service exists.
6. Document all of this in the patient's records, including a copy of a

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certified letter sent to the patient notifying discontinuance of further treatment.

Expert testimony

The standard of care that a clinician must possess and exercise is particularly within the knowledge of dental experts. However, there are occasional exceptions where the conduct involved is within the common knowledge of laypersons, in which case expert testimony is not required. In determining whether expert testimony is required to establish negligence, one California court commented: "The correct rule on the necessity of expert testimony has been summarized by Bob Dylan: 'You don't need a weatherman to know which way the wind blows.'"[170]

Incorrectly operating on the contralateral side because of mismounted radiographs or marking the wrong tooth on an endodontic referral card is an example of negligent conduct within the common knowledge of laypersons for which expert testimony is not required. The tort of battery results from unconsented treatment.

Increasingly, courts act as gatekeepers regarding the admissibility of scientific expert testimony. Anecdotal comparisons appear compelling, but such evidence may be judicially excluded in the courtroom. Some experts rely solely on their skill for experience-based observation rather than research testing of risk factors, such as that which occurs with epidemiologic research.

The U.S. Supreme Court (in the Daubert four-factors checklist[78]) held that in federal court an expert's testimony may be reliably admitted into evidence based on the following:

1. Whether the expert's technique or theory may be tested or refuted
2. Whether the technique or theory has been the subject of peer review or publication
3. Known or potential rate of technique error
4. Degree of acceptance of a theory or technique within the relevant scientific community

Courts determine whether testimony is based on the application of scientific principle or clinical experience. In general, courts are flexible and

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adaptable in determining whether a bright line separates scientific and unsupported nonscientific evidence. Often technical and specialized knowledge gained through continuing education and scientific journals, rather than an expert performing the research, is considered sufficient.

State courts are not bound to follow the Federal Rules of Evidence, but many do comply.[30] Nonetheless, U.S. Supreme Court decisions influence state courts that consider placing limitations on expert testimony. State court trial judges may either be more liberal or more restrictive in admitting expert witness testimony. If the trial court judge denies admission of scientific evidence as unreliable, untrustworthy, or irrelevant, the result may be to preclude an expert offering any opinion. On the other hand, the court may admit such evidence and instruct the jury members that they may consider the scientific basis for the expert opinions and give such evidence the evidentiary weight it deserves.

National Practitioner Data Bank

Insurance carriers are obligated to report all settlements to the National Practitioner Data Bank. These data are only accessible to hospitals for staff privilege credentialing and state licensing boards but not individual patients.

Medical negligence occurring in hospitals is too often underreported.[81] The Public Citizens' Health Research Group reports that more than half of U.S. hospitals have never reported an adverse incident to the National Practitioner Data Bank, an incredulous occurrence rate.[72]

Malpractice incidents

What to do if you are sued

If served with a summons or complaint, call your carrier immediately. Your carrier will assign an attorney. Be candid with the attorney. You will be asked to provide and interpret your dental records. Assemble all available records regarding your patient's treatment and review them thoroughly with your attorney prior to your deposition. Cell and office phone records can confirm calls to the patient, pharmacy, or specialists.

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Screw posts

Screw posts represent a restorative anachronism. The risk of root fracture is too great compared with the benefit, particularly when reasonable and superior passive alternatives exist (see Chapter 23). Even if the screw post is initially placed passively, the temptation to turn the screw is too great, considering human nature. Therefore screw posts are not a reasonable and prudent treatment choice.[68]

Paresthesia prevention

Endodontic surgery in the vicinity of the mandibular canal or mental foramen carries with it the significant risk of irreversible injury to the inferior alveolar or mental nerve. Consequently, the clinician must advise the patient in lay terms of the risk of temporary or permanent anesthesia or paresthesia before any surgery near these structures is performed. To document that adequate informed consent was provided, the clinician should have the patient execute a written informed consent form confirming that the patient was so advised. Informed consent is no defense if the surgery was negligently chosen or performed, because informed consent only applies to nonnegligent treatment risks. CBCT imaging has greatly reduced, if not eliminated entirely, the likelihood of IANC perforation. CBCT provides diagnostic guidance before therapy. Also, a final diagnosis with CBCT can confirm or rule out IANC penetration post treatment to facilitate prompt removal of foreign materials that have penetrated into the IANC. Also, sealants can chemically leak through the paper-thin porous (5% to 10%) IANC cortical borders even if the initial overfill only reaches but does not penetrate the underlying IANC. Delayed onset of paresthesia or dysesthesia up to 4 days can occur with sealant chemicals such as eugenol or resin components.

A clinician initiating root canal therapy on a mandibular posterior tooth must have (1) an awareness of the vital neurovascular bundle containing the IAN, which commonly approximates the ends of these roots, and (2) a diagnostic-quality image that accurately demonstrates both crestal borders of the IANC. Should the radiograph only demonstrate one border of the IANC, it is highly likely the evident border is the inferior crest of the IANC and not the superior crest.[206] If endodontics is done without adequate diagnostic radiographs, the patient is placed at great risk if there is anatomic variation or

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vital neural bundles lie in close proximity to these roots.[110,180,206,289,336] Also be mindful that as the IAN ascends toward the mental foramen, the IAN may have an anterior loop before exiting the mental foramen.[138,175,176,277] Overfilling or overinstrumentation of the root ends can cause traumatic or chemical injury to the IAN bundle, resulting in potentially permanent altered or total loss of sensation from paresthesia (partial numbness), anesthesia (total anesthesia), or dysesthesia (burning pain).[261]

The risk of overfills containing toxic materials causing irreversible neuropathies results from (1) inadequate imaging of the proximity of both borders of the IANC (Fig. 27.28, *A–C*), (2) inadequate measurements during cone measurement, (3) not limiting filling materials to within the confines of the shaped root canal space, and (4) not utilizing CBCT when instrumenting or performing surgery adjacent to vital structures such as the IANC and maxillary sinus.

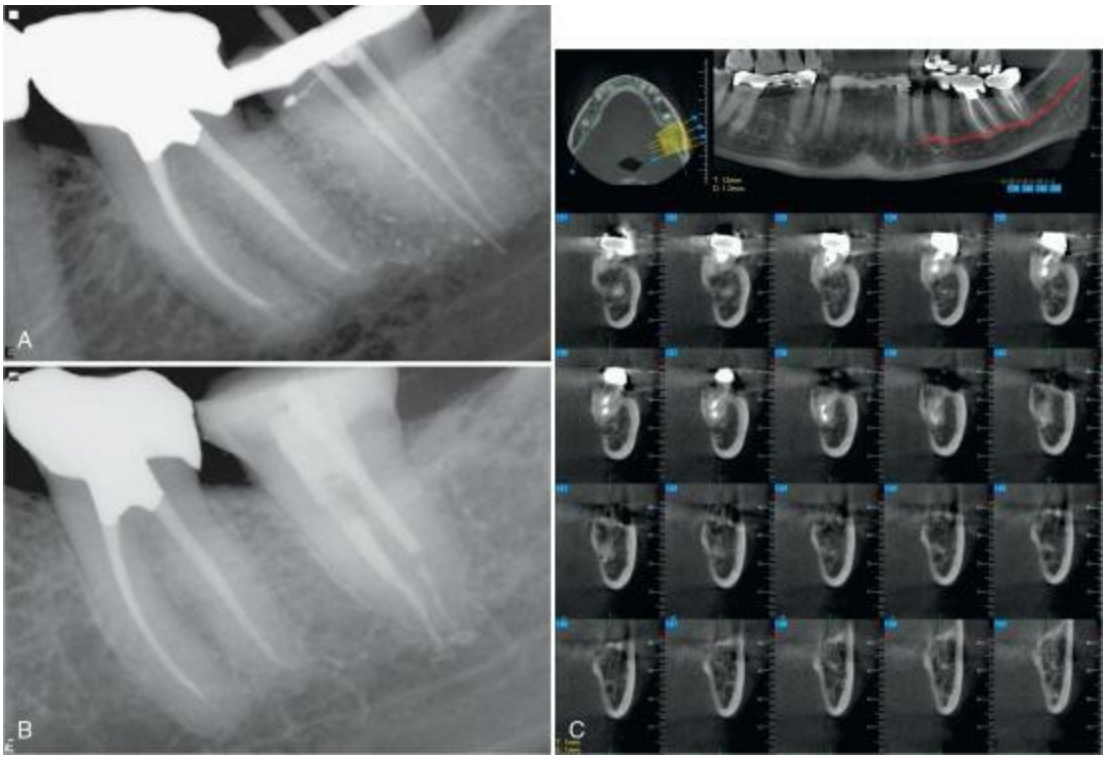

FIG. 27.28 **A,** Unclear periapical radiograph shows both cortical borders of inferior alveolar nerve canal (IANC). **B,** Cones placed into IANC, showing measurements beyond apices. **C,** Overfill into IANC better shown on cone-beam computed tomography (CBCT) than periapicals of **A** and **B.**

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A) Radiograph shows left tooth with radiolucent crown and IAN canals in front of the tooth.

B) Radiograph shows tooth with radiopaque crown and filled root canals.

C) Series of CBCT images show axial and panoramic view of teeth along with twenty coronal views of the first left tooth in the lower jaw.

The eugenol component of the root canal sealer part of the overfill is known to be cytotoxic. Consequently, endodontic overfill of the root apex is capable of producing not only mechanical damage[219] but also chemical injury. Accordingly, a prompt microsurgery referral to remove the injurious materials from the IAN inside the IANC nerve space before overfill material toxins are chemically set is mandatory.[254] Better yet is to take precautions to prevent *any* significant injurious overfill.

The use of electronic measuring devices such as an electronic apex locator is standard technology for accuracy when working deep within the root canal system. Combined with diagnostic quality radiographs, an apex locator helps provide accurate length readings. If it is determined that the apex is in extremely close proximity to the IANC, extra precaution must be taken to prevent overextension of instruments or materials, including slightly short fills and apical packing techniques.

Overfills

A general dentist utilized an electronic apex locator (EAL) to measure his file lengths during cleaning and shaping of the molar root canals. Relying exclusively on the accuracy of his EAL, the dentist did not take any midtreatment radiographs to verify that his EAL measurements were accurate. Unfortunately for both dentist and patient, the dentist had transported one of the root canals with resultant root perforation. Consequently, during obturation with a calcium hydroxide sealant, the root canal was overfilled into the underlying IANC. The patient suffered permanent paresthesia and dysesthesia. The Virgina verdict was $1.5 million (Brenner v. Khalil, Virginia Circuit Court, Arlington County, No. 013CL17000341-00) (see Fig. 27.32, later in this chapter).

Precise measurements during cleaning, shaping, and obturation compacting stages of endodontics minimize significant overfill incidents. Obturating

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filling materials with sealants may be not only toxic to nerve cells but also create a mechanical pressure injury to the nerve cells themselves. Immediate microsurgical referral should be done on a stat basis within the first 48 hours of neuropathic symptoms for retrieval of any overfill materials that "harpoon" the IANC to protect against irreversible neuropathies (see Fig. 27.30, *C,* presented later).[254] Also see animation of an adverse overfill.

Serious mechanical and chemical injury to the IAN results from a lack of recognition of the overfill piercing the IANC or disregard for neuropathic symptoms the patient reports any time between 4 hours and 4 days postoperatively that are consistent with neurologic deficits such as paresthesia or burning pain dysesthesia.

Referral to an oral surgeon/microsurgeon is time-dependent. Pogrel's study demonstrated complete reversal of neuropathic symptoms secondary to overfill if microsurgery was done within 48 hours.[254] Two thirds of Pogrel's patients gained significant improvement if microsurgery was done within 3 months of IAN overfill injuries. Delaying expeditious referral to a microsurgeon for removal of overfilled materials progressively worsens the toxic effects of the chemical injury over time. Delaying microsurgical referral risks permanent injury, owing to the time-dependent 48-hour window for complete reversal or within several weeks to greatly diminish the cytotoxic effects of overfill errors.

Microsurgical delay will close the window of opportunity to prevent permanent neurologic deficits of altered sensation, with resulting persistent chronic pain, numbness, and painful burning symptoms that extrusion overfills into the IANC can cause. Stat referral for microsurgical removal of paraformaldehyde-containing root canal filling materials and sealants is even more critically time dependent because of the greater toxicity and mummifying effects of these powders when mixed into pastes (e.g., N2 and RC2B). Risks include anaphylactic shock.[50,125,144,192]

For needed stat referrals, the referring clinician should phone, fax, and e-mail the microsurgeon. Patients lack the expertise to promptly obtain critically time-dependent referrals. When time is of the essence, the clinician and staff must assume direct responsibility to assure the stat referral to a microsurgeon is promptly appointed. Remind the patient that microsurgical delay is detrimental to corrective care.

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Sealant overfills

Neurotoxic and compressive effects of sealants are the most frequent causes of paresthesia after endodontic overfill.[129,185,262]

Additionally created injuries occur to the maxillary division of the trigeminal nerve or into the maxillary sinus membranes when sealants or bleach is extruded through maxillary teeth. Obturation and sealant materials should be used cautiously in all circumstances, especially in teeth in close proximity to the IANC and maxillary sinus.

The use of syringes for applying endodontic sealers, or obturation with the Lentulo spiral, requires extra precaution when application is done in close proximity to neural structures because precise control is compromised.

Short fills contribute to apical infections and may cause temporary paresthesia. Paresthesia reversal can occur with retreatment, gaining improved cleaning, shaping, and obturation to the anatomic apex.[3]

Standard of care requires that the clinician avoid unreasonable risks to prevent potentially serious or permanent harm to the patient. Treatment is negligent when a reasonably careful clinician should have foreseen and prevented unreasonable patient harm risks.

Treatment failure

A clinician should not guarantee treatment success. It is foolish to assure the patient of a perfect result.[67] Endodontic failures may occasionally occur despite adequate endodontic care.[161,311] Nevertheless, negligent contributing factors to endodontic failures include perforation, missed or transported canal, uninstrumented portion of a root canal, bacterial infiltrates by way of a leaky coronal restoration contaminating the root canal filling,[66,293,298] overextension errors, and inadequate isolation of the tooth from salivary contaminants during instrumentation due to lack of a dental dam.

Successful root canal treatment requires the prevention of microorganism toxins from the oral flora penetrating through the root canal system into the periapical tissues. Inadequate obturation of the root canal system is the most frequent cause of failure. Either coronal or apical leakage adversely affects success.[84,85,298]

Failing endodontics that necessitate endodontic disassembly should first be

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considered for a nonsurgical option because it is less invasive and therefore represents risk reduction. The clinician should first evaluate for coronal leakage, fractures, missed canals, silver point corrosion, and incomplete obturation. Root canal systems can then be recleaned, or correctly transported and then thoroughly disinfected, reshaped, and packed in three dimensions to avoid apical surgery by providing instead adequate coronal, lateral, and apical seal nonsurgically.

To avoid claims based on failed endodontics, the patient should be advised in advance of treatment of the inherent (but relatively small) risk of failure (i.e., about 5% to 10%). It may be adequate to advise the patient of the high statistical probability of success in endodontics if the clinical condition of the tooth and the clinician's past success rate warrant such representation.[145,291] Clinicians should avoid quoting the national success rate of endodontics when (1) the patient's tooth is of questionable endodontic and periodontal status and (2) the clinician is known to have an unusually high endodontic failure rate. A rate that varies markedly from national statistics suggests referral to an endodontist.

An endodontic treating clinician is also liable for failure to disclose evidence of other pathosis in the quadrant being treated. The patient should be advised of any periodontal disease that adversely affects the prognosis of abutment teeth for partial dentures, bridges, or an implant with a need for grafting if the endodontically treated tooth is lost. A clinician should also advise of cysts, fractures, or lesions of suspected neoplasms. In addition, the clinician must be careful not to ignore any evident pathosis that, if untreated, may adversely affect the dental or medical health of the patient. Again, a clinician who fails to plan treatment properly is planning for treatment failure.

Full disclosure

Disclosing a nonharmful mistake will not erode patient confidence. On the contrary, it bolsters trust by reassuring the patient that the dentist is honest and truthful. Increased trust through full transparency mitigates against legal action. Patients who trust their dentist are less likely to bring a malpractice suit. Truthful disclosures, sincere apologies, and appropriate acceptance of responsibility, including reimbursing corrective care, decreases litigation.

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Disclosing is ethical and preserves integrity. “No harm, no foul” is a misguided approach. It is the patient’s right to be informed of any intervention administered in the course of treatment that *could* have caused harm. On the other hand, major errors must be prudently disclosed to the patient for the sake of transparency, trust, integrity, and ethical responsibility.

Slips of the drill

A slip of the drill, like a slip of the tongue, may be unintentional. Nevertheless, it can cause harm. When a cut tongue or lip occurs, it is usually the result of operator error. To paraquote Alexander Pope, “to err is human, to forgive, divine.” To increase the likelihood that a patient will forego filing suit because of a cut lip or tongue, the clinician should follow these steps:

1. Inform the patient that the clinician regrets having injured the patient. This is not a legal admission of guilt in 29 states with “I’m sorry” protective statutes, but rather an admission that the clinician is a compassionate human being. In 2005, federal legislation to apply “I’m sorry” to all 50 states was proposed. The Senate bill sponsored by senators Clinton and Obama did not pass in Congress.
2. Repair the injured tissues or refer the patient to an oral or plastic surgeon, depending on the extent of the injury and whether a plastic revision is necessary if scarring is likely.
3. Advise the patient that the clinician will pay the bill for the referred treatment of the oral or plastic surgeon. Request the oral or plastic surgeon to send the bill directly to the clinician for payment. Send the oral or plastic surgeon’s bill to the clinician’s professional liability carrier. Some carriers will pay the claim under the medical payments provisions of the general liability policy for an “accident” rather than as a malpractice incident compensable under the professional liability policy. Call the patient periodically to check on healing, recovery, and any follow-up plastic or other surgeries.

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Fear factor

U.S. Centers for Disease Control and Prevention's National Center for Health Statistics report that cost is the overriding factor preventing adults from seeking dental care. Fear is the second reason for delayed dental care. Medicaid recipients were at five times greater risk for worse dental health than those with private coverage. Adults on Medicaid were twice as likely to have not visited a dentist in more than 5 years.[319]

Dental fear may result in patients delaying or avoiding dental care.[199] Frequent cancellations and missed appointments are characteristically associated with fearful dental patients. Although it is ordinarily a defense of contributory negligence if patients do not follow a clinician's treatment recommendations, the patient's advocate may contend that the defendant clinician negligently failed to diagnose a fearful patient. Fearful patients tend to avoid dental treatment because they believe it may exacerbate a prior traumatic dental experience. Referral to clinicians who specialize in treating fearful patients should be considered to facilitate comprehensive dental treatment and to avoid future emergency endodontic care because of repeatedly canceled treatment visits.

Dental anxiety and finances are the two most important barriers to patients obtaining regular dental care. Fearful dental patients avoid necessary treatment, delay recalls, and are reluctant to undergo painful procedures. Therefore it is essential that such patients be identified for proper management or referral for fear-reduction therapy. A patient who experiences intense anxiety in the dental chair, together with a history of avoiding dental care, suggests a diagnosis of dental phobia. Fearful dental patients fear loss of control during clinical treatment and require reassurance and reaffirmation that they have the power to halt the procedure by raising a hand or using another appropriate gesture. After trust is gained, additional procedures may be performed.

In addition to desensitizing techniques, the use of proven topical-anesthesia delivery systems helps to ensure a relatively painless injection of local anesthetics. Topical-anesthetic patches and oral-anesthetic rinses may prove a valuable aid for pain management of the fearful patient.

The use of psychological questionnaires, such as the Dental Anxiety Scale

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or the Modified Dental Anxiety Scale, may help to identify such individuals.[154] These simple questionnaires are short, quick, and easy to complete, and users are provided with cutoff scores that help the clinician identify patients who have psychological special needs. In this way, the clinician will be in a position to assist the dentally anxious or dentally phobic patient in accessing dental health care.

Pain management

Buffered local anesthetics are more effective than nonbuffered local anesthetics when used for mandibular or maxillary anesthesia in pulpally involved teeth. Buffering of local anesthetics has 2.29 times greater likelihood of achieving successful anesthesia.[n]

Every day, more than 130 people in the United States die after overdosing on opioids. The Centers for Disease Control and Prevention estimates that the total "economic burden" of prescription opioid misuse alone in the United States is $78.5 billion a year, including the costs of health care, lost productivity, addiction treatment, and criminal justice involvement. In 2017, more than 47,000 Americans died as a result of an opioid overdose, including prescription opioids, heroin, and illicitly manufactured fentanyl. Approximately 21% to 29% of patients prescribed opioids for chronic pain misuse them.[o]

To reduce the risk of opioid addiction, ibuprofen and acetaminophen are the preferred postoperative analgesics. A seminal research study showed that 400 mg ibuprofen/1000 mg acetaminophen was more efficacious than placebo, acetaminophen/codeine and ibuprofen/codeine.[p] See also CDC Guidelines for prescribing opioids for chronic pain.[q]

Leakage

Long-term seal of the root canal system is determined apically by the sealer and coronally by the final restoration.[36,134,228,293] Root canal–filled teeth should be permanently restored without undue delay to prevent leakage contamination of the previously obturated canal system, because varying canal shapes from round to oval prevent a 100% seal.[84,85,202] Bonded seals covering the canal surfaces should be used to control any leakage after

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compaction until a permanent restoration is cemented.[305] Besides an adequate coronal sealing, an adequate apical sealer should be well adhered to the canal walls.

The endodontic goal is to prevent bacterial contamination of the periradicular tissue by predictably providing adequately cleaned, shaped, and filled root canal systems. Any residual bacteria should be entombed in the root canal filling. A bacteria-tight apical seal should be designed to last long term with sealed portals to prevent reentry of microorganisms, which cause reentry recontamination and lead to endodontic failure.[84,298] Younger patients are more susceptible to bacterial penetration inside dentin tubules and thus recurring infections.[177]

Implant risk factors

Patient risk factors such as grinding teeth or diabetes increase the odds of implant failure.[166] Higher implant failure rates occur with surgeons who have less than 5 years of implant experience.[166]

Fluoride for caries prevention

In 2006, the ADA Council on Scientific Affairs published evidence-based research with fluoride recommendations for caries prevention. The ADA recommends that fluoride varnish treatments for at-risk patients should be done between two to four times annually, depending on risk classification. Fluoride varnish containing 5% sodium fluoride with 22,600 ppm fluoride ions help occlude dentin tubules and aid remineralization. Silver diamine fluoride is recognized as safe[r] and effective[s] to reduce the risk of progressive caries causing pulpal exposure.

Reasonable versus unreasonable errors in judgment

Although a clinician is legally responsible for unreasonable errors in judgment, mistakes occasionally happen despite adherence to the standards of reasonable care. A mistake does not prove malpractice unless it is caused by a malpractice error or omission.[141] For example, accessory or fourth canals on molar teeth are frequently difficult to locate and may tax the best clinicians. Failure to locate an accessory or fourth canal does not conclusively

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constitute an unreasonable error of judgment. Rather, this may represent a reasonable error of judgment in the performance of endodontics. Nevertheless, if the additional canal could have been diagnosed radiographically with diagnostic quality radiographs at different angles, the existence of a fourth canal should have been considered. Instrument and seal the extra canal for successful obturation.

Incorrect tooth treatment

A reasonable, nonnegligent mistake in judgment may occur because the clinician has difficulty localizing the source of endodontic pain. Vital pulps may, on occasion, be sacrificed in an attempt to diagnose the pain source, but it is unreasonable and therefore inexcusable to treat the wrong tooth if it is inadequately tested with pulp tests, misidentified on the referral slip, or if radiographs are mounted or read incorrectly. Also, treating large numbers of teeth endodontically (e.g., an entire quadrant) when attempting to localize chronic pain suggests pain is probably not of pulpal origin, and other differential diagnoses should be ruled out including atypical facial pain, referred pain, or TMD pain.

If the wrong tooth is treated because of an unreasonable mistake in judgment, the clinician should be compassionate, waive payment for all endodontic treatment, and offer to pay the fee for crowning the unnecessarily treated tooth.

Post retrieval

Ultrasonic instruments will vibrate and loosen the cement around posts.[68] The clinician can avoid overheating the post by proceeding slowly with a water coolant, periodically resting for cool down, and checking the post temperature periodically to be reasonably certain overheating is not occurring.[96,130,230] Fig. 27.21 demonstrates what happened when an endodontist negligently overheated the post during attempted removal, causing tissue necrosis, bone loss, need for augmentation procedures, irreversible pulpitis in an adjacent tooth, and the loss of two teeth.[252]

Broken files

Leaving broken files behind without referring the patient to an endodontist

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for attempted microscopic retrieval and not advising the patient of leakage potential may constitute fraudulent concealment (Figs. 27.29 and 27.30). Patients should be informed of such mishaps for (1) referral consultation or treatment; (2) advising the patient, who on his or her own may seek a second opinion; or (3) disclosure, so as to return if a flare-up occurs.

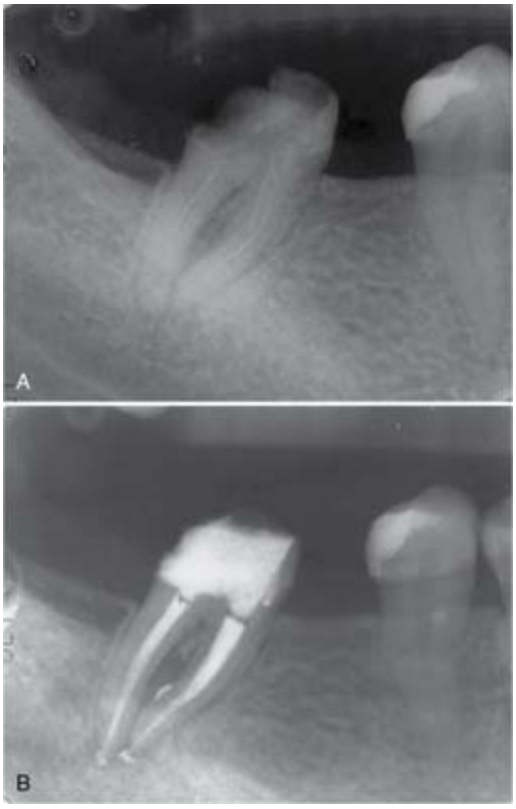

FIG. 27.29 **A,** Tooth #31 with three separated files, one in each canal. K-files used for cleaning canals and also obturation (1996). **B,** No apparent radiographic changes in 1998.

A) Radiograph shows thirty first tooth with radiolucent patches on the crown.

B) Radiograph shows thirty first tooth with filled crown and root canals.

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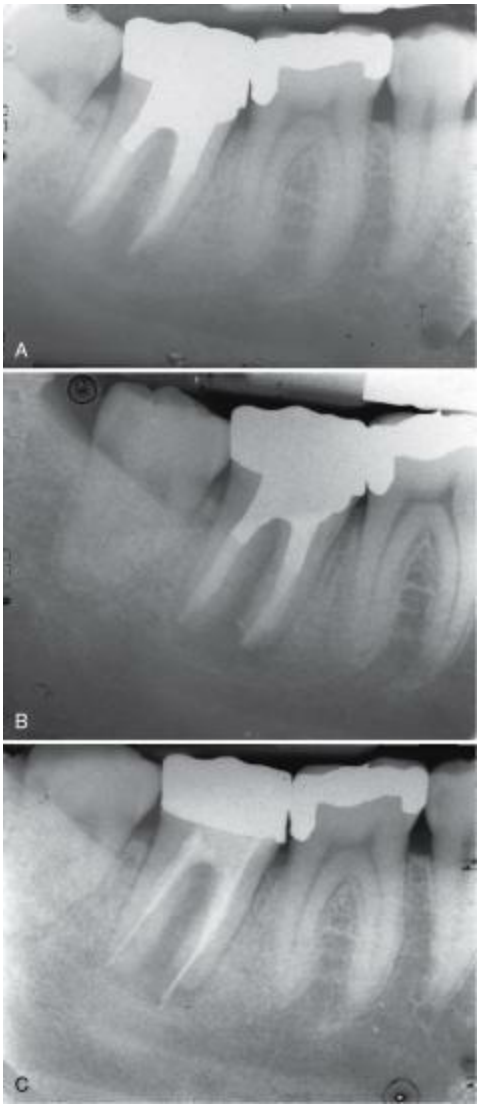

FIG. 27.30 **A,** Apical radiolucent changes with draining 9-mm buccal abscess (August 2003). **B,** Endodontist retreatment (August 2003). **C,** Successful retreatment with no buccal pocket (September 2003).

Set of three radiographs, marked A through C, shows gradual healing during the treatment of less-density bone tissues in buccal.

Source: (Courtesy James Y.S. Ho, DMD.)

Swallowing or aspirating an endodontic instrument

Use of a rubber dam in endodontics is mandatory.[14,294] Even if the endodontically treated tooth is broken down and cannot be clamped, a rubber dam, regardless of required modification, should be used in all instances (see Chapter 7). Not only is microbial contamination reduced with the use of a

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rubber dam, but the risk of a patient's aspirating or swallowing an endodontic instrument is eliminated (Fig. 27.31). Accordingly, if a patient swallows or aspirates a file, it is likely because of the clinician's failure to observe the standard of care. If a swallowing or inhalation incident does occur, the clinician should do the following:

1. Advise the patient that the clinician regrets and is sorry about what occurred.
2. Refer the patient for immediate medical care, including radiographic imaging, to determine if the instrument is lodged in the bronchus, esophagus, or stomach so that appropriate medical measures are taken promptly to remove it.
3. Offer to pay for the patient's out-of-pocket medical expenses and wage loss. Some professional liability policies will cover the incident as "medical payment" for an accident and not as a malpractice claim.

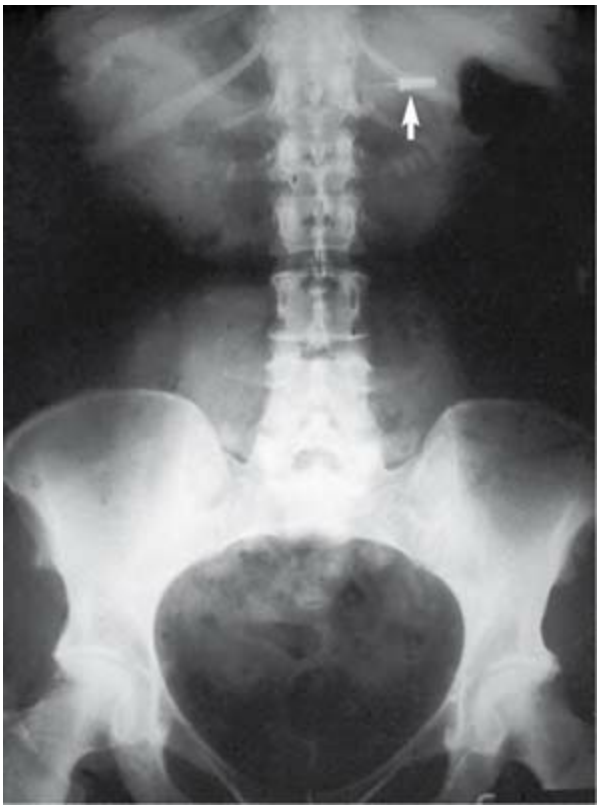

FIG. 27.31 Swallowed endodontic instrument demonstrates the wisdom of using a dental dam.

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Abdominal radiograph shows endodontic instrument in hypochondriac region.

Overextensions and overfills

A common error in utilizing periapical or Panorex imaging is presuming that when only one cortical border of the IANC is visible, the sole visible border is the superior border. Instead, virtually always when only one cortical border is clearly visible, it is the inferior cortical border.[206] (See Fig. 27.30, *B,* periapical, which may appear to show only one cortical border, and Fig. 27.30, *C,* a clearer CBCT image of the same area showing both cortical borders.)

Misassumption of the IANC borders results in mismeasurement of the IANC location. Because the IANC is approximately 3 mm in diameter, misassuming the sole observable cortical canal border to be the superior border (rather than the inferior border) can result in an imprudent treatment decision to delay microsurgery if overfill occurs and belated CBCT confirms an overfill inside the IANC. In Fig. 27.30, *B,* the defendant endodontist misdiagnosed the overfill as being superior to the IANC rather than inside the IAN canal. Resultant permanent paresthesia occurred from watching and waiting for sensory return rather than referring to a microsurgeon for a stat microsurgical removal.

A slight overextension of root canal filling with conventional obturation or sealants can occur without violating the standard of care if not in close proximity to the IANC or sinus (see Chapters 2 and 27). Gross overfill usually indicates faulty technique. Fig. 27.32, *A* and *B,* illustrates gross overfill of calcium hydroxide, causing permanent paresthesia. Nevertheless, so long as the overextension is not in contact with vital structures such as the IANC or sinuses, permanent harm is unlikely. An exception is paraformaldehyde-containing sealants, which cause a neurotoxic chemical burn type of injury that chemically diffuses through bone and soft-tissue spaces.

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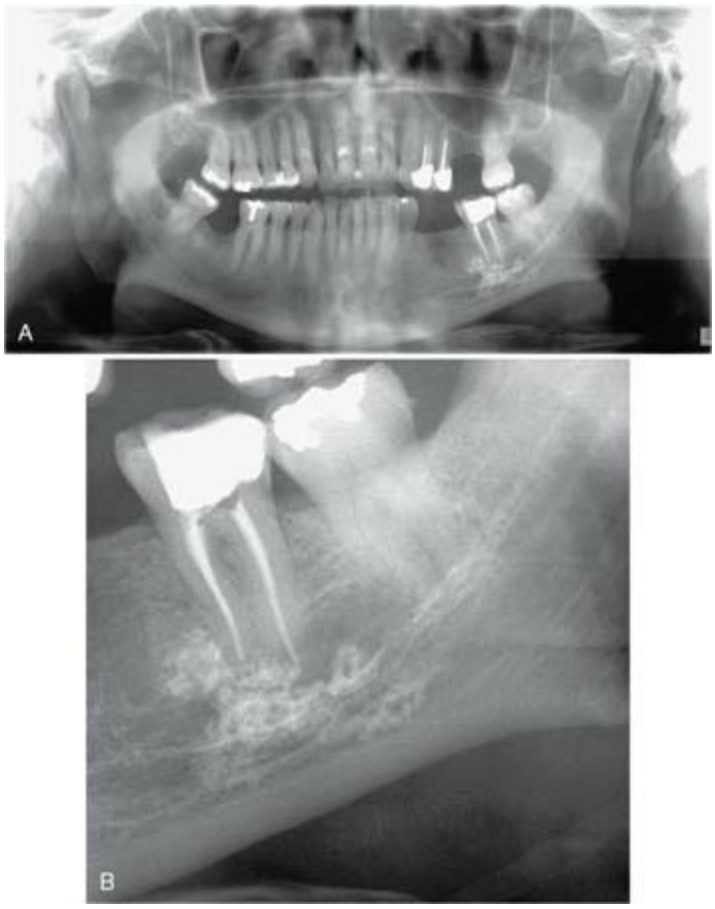

FIG. 27.32 **A–B,** Calcium hydroxide overfill, causing permanent paresthesia.

A) Panoramic radiograph shows the deposition of calcium hydroxide in lower left quadrant.

B) Radiograph shows the calcium hydroxide deposition below the second molar that has radiopaque crown and filled root canals.

If, however, postoperative pain is foreseeable as a result of overextension, the patient should be advised with postoperative written instructions of the likelihood of postoperative discomfort or paresthesia because of suspected contact of the sealant material with surrounding vital structures. A note should be made on the patient's chart of the overextension should symptoms later manifest. The clinician should observe with close follow-up visits and patient phone calls that evening, the next morning, and the following 3 days to rule out postoperative persistent numbness or dysesthesia pain resulting from any overfill. Slight overextensions with inert conventional endodontic sealers, such as gutta-percha with Grossman's sealant, can repair themselves

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and produce no irreversible changes if done without direct contact with the sinus or IANC or indirectly through chemical leakage and seepage beyond the initial area of operation.

Overextending the root canal filling material risks permanent consequences if the underlying IAN is initially "harpooned" with files. A portal of entry into the IANC results from overinstrumentation penetration. Flexible gutta-percha alone probably will not penetrate beyond the mature root into the IANC or other vital structures without prior excessive instrument perforation into the IANC.

Mental foramen distances can be overestimated on the panoramic radiograph. Panoramic radiography may show more deviation (+0.6 mm) from the perioperative measurement.[110,289] Paraformaldehyde-containing sealants can create cytotoxic chemical destruction of the IAN if placed in close proximity to, even though not directly contacting, the underlying IAN (see reference, Sargenti Opposition Society [SOS]).[278] On the other hand, conventional obturators and sealants usually require coming closer to contact with the IAN before resulting permanent anesthesia, paresthesia, or dysesthesia occurs (Fig. 27.33). Consequently, the incidence of permanent sequelae with conventional filling materials is lower than the greater cytotoxic potential with paraformaldehyde-containing sealants.[234] Because of the higher risks associated with paraformaldehyde-containing endodontic materials, use of N2, RC2B, or similar toxic pastes is contraindicated and violates the standard of care.[11] Permanent injury risk is substantially less likely with traditional eugenol-containing filling materials, less toxic chemicals than paraformaldehyde, which destroys and mummifies nerve tissues. When safer, less risky alternative effective therapy exists, it is unreasonable (and substandard) to elect an unsafe alternative methodology. Also, the doctrine of informed consent is highly relevant because it is contrary to public policy to request a patient to assume inherently dangerous treatment risks that are reasonably avoidable with safer and more predictable methodologies. When the risk exceeds the benefits, such risks are regarded as negligent and thus should be avoided and not undertaken.

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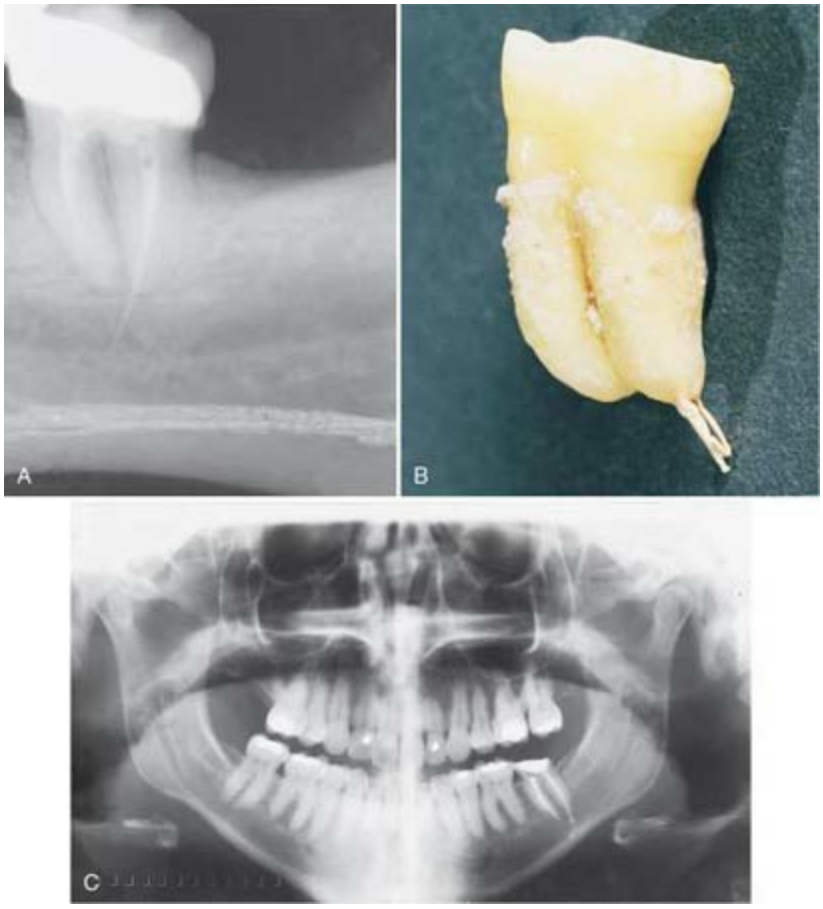

FIG. 27.33 **A-C,** Overextended gutta-percha and extracted tooth with gutta-percha intact.

A) Radiograph shows a tooth, in which the left root canal has extended gutta percha.

B) Specimen shows tooth with gutta percha protruding from the root apex.

C) Panoramic radiograph shows first molar with long gutta-percha in the lower left jaw.

Any significant overextension should be considered for immediate retreatment by attempted retrieval of overextended gutta-percha and sealant.[135] The clinician also has the option of immediately referring the patient to an endodontist for retrieval before the sealant sets. Conventional filling agents such as gutta-percha do not penetrate the cortical walls of the IANC unless probably preceded by prior penetration with overinstrumentation. This principle can apply to sinus perforation. If, despite local anesthesia, the patient feels an electric shock during mandibular molar

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or premolar instrumentation, this may be a warning sign that the IAN was pierced with endodontic files. If this occurs, the root canal should not be filled; instead, periapical radiographs at different angles should be exposed with instruments in place to confirm whether any IAN penetration resulted. Removal of gutta-percha and sealants that have entered the IANC should be attempted as soon as possible (preferably no later than within the first 24 hours).[254] The eugenol component of the sealant causes an inflammatory reaction in a constricted space, which is best relieved by retrieval. If retrieval fails, a decortication microsurgical procedure with an oral surgeon is indicated at the earliest possible time (preferably within the first 24 hours) owing not only to eugenol chemical toxicity but also eugenol reaction with gutta-percha, which causes expansion and thus a mechanical compression ischemic injury.[219,254]

Compartment syndrome

Inflammatory edema that compresses and compromises blood supply to soft tissues and nerves in limited spaces with resulting ischemia is termed *compartment syndrome*.[285,339] Compartment syndromes are a group of conditions that result from increased pressure within a limited anatomic space, acutely compromising the circulation and ultimately threatening the function of the tissue within that space. Compartment syndrome occurs from an elevation of the interstitial pressure in a closed osseofascial compartment that results in microvascular compromise. The pathophysiology of compartment syndrome is an insult to normal local tissue homeostasis that results in increased tissue pressure, decreased capillary blood flow, and local tissue necrosis caused by oxygen deprivation. Compartment syndrome is caused by localized hemorrhage or postischemic swelling. The pathophysiology of compartment syndrome is a consequence of closure of small vessels. Increased compartment pressure increases the pressure on the walls of arterioles within the compartment. Increased local pressure also occludes small veins, resulting in venous hypertension within the compartment. The arteriovenous gradient in the region of the pressurized tissue becomes insufficient for tissue perfusion.

The clinician should have a high index of suspicion whenever a nerve is located inside a closed bony compartment where nerve injury within the

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surrounding neurovascular bundle has the potential for bleeding or swelling. Compartment syndromes are characterized by pain beyond what should be experienced from the initial injury. Also, diminished sensation may be noted in the distribution of the nerve within a compartment that is being compressed, such as the IAN, which is enclosed by bone on all sides.

Root canal followed by root resection without an adequate retrograde restoration will likely fail. Open drainage is no longer recommended. Dissect out subapical granulation tissue cleanly and without damage to vital structures. Caution should be taken when granulation tissues are attached to vital anatomic structures such as neurovascular bundles or sinus linings. A potential for recontamination exists unless the root apex is removed. Apical resection of 3 mm will remove potential problems. This resection will additionally permit inspection. A straight fissure bur design with appropriate air venting is recommended rather than a conventional air rotor to prevent air emphysema.

Currently preferred methodology utilizes specifically designed ultrasonic tips inserted into a piezoelectric ultrasonic handpiece. Amalgam is no longer recommended as a material for root-end filling. MTA and newer silicate trioxide materials are recommended.[243]

Periradicular surgery

Accessing mandibular posterior teeth can generate increased heat during removal of significant quantities of bone to gain apical access. Thus copious coolants must be applied to avoid risking injury to adjacent structures. Molar teeth surgical access should avoid the facial artery as it crosses the border of the mandible adjacent to the first molar.

Avoiding the inferior alveolar nerve canal

When placing implants or performing apicoectomies in close proximity to the mental foramen in the bicuspid region, the clinician should consider the anterior loop of the IAN.[277] The IAN ascends as it approaches the mental foramen, compared with the IAN's more inferior location in the molar region. The IANC varies from buccal to lingual as it courses the length of the mandible. Thus CBCT imaging aids in assessing the IANC location for planning implant placement or apical surgery[75,97] (Fig. 27.34, *A–C*[138]; this

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Los Angeles case settled for $915,000, with permanent dysesthesia).

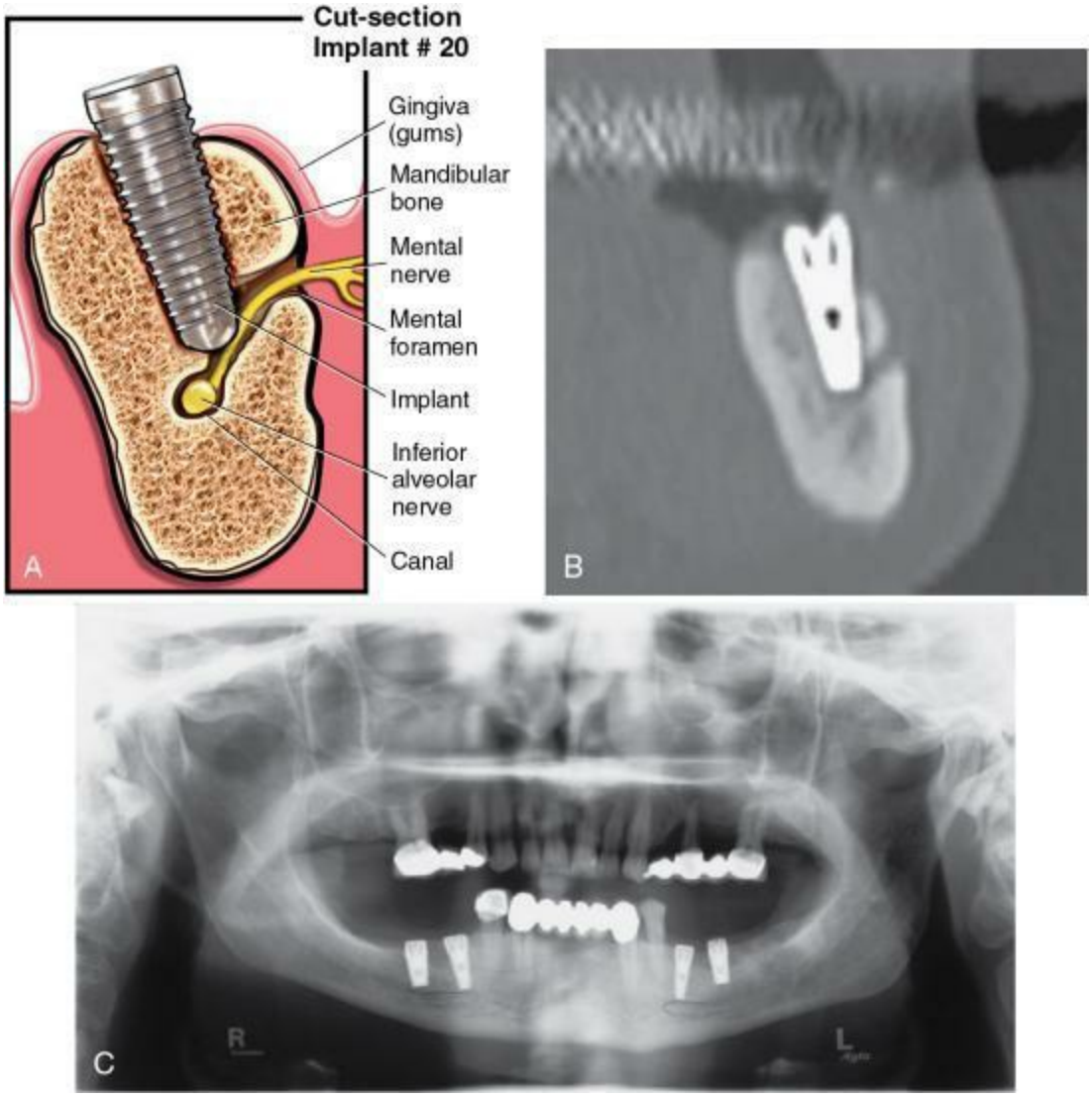

FIG. 27.34 **A,** Illustration showing implant placed into ascending inferior alveolar nerve canal (IANC). **B,** Cone-beam computed tomography (CBCT) image of implant into ascending IANC. Settlement of $915,000. **C,** Implant into IANC; immediate postoperative Panorex image. Same patient but with CBCT image.

A) Cross-section diagram of tooth shows labels as follows: Gingiva (gums), mandibular bone, mental nerve, mental foramen, implant, inferior alveolar nerve, and canal.

B) Radiograph shows tooth with implant.

C) Panoramic radiograph shows four implants in lower jaw.

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Fig. 27.35 illustrates avoidance of the IANC to demonstrate that IANC penetration with the implant or drill is a preventable adverse event. (Also see Fig. 27.38, later in this chapter.)

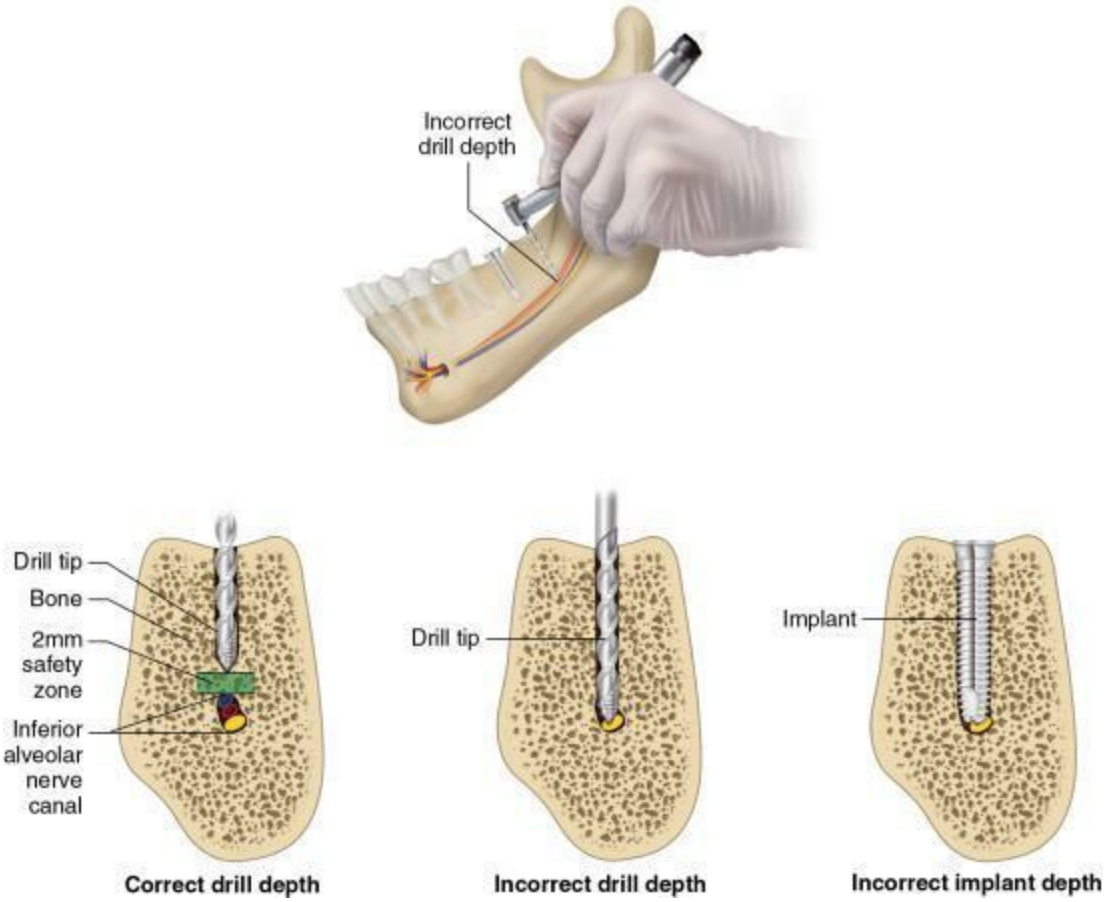

FIG. 27.35 Medicolegal implant image.

The gloved hand places drill into the nerve in the lower jaw. Label reads, incorrect drill depth.

Labels with correct drill depth are as follows: drill tip, bone, 2 millimeters safety zone, and inferior alveolar nerve canal. In incorrect drill depth, the drill tip is directly placed deep into the nerve canal. In incorrect implant depth, the implant is inserted deep into the nerve canal.

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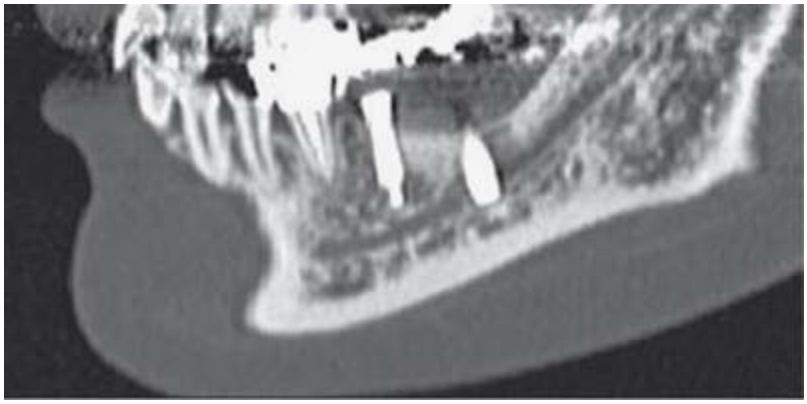

FIG. 27.38 Implant “harpooned” the inferior alveolar nerve canal.

Radiograph shows lower jaw with implants in the inferior alveolar nerve canal.

Mental nerve injuries

The mental nerve, as the terminal branch of the IAN, is at risk during elevation of the mandibular mucoperiosteum. Careful flap design and elevation are important to avoid mental nerve injury while working on the buccal surface of the mandible in the region of the mental foramen.[207] In performing incision and drainage in the region of the mental foramen, a diagnostically accurate radiograph is necessary to avoid incising through the course of the mental nerve.[84,180]

When carrying out PRS in the mandible, consideration must be given to the path of the inferior dental nerve bundle in the mandibular canal, which lies in close proximity to the root apices.[277] Be aware of the position of the mental nerve exiting through the mental foramen. A shallow sulcus, coupled with a prominent mandibular protuberance, together with the lingual inclination of the roots may make access to the root apices difficult. Approximately 40% of lower incisors have two root canals, which may preclude adequate resection and retrograde filling of the root apex, which may ultimately lead to treatment failure.

Local anesthesia: Septocaine

Septocaine and Articaine are not suitable for an inferior dental nerve block due to the potential increased risk of paresthesia and nerve damage.[91] Prudent practice is to avoid utilizing 4% solutions of local anesthetics, such as

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Septocaine and Articaine, during inferior alveolar block injections. When the risk exceeds the benefit, the reasonably careful dentist wisely chooses to not subject the patient to such an unreasonable risk of harm. Both Septocaine and Articaine are associated with increased risks of permanent neuropathic injuries of paresthesia and/or dysesthesia resulting from mandibular block injections with these 4% solutions. Since efficacious results can be achieved with conventional 2% local anesthetics such as lidocaine, Septocaine and Articaine are contraindicated for mandibular blocks.[t]

It is the dentist's choice and decision which local anesthetic to utilize for a particular endodontic procedure. A patient should not be offered a choice between a reasonably acceptable local anesthetic and an unreasonable choice of a 4% solution for a mandibular block. Informed consent only applies to nonnegligent treatment risks and alternative choices. A patient therefore should not be asked to consent to a negligent choice of Septocaine or Articaine for a mandibular block injection.

Implant versus endodontics

A natural tooth with its periodontal support, function, and proprioception is superior to an implant-retained restoration. Whenever reasonably feasible, teeth should be retained.

Reasonable clinicians may differ in recommending extraction versus PRS depending on location near vital structures and clinical conditions. Extraction of the diseased tooth and placement of a dental implant has a better long-term prognosis and thus should be the treatment of choice is one author's conclusion.[275] Conversely, every reasonable effort should be made to retain the natural tooth with its periodontal support, function, and proprioception. Thus an implant-retained restoration should be considered as the last possible treatment option as concluded by other clinicians.[24,73,310]

Infraocclusion of the implant restoration in teenage patients with residual craniofacial growth occurs as the adjacent teeth erupt. Therefore delay implant placement until dental and skeletal maturation occurs.[309]

Aesthetics is an important outcome for patients receiving implant-supported restorations. To achieve this outcome, patients require good bone volume and quality, as well as soft-tissue height. Following extraction, the alveolar bone resorbs vertically, as well as horizontally (buccolingually).

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Socket preservation intervention grafting may aid in reducing the bony dimensional changes after tooth extraction, but ridge resorption is not completely prevented. Intervention techniques including autografts, allografts, xenografts, guided bone regeneration (GBR) and growth factors accomplish varying degrees of success in maintaining the anatomic dimensions of the alveolus before implantation.[54]

Current use of silver points

Based on what has been known for more than 3 decades, use of silver points in lieu of gutta-percha or other conventional endodontic filling materials represents a departure from the current standard of care.[13] This is because silver points corrode in time, and a tight 3D apical seal is lost. Fig. 27.36, *A–C*, represents gross overextension with a silver point that ultimately caused the loss of tooth #14 as a result of endodontic failure.

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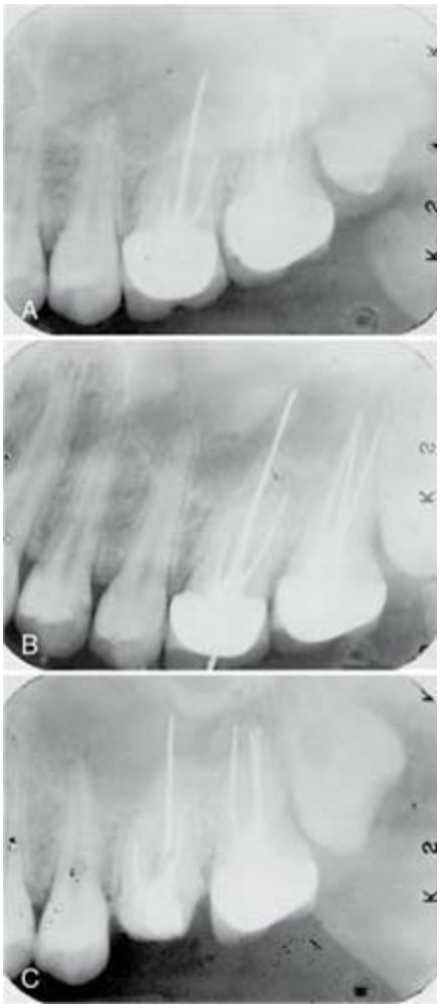

FIG. 27.36 Gross overfill into sinus with a silver point, which ultimately caused sinusitis and loss of tooth #14 as a result of endodontic failure.

Set of three radiographs, marked A through C, shows the changes in length of gutta-percha taking place during the loss of fourteenth tooth.

The AAE Position Statement does not recommend the prophylactic revision of silver point obturation, unless there is clear evidence of endodontic pathosis or if the silver points complicate proper restoration of the tooth.[13]

N2 (sargenti paste)

Dental literature reports that permanent paresthesias are associated with gross overfilling with paraformaldehyde sealant (N2) (Fig. 27.37).[200,234] Current use of paraformaldehyde-containing endodontic sealants is not merely the result of a philosophic difference between two respectable schools of thought.

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Rather, the distinction is between the reasonable and prudent school of thought that advocates conservative conventional endodontics and the imprudent and radical school of paraformaldehyde providers who unreasonably risk permanent, deleterious injury with N2 overextensions. Regardless of the small number of clinicians professing to use N2, it is cytotoxically unsafe and should be avoided. A customary negligent practice by some clinicians is no defense to safe and prudent practice, which the standard of care requires. No matter how few or how many do it wrong, this never makes it right.

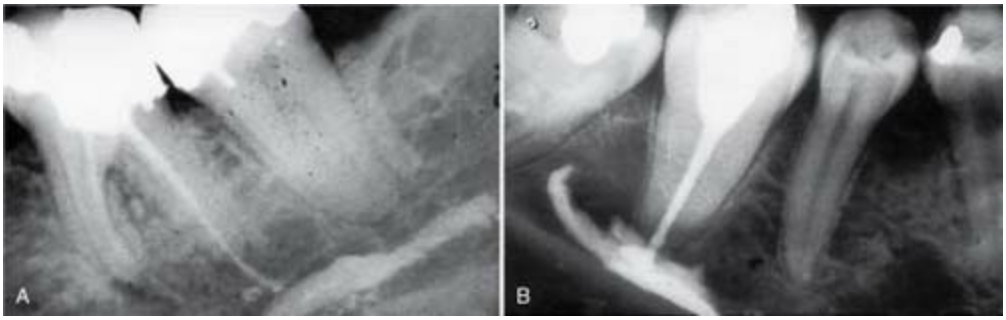

FIG. 27.37 **A** and **B,** Overextensions of Sargenti paste filling the inferior alveolar nerve canal. Both cases could have been avoided if the practitioners had selected a conventional sealing material and used a technique that emphasizes working length control.

Set of two radiographs, marked A and B, shows two different teeth with extended N2 sealant.

The AAE recommends against the use of paraformaldehyde-containing materials as they have proved to be both unsafe and ineffective. Accordingly, the use of such materials is below the standard of care for endodontic treatment.[11]

Clinicians may be liable for fraudulent concealment, intentional misrepresentation, or co-conspiracy if they discovered that a previous clinician's negligence is the cause of dental disease and both the prior clinician and subsequent treater concealed the prior clinician's negligence. For instance, if a gross overextension of a paraformaldehyde packing or sealant is evident radiographically and the patient reports that another clinician caused the overfill (that resulted in permanent lip and chin anesthesia), subsequent treating clinicians may be liable for fraudulent

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concealment if they misinform the patient that the anesthesia will probably disappear shortly and that using N2 merely reflects a philosophic difference rather than substandard practice. Some clinicians who differ on therapies regard such differences as controversial. The difference between standard of care and substandard practice is not controversial but rather an indisputable difference between right and wrong. For instance, quackery based on pseudoscience is not controversial. Instead it is fraudulent practice.[37,224] Likewise, if the radiographs indicate sealant is inside the IANC and the patient complains of persistent anesthesia, the patient should not be told to wait for return of sensation. The clinician should refer the patient immediately for microsurgical consultation for decortication and decompression surgery.[263]

The Federal Food, Drug, and Cosmetic Act prohibits interstate shipment of an unapproved drug or individual components used to compound the drug.[100] On February 12, 1993, the FDA Dental Advisory Panel confirmed that N2's safety and effectiveness remain unproven. N2 may not be shipped interstate or distributed intrastate if any of the N2 ingredients were acquired interstate. Mail-order shipments of N2 from out-of-state pharmacies in quantities greater than for single-patient use are considered a bulk sales order rather than a prescription, thus violating FDA regulations.[63] A San Francisco jury awarded punitive damages against an N2-distributing New York pharmacy for knowingly shipping N2 in violation of FDA regulations, done with deliberate disregard for patient safety.[162]

Defective restorations

Marginal gaps greater than 50 μm lead to cement dissolution and cause 10% of crown failures within 7 years after cementation. Dull or worn explorers substantially increase the likelihood of nondetection of open margins. A sharp explorer can detect margin defects as small as a 35-μm opening.[36] Accordingly, a sharp clinician should utilize a sharp explorer to detect open margins. Open crown margins contribute to endodontic failure and should be avoided (see Chapter 23).

Restoration marginal integrity

A pathologic apical lesion will resolve when the origin of infection is

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eradicated, the root canal system effectively sealed, and a satisfactory coronal restoration placed. Many clinical studies report a high success rate for nonsurgical root canal treatment (NSRCT) of greater than 90% but a weighted long-term average of 83%.[u]

Malpractice prophylaxis

Most negligently injured patients do not sue.[51] Nonetheless, litigation serves a prophylactic purpose because litigation fears make some clinicians more careful and help promote professional guidelines. For example, after the American Society of Anesthesiologists adopted practice guidelines to reduce patient harm, deaths and professional liability premiums decreased dramatically.[252,283]

Allergies

All dentists must be periodically trained in handling dental emergencies. Dental Practice Acts typically require training in Basic Life Support for dental license renewal. A common misconception by both patient and dentist is that reactions to local anesthetics were due to an allergic reaction. Allergic reactions from local anesthetics are extremely rare. Instead the patient more likely has experienced an overdose of local anesthetic from inadvertent injection into the bloodstream. Such syncope reactions require reassuring the patient and administering oxygen.

To avoid overdosing with local anesthetics or causing a toxic dosage of a vasoconstrictor such as epinephrine, two precautions are necessary. First, aspirate the local anesthetic syringe in two planes perpendicular to each other. Second, inject slowly. Both precautions are designed to prevent the injection of local anesthetics into adjacent blood vessels.[v]

One-visit versus two-visit endodontics

One-visit endodontics, if done well, will allow disinfection of the canals and obturation without risk of recontamination through the coronal seal between visits. Treatment of apical periodontitis with signs or symptoms of a beginning lesion is determined by the sound judgment choice of the clinician. Either one or two visits will comply with the endodontic standard of care.[242]

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Disclosure errors

Should dental negligence harm a patient, then full disclosure, including accepting the clinician's responsibility along with providing an apology and explanation, results in the best outcome for the clinician and patient. Also, assurance of efforts to prevent a recurrence results in greater patient satisfaction and retains trust with the disclosing clinician to sustain a continuing clinician-patient relationship. Patients want to be told of treatment errors, regardless of whether the error can be corrected. One study in which there was full disclosure of negligently caused therapeutic errors resulted in only one patient thereafter seeking legal advice.[210] In the same study, patients paradoxically acknowledged, "It is realistic to expect that doctors will make errors.... Patients have a right to expect that their doctors will not make errors." Nonetheless, virtually all patients concluded, "Patients should be able to trust their doctors to give them the right care." As noted earlier, apologetic expressions of sorrow or empathy for a patient's injuries are barred in evidence from being construed as admissions of fault in 29 states.[182] Protecting the patient's best interest includes telling the patient when negligent errors occur.

Temporomandibular disorders

Occlusal prematurities can trigger TMDs in patients with a prior TMD history, so during or following endodontic therapy for such a patient, it is essential that temporary and final restorations not open the bite or significantly alter existing occlusion.[194] Hyperocclusion is a potential TMD trigger that can result from open bite or on a contralateral side, when a terminal abutment tooth is taken out of occlusion.

Patient rapport

Good patient relations are 15% dependent on the clinician's competency to cure and 85% dependent on the clinician's ability to assure the patient that the treatment being given will be done with the clinician's best judgment and care.

Rapport between clinician and patient reduces the likelihood the patient will sue, despite an adverse result. The clinician can develop rapport by

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demonstrating genuine interest in the patient and making the patient feel valued. Patients feel important if they are seated in the operatory within a reasonable time after arriving. The longer a patient is kept waiting, the more frustration and animosity build. If the patient cannot be seen within a reasonable time, a staff member should communicate the reason and, if appropriate, offer to reschedule the appointment. Staff or the clinician should telephone the patient at the end of the day after any difficult procedure or surgery to check on the patient's status and remind the patient to follow postoperative instructions. The clinician should record any patient complaints, symptoms, and noncompliance with instructions. The latter can be used as evidence of patient contributory negligence if litigation occurs.

Rapport building blocks

The best communicators listen more than they speak. When they do speak, it is mostly to clarify what the patient has said.

In discussing the patient's complaints, the clinician should ask, "What do you think is causing the problem?" Otherwise, the clinician may solve the patient's dental problem while failing to solve the patient's perceived problem. For instance, a patient may fear that a retained endodontic file is carcinogenic unless this fear is allayed with a careful explanation that it is not.

Telephone communications

Good telephone communication is a matter of asking the right questions, such as asking a patient complaining of postoperative swelling if there is difficulty breathing or swallowing, as well as the degree and location of swelling. In cases of suspected infection, clinicians should ask the patient or family member to call back with a temperature reading to verify the patient is afebrile.

Post perforation

Post selection is important for minimizing the risk of perforations.[68] Generally, posts should not exceed one third of the mesiodistal width of a tooth, should follow the canal anatomy, and should leave 4 to 5 mm for sealant in the opened post space; this is described more thoroughly in Chapter

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23. When a tooth is already crowned, access to the root canal entrance surface should be located before rubber dam placement. This will aid in orienting the root's long axis, which may vary from the prosthetic crown's long axis. Fig. 27.39, *A* and *B*, represents an endodontist's perforation of tooth #7 because of difficulty locating the entrance into the root canal apical to the pulp chamber.[107] Fig. 27.40 shows a general clinician's misaligned post and perforation of tooth #5.

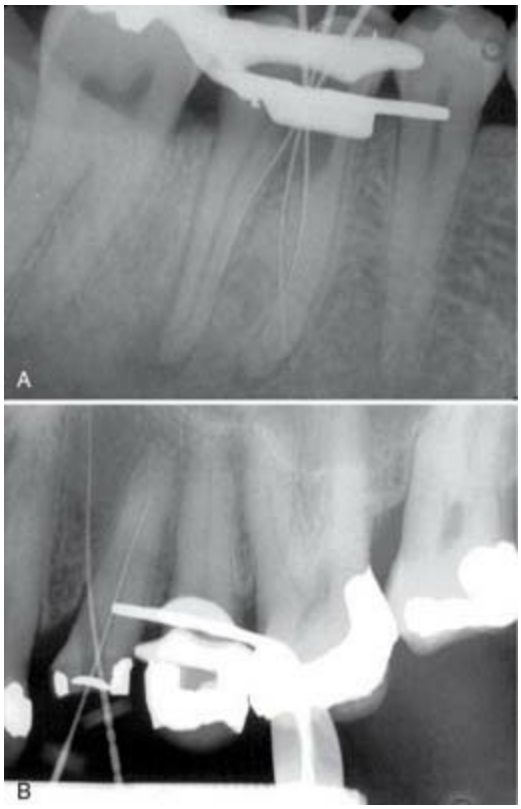

FIG. 27.39 Avoidable perforation.

A) Radiograph shows three gutta percha extended along the tooth.

B) Radiograph shows two gutta percha extended beyond the root of seventh tooth.

Source: (Courtesy Stephen Cohen, MA, DDS.)

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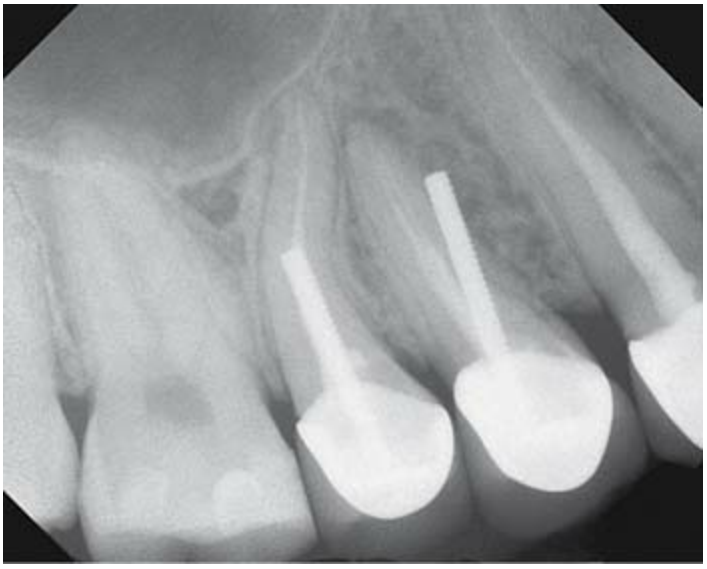

FIG. 27.40 Post perforation of tooth #5.

Radiograph shows four teeth with the second tooth having implant extended up to mid of the root and third tooth having oblique implant.

Ordinarily, a careful clinician performing endodontic therapy should be able to avoid post perforations. If post perforation occurs, early diagnosis and treatment are important. Belated diagnosis and treatment substantially increase the risk of endodontic failure. If the perforation is relatively small (i.e., 1 mm or less) and promptly diagnosed at the time of the post perforation, immediate treatment with intracanal sealants (MTA) or tricalcium silicate in the area of the perforation will probably succeed. However, delayed diagnosis and treatment (beyond 24 to 72 hours) result in bacterial contamination in the area surrounding the perforation. Delayed perforation repair therapy can cause periodontal or endodontic lesions and lateral periodontal abscesses occurring secondary to delayed diagnosis, which usually prognosticate a high failure risk.

Perforation prevention

Irreversible endodontic complication, such as furcation perforation during pulpal chamber access, is usually preventable with due care.[228] Furcation perforations are avoided by adhering to basic principles of pulpal chamber removal such as cleaning and shaping in a coronal rather than an apical direction. Innovative bur designs can access the pulp chamber without risking

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furcation perforation by preventing cutting or ditching of the pulp chamber by use of a non-end-cutting bur.[87]

Bleach injury

Because of the varying shapes of the root canal system in the apical third after cleaning, shaping, and obturation, some residual bacteria may remain, which are entombed. Sodium hypochlorite is the usual disinfectant irrigant used to minimize these remaining bacteria.[268] Apical or lateral extrusion occurs when bleach is extruded past the protective apical constriction or through a root perforation. The common denominators of bleach injuries are sudden severe pain and facial swelling.[2,101,186,214,249,260,268,336] Because of the clinician's concern for emergency treatment and the patient's pain, further endodontic therapy is deferred and usually not completed at the same visit. However, two cases report that compaction was completed during the same visit, despite a bleach injury.[215,276] One article reports that subsequent flare-up may be related to the original bleach injury.[52]

Prevention of bleach extrusion is done with bleach syringes with safe, side-venting needles constantly moving to prevent hydrostatic pressure buildup from bleach fluid. Sodium hypochlorite is extruded into the periapical tissues during root canal therapy if excessive pressure is placed on the irrigating syringe, resulting in an expression of sodium hypochlorite beyond the apex. Subsequent pain is caused by the solution diffusing into the surrounding bone and thire, causing swelling, tissue necrosis, and paresthesia. Surgery is usually contraindicated, and palliative treatment such as cold compresses accompanied by reassurance that most symptoms will dissipate is usually adequate.[186] However, if the bleach diffuses into vital nerve tissue of the infraorbital or IANs, permanent paresthesia and occasional dysesthesia may result.[101,214,249,336] Figs. 27.41 and 27.42, *A–C,* represent bleach injuries secondary to a root canal perforation. Paresthesia persists. Note the periapical film does not show the perforation, but the CBCT does.

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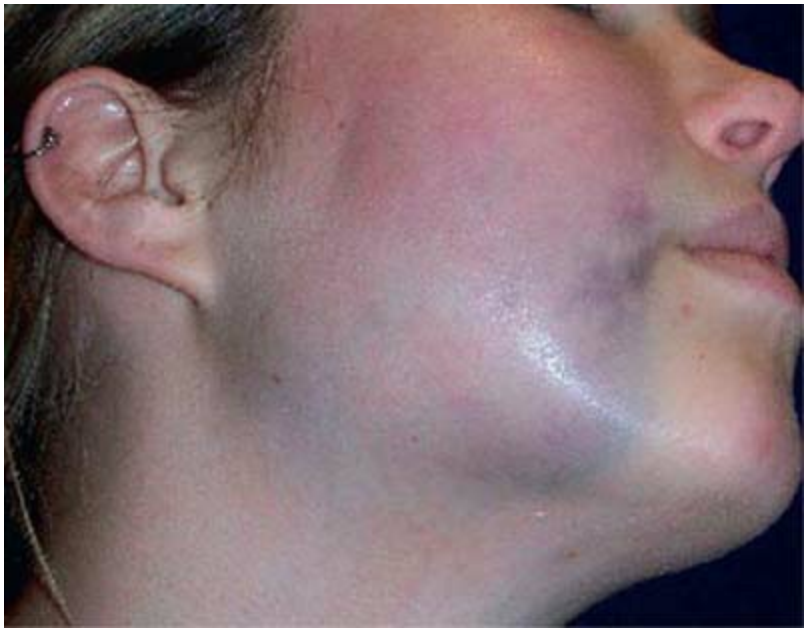

FIG. 27.41 Bleach injury.

Close-up of side view of the patient's face shows swollen buccal region on the left with bluish patches.

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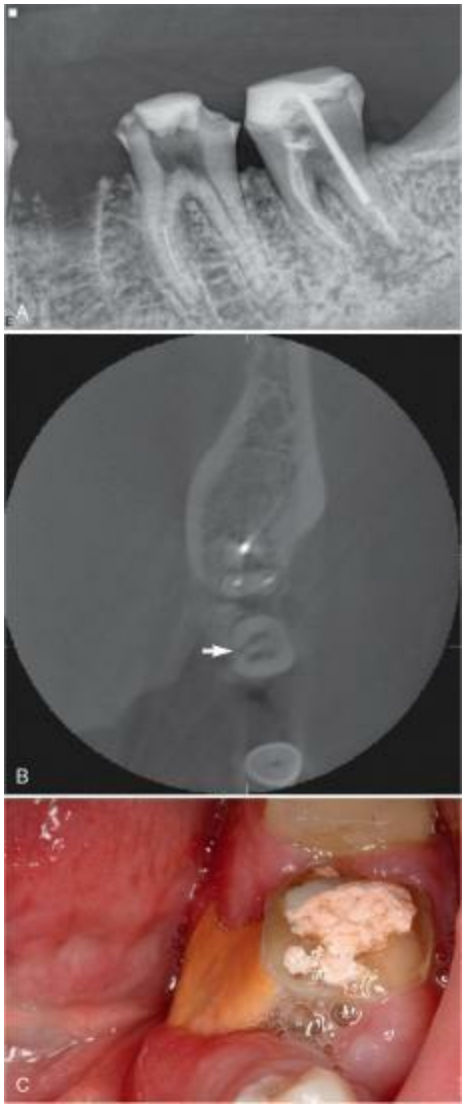

FIG. 27.42 **A,** Periapical tooth #19 showing fractured file but no perforation. **B,** Accuitomo cone-beam computed tomography (CBCT) showing perforation on the lingual side above the tooth #19 furcation. Perforation not evident in periapical view. Image demonstrates Accuitomo accuracy. **C,** Bleach extrusion through tooth #19 perforation, with resultant slough of lingual tissue.

A) Radiograph shows nineteenth tooth with fractured file near the root apex. The adjacent tooth has radiopaque pulp chamber and radiolucent root canals. The gum has porous tissue.

B) Radiograph shows a tooth with oval chambers.

C) Close-up view of nineteenth tooth shows bleach squeezing out from the crown.

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Esthetic bleaching

Use of 35% H_2O_2 as a bleaching agent can be clinically adverse in the long term or after recurring bleaching treatments.

Precautions include the following:

- Reduce hydrogen peroxide concentration
- Reduce the time of each application, and increase the time between applications
- Avoid reaction catalysts, such as lamps or lasers
- Apply with a dental dam

Post-bleaching sensitivity is mostly due to reversible pulpitis that may progress in some cases to irreversible pulpitis.

Cores

Incorrect choice of cores can contribute to failure, including fractures. Some manufacturers (e.g., 3M ESPE, St. Paul, MN, for Ketac silver) recommend against use of their core material unless at least two thirds of the tooth remains before buildup. Failure to follow the manufacturer's directions can be considered when an expert determines whether the standard of care was met.

Resin-reinforced post-and-core systems show promise for structurally weakened incisors, but long-term longevity has not been reported. A ferrule or other counterrotational core design is an important consideration for fracture resistance and retention,[155] although it has not been proven to be statistically significant for the resin-reinforced core systems.[279]

Absorbable hemostatic agents causing neuropathy

Absorbable collagen hemostatic agents should not be placed on or adjacent to peripheral nerves because of the potential for neural injuries, particularly in bony nerve canals.[105,200] Also, as this material is absorbed, its chemotactic properties promote collagen formation and scarring. Compression injuries of peripheral nerves in bony canals can result from expanding scar tissue. FDA adverse incident reports list 11 patients with severe neural defects, including

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paraplegia secondary to absorbable collagen products placed in the spinal canal for hemorrhage control. Thus the FDA warned of paralysis from absorbable hemostatic agents.[105,200] Accordingly, after hemorrhage is controlled with hemostatic agents, absorbable collagen agents should not be left in situ or near bony neural spaces. This will avoid having remaining hemostatic material cause swelling, pressure, or migration to adjacent neural tissues. The minimum amount of collagen agents necessary to achieve hemostasis should be used.

Pneumomediastinum (air embolus)

When performing endodontic surgery, a surgical handpiece should be used that ventilates air through the back of the handpiece rather than an air turbine that directs air into open tissue spaces. Pneumomediastinum, also known as *mediastinal emphysema*, may result from air embolisms dissecting down the neck facial planes from air forced into the submandibular or sublingual spaces contiguous with neck spaces.[40,301]

Broken needle

Scanning electron microscopy (SEM) with an energy-dispersive x-ray spectrometer attachment can analyze whether a broken instrument is due to excessive operator trauma or a manufacturing defect. Fig. 27.43 represents a combination of low-cycle bending fatigue and tensile overload. Accordingly, the local anesthetic needle likely fractured because of bending deformation as it struck the ramus.

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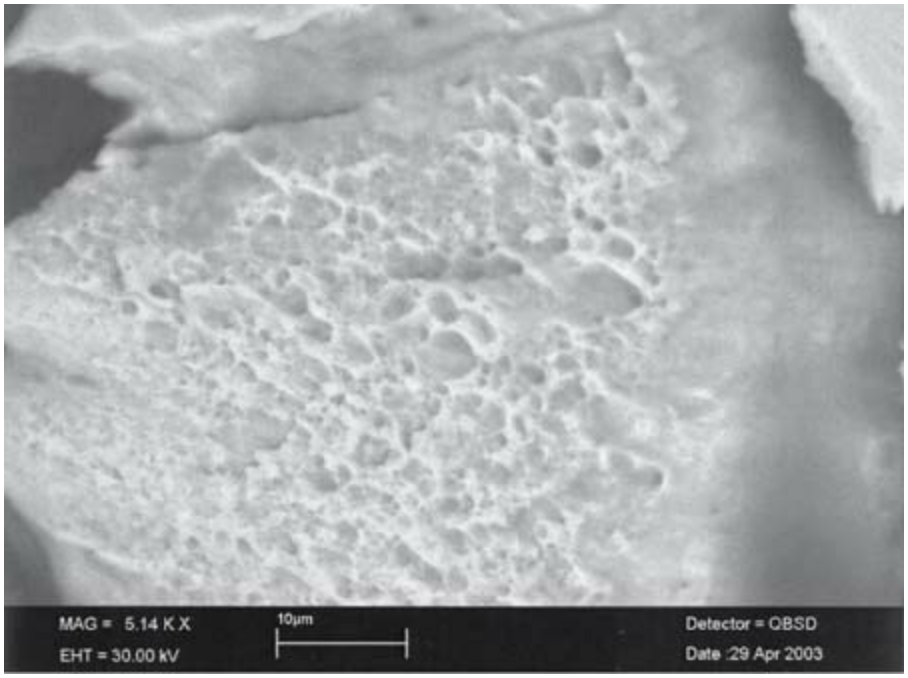

FIG. 27.43 Tensile overload shown in elongated ductile dimple voids.

Scanning electron micrograph shows ductile fracture. The scale reads, 10 micrometers.

Ductile rupture in metals results from shearing along planes that are oriented at 45 degrees to the tensile stress. Tensile "necking" results from multiple slipping deformations in all the 45-degree planes to the needle axis because of significant overload. Final rupture results from a combination of bending and tension. Fig. 27.44 shows the needle tip bent and blunted as it hit a hard object. No manufacturing defects were found. Fig. 27.45, *A* and *B*, shows SEM views of the fracture. Fig. 27.45, *C*, is a SEM view of the needle fracture taken at the hub side of the fracture. These illustrations demonstrate why longer 25-gauge needles should be used for mandibular blocks rather than shorter 30-gauge needles.[207]

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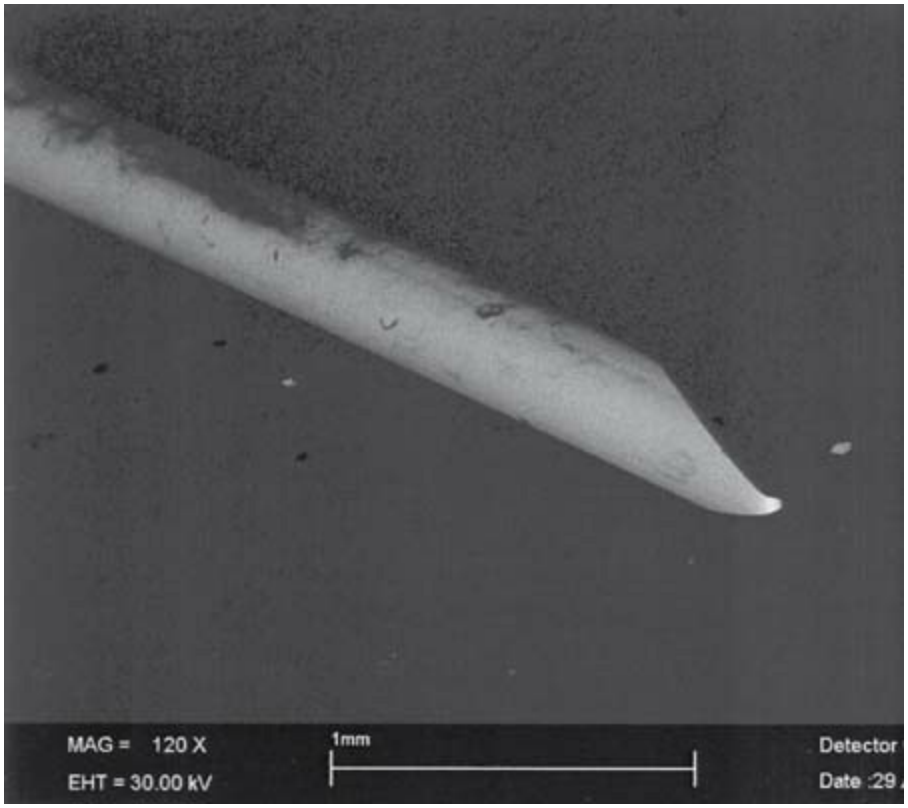

FIG. 27.44 Needle blunting of broken needle tip demonstrating striking hard object (ramus).

Scan shows curved tip of the needle. The scale reads, 1 millimeter.

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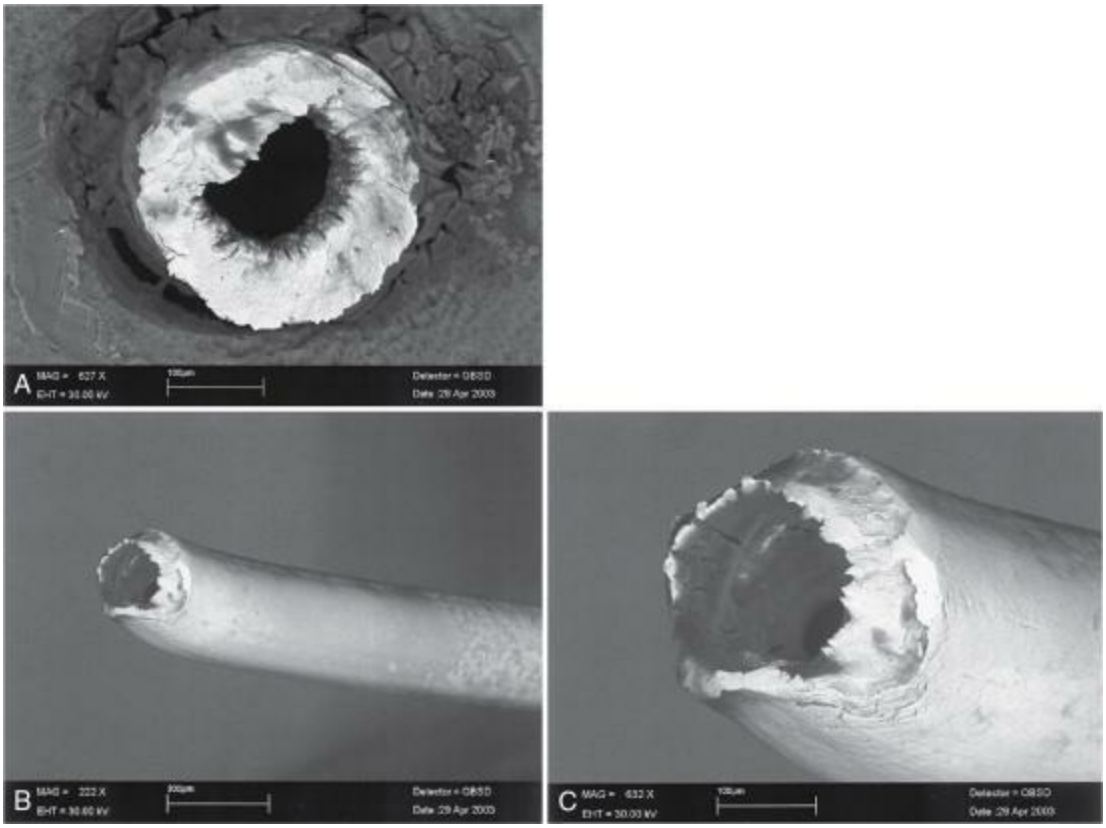

FIG. 27.45 **A,** Needle fracture with scanning electron microscopy (SEM). **B,** Needle fracture, side view (SEM). **C,** Needle fracture at high SEM magnification.

Set of three scanning electron micrographs, marked A through C, shows top, side, and highly magnified view of fractures at needle tips.

Causes of a broken local anesthesia needle that are preventable are as follows[207]:

- Use of a 30-gauge needle for mandibular block
- Bending needle
- Insertion of needle to the hub
- Multiple use of the same needle

Sterilization

The nonsurgical use of the dental unit water supply should comply with the Environmental Protection Agency regulatory standard to not exceed 500 CFU/mL.[188]

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CDC guidelines require sterile water or sterile solutions for all surgical procedures.[188] Disinfected water from handpieces or ultrasonic devices is no longer acceptable as a water coolant during surgery. Instead, an ultrasonic device that can deliver a sterile irrigant during surgery should be used. Dental unit waterlines are available with tubing that can be disposed or autoclaved for surgery. Some ultrasonic devices are also available with disposable tubing to maintain a sterile water coolant delivery system.

Rotary NiTi files likely contain bioburden material that, after sterilization, may act as a foreign body. Efficiency degradation after sterilization should suggest to the prudent clinician a single-use philosophy.[31]

Digital radiographic devices that contact the oral mucosa region require a combination of barrier protective sheath and chemical disinfection in accordance with prudent manufacturers' directions.[151]

Medication errors

The FDA has received more than 95,000 reports of medication errors since the year 2000. Medication errors involve the wrong drug, an extra or wrong dose, omission of a drug, or administering a drug by the wrong route or at an incorrect time.[190]

Medication errors are often preventable.[306] Two major sources of errors in prescribing are poor penmanship and the use of error-prone abbreviations. Writing "1.0 mg" can be read as "10 mg" if the decimal point is not clearly visible. The FDA recommends that no trailing zeros be used when denoting doses expressed as whole numbers and that preceding zeros be used whenever a decimal point is needed for a dose that must be administered as a fraction of a whole number (e.g., 1 mg, 0.5 mg). Recent drug label changes can be readily accessed on the National Library of Medicine's DailyMed website.

Posttrauma therapy

The reader is referred to Chapter 21 for a full discussion of how to treat and manage patients who have sustained a traumatic injury.

Millennium management of endodontic advances

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Microsurgical endodontics is an example of improved endodontic technology; use of magnifying loupes or similar devices may prove inadequate for apical surgery or fractured instrument retrieval compared with microscopes. Therefore the clinician should adopt proven improvements in the endodontic field. Three-dimensional reconstruction of mandibles can be accomplished with cone-beam volumetric tomography to accurately locate the course of the IANC or mental foramen. Fig. 27.46 shows an example of volumetric tomography usage for implant placement or apical surgery.

FIG. 27.46 Cone-beam computed tomography usage for implant placement or apical surgery.

Specimen shows lower jaw with stained implant.

If studies demonstrate significantly superior results for some alternative to surgical endodontics, the informed consent standard of care may require that the patient be advised of the alternative technique, even if it is more expensive. There may be more than one path to success. So long as the clinician uses reasonably acceptable techniques and informs the patient of reasonable alternatives, the standard of care is met. Microscopic apical surgery has gained general acceptance, is performed by the majority of endodontists, and represents the current standard of care.[15,76,166,183]

Statistical research

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The standard of care usually does not mandate incorporation of every new technology. However, in those states with informed consent laws that are based on what a prudent patient would want to know rather than what prudent clinicians should do, the patient may argue that an alternative technology or technique used by a different clinician would have been chosen had the clinician provided the patient with such information. Microscopic endodontics for broken file retrieval and CBCT to aid apical surgeries and implant placement are pertinent examples of what a reasonable patient would wish to know.[75,157]

Other clinicians' substandard treatment

Clinicians should not be overly protective of blatant examples of another clinician's substandard dental treatment. On discovery of apparent negligent treatment by a previous clinician, the clinician should consider investigation. Begin by obtaining the patient's written authorization for transfer of a copy of the previous clinician's records, including radiographs. If negligence is still suspected after reviewing the records, the clinician should consider talking with the previous clinician to learn the circumstances of what occurred during the patient's past treatment (after obtaining the patient's written consent pursuant to HIPAA).

On discovery of a gross violation of the standard of care, a clinician has an ethical responsibility to report the matter to the local dental society, peer review, dental licensing board, or agency.[18,122] If the patient was misinformed and later discovers patently obvious negligent treatment, the subsequent clinician could arguably be sued as a co-conspirator to fraudulent concealment of the prior clinician's neglect.

Peer review

If, despite good rapport, candid disclosure, and an offer to pay corrective medical or surgical bills, the patient is still unsatisfied, the clinician should consider referring the patient to peer review. Peer-review committees award damages for out-of-pocket losses but not for pain and suffering or lost wages. Consequently, even if the committee's decision is adverse to the clinician, the damage award will probably be less than a jury's verdict. If peer review finds for the clinician, the patient may be discouraged from proceeding further with

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litigation. Peer review proceedings, including the committee's decision, are not admissible in court.[61] Insurance carriers usually honor and pay a peer-review committee award, because a fair adjudication of the merits has been determined; the award is usually less than a jury would award. Also defense costs, including attorney's fees, are saved with peer review.

Human Immunodeficiency Virus and Endodontics

A clinician may not ethically refuse to treat an HIV-seropositive patient solely because of such diagnosis.[82] Although in the 1980s, no federal law had clearly extended the protection of the handicapped laws to patients with AIDS, federal congressional action in 1990 extended this protection to the dental office setting with the passage of the Americans with Disabilities Act.[23] Many states already offer additional protection under state law.[59,83]

Confidentiality for patients disclosing their HIV status is important. An inadvertent disclosure to an insurance carrier or to other third parties without any need to know may result in cancellation of the patient's health, disability, or life insurance. This cancellation could result in a claim against the clinician whose office disclosed the information without authorization. Therefore employees should sign the confidentiality agreement shown in Fig. 27.47. In signing this agreement, the staff may be alerted to the seriousness and importance of maintaining the confidentiality of patient health histories, because these histories may document AIDS, venereal disease, or other socially stigmatizing diseases.

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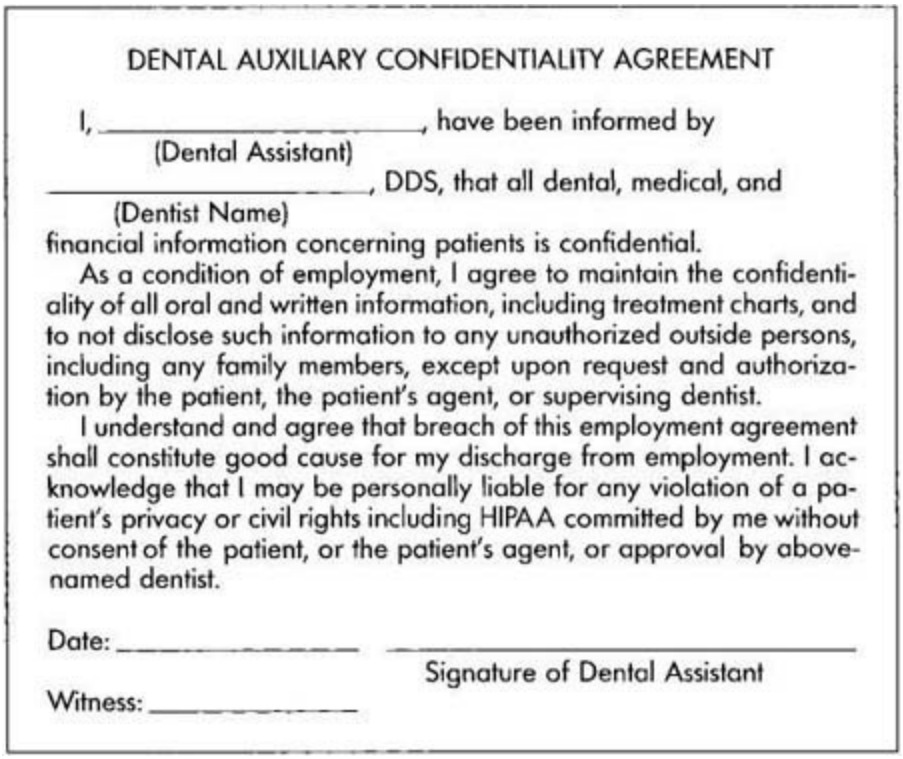

DENTAL AUXILIARY CONFIDENTIALITY AGREEMENT

I, _____, have been informed by
(Dental Assistant)
_____, DDS, that all dental, medical, and
(Dentist Name)
financial information concerning patients is confidential.

As a condition of employment, I agree to maintain the confidentiality of all oral and written information, including treatment charts, and to not disclose such information to any unauthorized outside persons, including any family members, except upon request and authorization by the patient, the patient's agent, or supervising dentist.

I understand and agree that breach of this employment agreement shall constitute good cause for my discharge from employment. I acknowledge that I may be personally liable for any violation of a patient's privacy or civil rights including HIPAA committed by me without consent of the patient, or the patient's agent, or approval by above-named dentist.

Date: _____ _____
Signature of Dental Assistant

Witness: _____

FIG. 27.47 Dental auxiliary confidentiality agreement.

Dental auxiliary confidentiality agreement is as follows:

I, blank space (dental assistant), have been informed by blank name (dentist name), DDS, that all dental, medical, and financial information concerning patients is confidential. As a condition of employment, I agree to maintain the confidentiality of all oral and written information, including treatment charts, and to not disclose such information to any unauthorized outside persons, including any family members, except upon request and authorization by the patient, the patient's agent, or supervising dentist.

I understand and agree that breach of this employment agreement shall constitute good cause for my discharge from employment. I acknowledge that I may be personally liable for any violation of a patient's privacy or civil rights including HIPAA committed by me without consent of the patient, or the patient's agent, or approval by above-named dentist.

Date: Blank space.

Witness: Blank space.

Signature of Dental Assistant with a blank space above.

If a patient requests that the clinician not inform the staff of his or her HIV status, the clinician should refuse to treat that patient. This information is

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essential to staff members who may come in contact with the infection.[48] An accidental needle stick with HIV-infected blood, which carries a risk of approximately 1 in 250 chances of seroconversion, may occur. Current medical protocol includes prophylactic administration of zidovudine (also known as azidothymidine [AZT]), either to prevent or to slow the manifestation of AIDS from a deep, penetrating, accidental needle-stick exposure.

Although a treating clinician risks devastating a dental practice by informing patients that the clinician has contracted AIDS, the legal risk of not informing patients is much greater. The health care provider may be required to advise patients of positive HIV test results under the doctrine of informed consent (i.e., advising of a known risk of harm from accidental exposure).[88] Even if an uninformed patient never contracts AIDS, in those states that use a reasonable patient standard for disclosure of material risks of treatment (which could include accidental direct contact such as an unintended cut or needle stick), the patient may decide to bring an action for intentional concealment as a variant of informed consent and seek to recover emotional distress and punitive damages. Conversely, patients may be legally liable for intentionally misrepresenting their health history regarding their HIV status.[51]

Summary

If the clinician performs endodontics within the standard of care as described in this chapter, there should be little concern that a lawsuit for professional negligence will be successful. Prophylactic measures suggested in this chapter should lessen the likelihood of litigation by reducing, if not eliminating, avoidable risks associated with endodontic care.

Both the patient and the clinician benefit from risk reduction.[119] To do it wrong does not take long, but it is far better for the clinician to take the extra precautionary time to do it right. We are in a profession that deserves the public's trust, but only if that trust is earned. Clinicians deservedly earn public trust by providing safe and excellent quality patient care. The best prophylaxis against being sued is to make protecting the patient's best interest our paramount goal. No matter how many others do it wrong, this

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never makes it right. Our ethical and legal obligation adheres to the identical bedrock principle, which is service to our patients first and foremost.

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References

1. Aetna Health, Inc. v. Davila, 542 U.S. 200 (2004).
2. Agency for Toxic Substances and Disease Registry. Medical management for calcium hypochlorite and sodium hypochlorite 2009; Department of Health and Human Services Atlanta www.atsdr.cdc.gov/MHMI/mmmg184.html.
3. Ahonen M, Tjaderhane L. *Endodontic-related paresthesia: a case report and literature review*, J Endod 2011;37: 1460.
4. Akkayan B, Gulnez T. *Resistance to fracture of endodontically treated teeth with different post systems*, J Prosthet Dent 2002;87: 431.
5. Alcaraz Martinez Beneyto M, Parra C, Y, et al: *Is it true that the radiation dose to which patients are exposed has decreased with modern radiographic films?*, Dentomaxillofac Radiol 2009;38: 92.
6. Alexander RE. *Eleven myths of dentoalveolar surgery*, J Am Dent Assoc 1998;129: 1271.
7. Allerbring M, Haelgerstam G. *Chronic idiopathic orofacial pain: a long-term follow-up study*, Acta Odontol Scand 2004;62: 66.
8. Deleted in page review.
9. American Academy of Periodontology. *Guidelines for periodontal therapy*, J Periodontol 2001;72: 1624.
10. Deleted in page review.
11. American Association of Endodontists: AAE Position Statement. Concerning paraformaldehyde-containing endodontic filling materials and sealers, 1998.
12. American Association of Endodontists: Endodontics—

Telegram: @dental_k

www.konkur.in

Colleagues for Excellence, Prescription for the Future—Reasonable use of antibiotics in endodontic therapy (vol 2), spring/summer 1999.

13. American Association of Endodontists: *AAE Position Statement. Use of silver points*, 2007.
14. American Association of Endodontists: *AAE Position Statement. Dental dams*, 2010.
15. American Association of Endodontists: AAE Position Statement. Use of microscopes and other magnification techniques, 2012.
16. American Association of Endodontists: AAE Position Statement. Scope of endodontics: regenerative endodontics, 2013.
17. American Dental Association (ADA) Council on Scientific Affairs. *Dental unit waterlines: approaching the year 2000*, J Am Dent Assoc 1999;130: 1653.

17a American Dental Association Council on Scientific Affairs. *The use of prophylactic antibiotics prior to dental procedures in patients with prosthetic joints*, JADA 2015;4: 11.

18. American Dental Association (ADA): *Principles of Ethics and Code of Professional Conduct § I.B*, 2005. www.ada.org/prof/prac/law/code/index.asp. Accessed October 8, 2009.
19. American Dental Association Council on Scientific Affairs: Dental radiographic examinations: recommendations for patient selection and limiting radiation exposure, Revised 2012.
20. American Dental Association. *The use of cone-beam computed tomography in dentistry: an advisory statement from the American Dental Association Council on Scientific Affairs*, J Am Dent Assoc 2012;143: 899.

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www.konkur.in

21. American Dental Association and American Academy of Orthopaedic Surgeons: *Prevention of orthopaedic implant infection in patients undergoing dental procedures*, February 2012.
22. American Medical Association: *Survey*, 2010.
23. Americans with Disabilities Act of 1990, 42 U.S.C. §§ 12101 et seq.
24. Andersson L, Kahnberg KE, Pogrel MA. Oral and maxillofacial surgery 2010; Wiley & Blackwell 310.
25. Andrews N, Vigoren G. *Ergonomics: muscle fatigue, posture, magnification, and illumination*, Compend Contin Educ Dent 2002;23: 261.
26. Angell M. *The pharmaceutical industry—to whom is it accountable?*, N Engl J Med 2000;342: 1902.
27. Angell M. *Industry sponsored clinical research: a broken system*, JAMA 2008;300: 1069.
28. Annas GJ. *Globalized clinical trials and informed consent*, N Engl J Med 2009;360: 2050.
29. Aoyama D. *Tangled webs and truth*, WSMA Report 2004.
30. Arai Bunting v. Jamison, 984 P.2d 467 (Wy. 1999).
31. Arens FC, Hoen MM, Steiman HR, et al: *Evolution of single-use rotary nickel instruments*, J Endod 2003;29: 664.
32. Armstrong J. *Retailer held liable for ephedra stroke*, San Francisco Daily Journal 2004; Aug 27.
33. FDA accused of silencing Vioxx warnings, *The Dallas Morning News*, Oct 8, 2004.
34. Axelrod K. *Even Bush administration data discredits civil justice myths*, Trials 2009; Jan 11.
35. Baghdadi R: The FDA roundtable part I: The regulators' view of drug development, RPM Report 4, 2008.
36. Baldissara P, Baldissara S, Scotti R. *Reliability of tactile*

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www.konkur.in

perception using sharp and dull explorers in marginal opening identification, Int J Prosthodont 1998;11: 591.
37. Barrett S: Be wary of Sargenti root canal treatment, Dental Watch, 2008. www.dentalwatch.org/questionable/sargenti/overview.html Accessed September 8, 2009.
38. Barton v. Owen, 71 Cal.App.3d 484, 139 Cal.Rptr. 494 (1977).
39. Bates DW, Cullen DJ, Laird N, et al: *Incidence of adverse drug events and potential adverse drug events: implications for prevention*, JAMA 1995;274: 29.
40. Battrum DE, Gutmann JL. *Implications, prevention, and management of subcutaneous emphysema during endodontic treatment*, Endod Dent Traumatol 1995;11: 100.
41. Benjamin P. *Pain after routine endodontic therapy may not have originated from the treated tooth*, J Am Dent Assoc 2011;142: 1383.
42. Benovitz N, Haller C. *New evidence of harm from herbal supplement: UCSF team cites 54 deaths since mid 90s*, San Francisco Chronicle 2000; Nov 7.
43. Berhelsen CL, Stilley KR. *Automated personal health inventory for dentistry: a pilot study*, J Am Dent Assoc 2000;131: 59.
44. Berry J. *Physicians report boost in use of world wide web*, ADA News 2000;31: 3.
45. Bhulla K, Waglechner N, Pawlowski A, et al: *Antibiotic resistance is prevalent in isolated cave microbiome*, PLoS One 2012;7: e34953.
46. Black's law dictionary ed 9 2010; West Publishing Eagan, MN.
47. Boodoo JM. *Compounding problems and compounding*

Telegram: @dental_k

www.konkur.in

confusion: federal regulation of compounded drug products and the FDAMA circuit split, Am J Law Med 2010;36: 220.

48. Boulais v. Lustig, Los Angeles Superior Court (California), Case No. BC038105.
49. Bragdon v. Abbott, 524 U.S. 624 (1998).
50. Braun J, Zana H, Purohit A, et al: *Anaphylactic reactions to formaldehyde in root canal sealant after endodontic treatment: four cases of anaphylactic shock and three of generalized urticaria [rash]*, Allergy 2003;58: 1210.
51. Brennan TA, Leape LL, Laird NM, et al: *Incidence of adverse events and negligence in hospitalized patients: results of the Harvard Practice Study*, N Engl J Med 1991;324: 370.
52. Brown DC, Moore BK, Brown CE Jr, et al: *An in vitro study of apical extrusion of sodium hypochlorite during endodontic canal preparation*, J Endod 1995;21: 587.
53. Brown R, Hadley JN, Chambers DW. *An evaluation of Ektaspeed Plus film versus ultraspeed film for endodontic working length determination*, J Endod 1998;24: 54.
54. Bryne G. *Socket preservation of implant sites*, J Am Dent Assoc 2012;143: 1139.
55. Burton TM. *Unfavorable drug study sparks battle over publication of results*, Wall St J B1 2000; Nov 1.
56. Cal Insurance Code, § 533.
57. Cal Practice Guide (Rutter) Insurance Litigation, §§ 13:192-213:451.
58. California Business and Professions Code, 1680(s), 1683(a).
59. *California Court upholds ban on AIDS discrimination*, ADA News 1990;21: 8.
60. California Dental Association. Improved patient bill of

Telegram: @dental_k

www.konkur.in

rights unveiled, California Dental Association Update 1996; California Dental Association Sacramento.
61. California Evidence Code, § 1157.
62. California Health and Safety Code, § 123110(j).
63. Cedars North Towers Pharmacy, Inc. v. U.S.A, District Court, Fla, Case No. 77-4965 (Order on Motion for Summary Judgment dated Aug. 18, 1978).
64. Cedars-Sinai Medical Center v. Superior Court, 18 Cal. 4th 1, 954 P.2d 511 (1998).
65. Centers for Medicare and Medicaid Services, Office of the Actuary, National Health Statistics Group. National health care expenditures data 2012; Baltimore January.
66. Cheung G. *Endodontic failures*, Int Dent J 1996;146: 131.
67. Christ v. Lipsitz, 99 Cal.App.3d 894 160 Cal.Rptr. 498 (1979).
68. Christensen GJ. *Are posts really necessary in endodontically treated teeth?*, Dent Econ 2012; Nov.
69. Clarke v. Hoek, 174 Cal.App.3d 208, 219 Cal Rptr. 845 (1985).
70. Cleveland JL, Foster M, Barker L, et al: *Advancing infection control in dental care settings*, J Am Dent Assoc 2012;143: 1127.
71. Cobbs v. Grant, 8 Cal.3d 229, 502 P.2d 1 (1972).
72. C.F.R. § 60 (1989).
73. Cohn SA. *Treatment choices for negative outcomes with non-surgical root canal treatment: non-surgical retreatment vs. surgical retreatment vs. implants*, Endod Topics 2005;11: 4.
74. Colline W. *Should I stop taking herbs before surgery?*, WebMD 2000; Sept 16.
75. Cotton TP, Geisler TM, Holden DT, et al: *Endodontic applications of cone-beam volumetric tomography*, J

Telegram: @dental_k

www.konkur.in

Endod 2007;33: 1121.
76. Creasy JE, Mines P, Sweet M. *Surgical trends among endodontists: the results of a web-based survey*, J Endod 2009;35: 30.
77. Croskey, Heeseman, and Popik, California Practice Guide Insurance Litigation. §§ 13:192-113:451, 2009; California Insurance Code, § 533.
78. Daubert v. Merrell Dow Pharmaceuticals, Inc, 509 U.S. 579 (1993).
79. Daum v. SpineCare Medical Group, Inc, 52 Cal.App.4th, 61 Cal.Rptr.2d 260 1285 (1997).
80. Davenport MS, Dillman JR, Cohan RH, et al: *Effect of abrupt substitution of Gadobenate dimeglumine for gadopentetate dimeglumine on rate of allergic-like reactions*, Radiology 2013;266: 773.
81. Davies P. *Fatal medical errors are growing problem*, Boston Globe 2004; July 27.
82. Davis M. *Dentistry and AIDS: an ethical opinion*, J Am Dent Assoc Suppl 9-5, 1989;119: 95.
83. Decision and Order of the New York City Commission on Human Rights, Whitmore v. Northern Dispensary (Aug 17, 1988).
84. De-Deus G, Murad C, Paciornik S, et al: *The effect of the canal-filled area on the bacterial leakage of oval-shaped canals*, Int Endod J 2008;41: 183.
85. De-Deus G, Reis C, Beznos D, et al: *Limited ability of three commonly used thermoplasticized gutta-percha techniques in filling oval shaped canals*, J Endod 2008;34: 1401.
86. Department of Health. An organization with a memory: Report of an expert group on learning from adverse events in the NHS 2000; Stationery Office London.

Telegram: @dental_k

www.konkur.in

87. Deutsch AS, Musikant BL. *Morphological measurements of anatomic landmarks in human maxillary and mandibular molar pulp chambers*, J Endod 2004;30: 388.
88. Doe v. U.S. Attorney General, 814 F. Supp. 844 (N.D. *Cal.* 1992).
89. Dolan v. Borelli, 13 Cal.App.4th, 816, 16 Cal.Rptr.2d 714 (1993).
90. Dominici JT, Clark S, Scheetz J, et al: *Analysis of heat generation using ultrasonic vibration for post removal*, J Endod 2005;31: 301.
91. Dower J. *Articaine v. Lidocaine*, CDA J 2007;35: 240.
92. Edwards BJ, Hellstein JW, Jacobsen PL, et al: *American Dental Association Council on Scientific affairs Expert Panel on Bisphosphonate—Associated Osteonecrosis of the Jaw. Updated recommendations for managing the care of patients receiving oral bisphosphonate therapy: an advisory statement from the American Dental Association Council on Scientific Affairs*, J Am Dent Assoc 2008;139: 1674.
93. Eke PI, Dye BA, Wei L, et al: *Prevalence of periodontitis in U.S. adults*, J Dent Res 2012;91: 914.
94. Emmott LF. *Essential questions regarding data, backup, and online backups*, J Am Dent Assoc 2004;135: 1005.
95. Eriksson AR, Albrektsson T. *Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit*, J Prosthet Dent 1983;50: 101.
96. Eriksson R, Adell R. *Temperatures during drilling for the placement of implants using the osseointegration technique*, J Oral Maxillofac Surg 1986;44: 4.
97. Estrela C, Bueno MR, Azevedo BC, et al: *A new periapical index based on cone beam computed*

Telegram: @dental_k

www.konkur.in

tomography, J Endod 2008;34: 1325.
98. Evans AW, Leeson RMA, Newton-John T, et al: *Are oral surgeons as good as they think they are?*, Br Dent J 2005;198: 765.
99. Fda.gov/radiation-emittingproducts/radiationemittingproductsandprocedures/r Rays/ucm315011.htm.
100. FDCA 21 U.S.C. § 301 et seq. (2008).
101. Farren ST, Sadoff RS, Penna KJ. *Sodium hypochlorite chemical burn*, NY State Dent J 2008;74: 61.
102. Federal Register 58:43442-43445, Aug 16, 1993.
103. Federal Rule of Civil Procedures 37(b)(2)(C).
104. Federal Truth in Lending Act, 15 U.S.C. §§ 1601 et seq.
105. Feigal DW: FDA public health notification: paralysis from absorbable hemostatic agents, April 24, 2004.
106. Felland L, Reschovsky JD: *More nonelderly Americans face problems affording prescription drugs*, *Tracking Report* 22, Center for Studying Health System Change, January 2009, Accessed October 8, 2009 at www.hschange.com/CONTENT/1039.
107. Flores v. Michaelian, San Mateo County Superior Court (California), Case No. CIV430666, 2003.
108. Flowers v. Torrence Memorial Hospital Medical Center, 8 Cal.4th 992, 884 P.2d 142 (1994).
109. Figini L, Lodi G, Gorni F, et al: *Single versus multiple visits for endodontic treatment of permanent teeth*, Cochrane Database Syst Rev 4, 2007;17: Oct.
110. Fishel D, Buchner A, Hershkowith A, Kaffe I. *Roentgenologic study of the mental foramen*, Oral Surg Oral Med Oral Pathol 1976;41: 682.
111. FDA: Ensuring the safety of marketed medical devices: CDRH's Medical Device Postmarket Safety Program,

Telegram: @dental_k

www.konkur.in

January 18, 2006. www.fda.gov/cdrh/postmarket/mdpi-report.html.
112. FDA: www.fda.gov/Safety/MedWatch/default.htm.
113. FDA: The Future of Drug Safety—Promoting and Protecting the Public Health, FDA's Response to the Institute of Medicine's 2006 Report, January 2007. www.fda.gov/oc/reports/iom013007.pdf.
114. Food and Drug Administration Amendments Act of 2007 (FDAAA), Pub. Law 110-185.
115. FDA. *Sentinel Network to promote medical product safety: Public meeting*, Fed Regist 11, 2007;72:.
116. Forss H, Widstrom E. *Reasons for restorative therapy and the longevity of restorations in adults*, Acta Odontol Scand 2004;62: 82.
117. Franklin v. Albert, 380 Mass. 611, 411 N.E.2d 458 (1980).
117a *A fresh take on the adverse event landscape*, Clin Res 2, 2019;33: https://acrpnet.org/2019/02/12/a-fresh-take-on-the-adverse-event-landscape/. Accessed July 19, 2020.
118. Freudenheim M. To err is human: building safe hospitals 2004; New York Times April 6.
119. Frisk F, Hakenberg M, Ahlqwist M, et al: *Endodontic variables and coronary heart disease*, Acta Odontol Scand 2003;61: 257.
120. Gagliani M, Taschieri S, Molinari R. *Ultrasonic root end preparation: influence of cutting angle on the apical seal*, J Endod 1998;24: 726.
121. Gagnon MA, Lexchin J. *The cost of pushing pills: a new estimate of pharmaceutical promotion expenditures in the United States*, PloS Med 2008;5: e1.
122. Gamba TW. *Ethical Moment*, J Am Dent Assoc 2009;140: 107.

Telegram: @dental_k

www.konkur.in

123. Garofalo B, Fishberg ML. *Noncompete agreements*, Nat Law J 2000;B7: Jan 17.
124. Gavala S, Donta C, Tsiklakis K, et al: *Radiation dose reduction in direct digital panoramic radiography*, Eur J Radiol 2009;71: 42.
125. Geurtsen W, Leinenbach F, Krage T, et al: *IgE allergy due to formaldehyde paste during endodontic treatment. Apropos of 4 cases: 2 with anaphylactic shock and 2 with generalized urticaria*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 592.
126. Gibbs SJ. *Effective dose equivalent and effective dose: comparison for common projections in oral and maxillofacial radiology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90: 538.
127. Glickman SW, McHutchison JG, Peterson ED, et al: *Ethical and scientific implications of the globalization of clinical research*, N Engl J Med 2009;360: 816.
128. Glover v. DENTSPLY International, Los Angeles County Superior Court (California), Case No. BC279973.
129. Gluskin A, Lai G, Peters C, et al: *The double-edged sword of calcium hydroxide in endodontics*, JADA 2020;151: 317.
130. Gluskin AH, Ruddle CJ, Zinman EJ. *Thermal injury through Intraradicular heat transfer using ultrasonic devices: precautions and practical preventive strategies*, J Am Dent Assoc 2005;136: 1286.
131. Goldman RP. *Your duty to refer*, ADA Legal Adviser 1998;2: 6.
132. Goldman RP, Brown JL. The California clinician's legal handbook 1998; Law Offices of Ronald Goldman San Francisco.
133. Goldstein DB, Tate SK, Sisodiya SM. *Pharmacogenetics*

Telegram: @dental_k

www.konkur.in

goes genomic, Nat Rev Genet 2003;4: 937.
134. Goodacre CJ. *Five factors to be considered when restoring endodontically treated teeth*, Pract Proc Aesthet Dent 2004;16: 455.
135. Gonzalez-Martin M, Torres-Lageres D, Gutierrez-Perez J, et al: *Inferior alveolar nerve paresthesia after overfilling of endodontic sealer into the mandibular canal*, J Endod 2010;36: 1419.
136. Graskemper P. *The standard of care in dentistry: where did it come from? How has it evolved?*, J Am Dent Assoc 2004;135: 1449.
137. Greenberg BL, Kantor ML. *The clinician's guide to the literature*, J Am Dent Assoc 2009;140: 48.
138. Greenstein G, Tarnow D. *The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review*, J Periodontol 2006;77: 1933.
139. Grippo JO, Simring M, Schreiner S. *Attrition, abrasion, corrosion and abfraction revisited: a new perspective on tooth surface lesions*, J Am Dent Assoc 2004;135: 1109.
140. Grumbach K, Osmond D, Vranizan K, et al: *Primary care physicians' experience of financial incentives in managed care systems*, N Engl J Med 1999;341: 2008.
141. Gurdin v. Dongieux, 468 So.2d 1241 (La. Ct. App. 1985).
142. Hadley DL, Replogle KJ, Kirkam JC, et al: *A comparison of five radiographic systems to D-speed film in the detection of artificial bone lesions*, J Endod 2008;34: 1111.
143. Hadley J. *Dental radiology quality of care: the clinician makes the difference*, J Calif Dent Assoc 1995;23: 17.
144. Haïkel Y, Braun JJ, Zana H, et al: *Anaphylactic shock during endodontic treatment due to allergy to*

Telegram: @dental_k

www.konkur.in

formaldehyde in a root canal sealant, J Endod 2000;26: 529.

145. Hales v. Pittman, 118 Ariz. 305, 576 P.2d 493 (1978).
146. Haller CA, Benovitz NL. *Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids*, N Engl J Med 2000;343: 1833.
147. Hardison DC, Schnetzer T. *Using information technology to improve the quality and efficiency of clinical trial research in academic medical centers*, Qual Manage Health Care 1999;7: 37.
148. HITECH—Health Information Technology for Economic and Clinical Health, HIPAA Final Omnibus Rule, 2013.
149. The Health Insurance Portability and Accountability Act of 1996 ("HIPAA"), Pub. Law 104-191; 45 C.F.R. §§ 160, 164.
150. Helling v. Carey, 83 Wash.2d 514, 519 P.2d 981 (1974); but see Meeks v. Marx, 15 Wash. App. 571, 550 P.2d 1158 (1976).
151. Hoen MH, Braud JJ. *An endodontic perspective of contemporary infection control procedures*, Compendium 2004;25: 511.
152. Hoffman J, Shah ND, Vermeulen LC, et al: *Projecting future drug expenditures—2007.*, Am J Health Syst Pharm 2007;64: 298.
153. Horwitz RI, Singer BH, Makuch RW, et al: *Can treatment that is helpful on average be harmful to some patients? A study of the conflicting information needs of clinical inquiry and drug regulation*, J Clin Epidemiol 1996;49: 395- See also comment in *J Clin Epidemiol* 50:749, 1997.
154. Humphris GM, Morrison T, Lindsay SJ. *The modified*

Telegram: @dental_k

www.konkur.in

dental anxiety scale: validation and United Kingdom norms, Community Dent Health 1995;12: 143.
155. Hunt P, Gogamoiu D. *Evaluation of post and core systems*, J Esthet Dent 1996;8: 74.
156. Hussey PS, Wertheimer S, Mehrotra A. *The association between health care quality and cost: a systematic review*, Ann Intern Med 2013;158: 27.
157. Huumonen S, Kvist T, Grondahl K, et al: *Diagnostic value of computed tomography in retreatment of root fillings in maxillary molars*, Int Endod J 2006;39: 827.
158. Hyder AA, Wali SA, Khan AN, et al: *Ethical review of research: a perspective from developing country researches*, J Med Ethics 2004;30: 68.
159. Illinois Dental Practice Act, 225 ILCS 25/23(7)(2009).
160. In re: Silicone Breast Implant Litigation, Allegheny County Court of Common Pleas (Pennsylvania), Case No. GD94-14550, Order dated Feb 22, 2004.
161. Ingle J, Bakland L, Craig J. Endodontics ed 6 2008; BC Decker Philadelphia.
162. Irsheid v. Elbee Chemists and Available Products, Inc, San Francisco Superior Court (California), Case No. 908373, 1992.
163. Jacobson JJ, Walker C. *Antibiotic usage for prosthesis*, ADA Guide to Dental Therapeutics ed 3 2003; American Dental Association.
164. Jarvis J. *Doctor misconduct rarely reported, Ft*, Worth Star Telegram 2004; May 20.
165. Jeffcoat M. *If we don't do it, who will? Dentistry can't skirt medical complexities*, J Am Dent Assoc 2004;135: 1076.
166. Johnson W. Color atlas of endodontics 2002; Saunders Philadelphia.

Telegram: @dental_k

www.konkur.in

167. Joint Commission Universal Protocol Update 2009. http://www.jointcommission.org/standards_information/up. Aaccessed August 25, 2015.
168. Jones G, Behrents R, Bailey G. *Legal considerations for digitized images*, Gen Dent 1996;44: 242.
169. Jorge EG, Tanomaru-Filho M, Goncalves M, et al: *Detection of periapical lesion development by conventional radiography or computed tomography*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 56.
170. Jorgensen v. Beach "N" Bay Realty, Inc, 125 Cal.App.3d 155, 177 Cal.Rptr. 882 (1981).
171. Journal of the American Medical Association. *Physicians do not discuss CT risks with most patients*, JAMA Int Med 2013; March 4.
172. Judicial Council of California Civil Jury Instruction (CACI) 501 (2020).
173. Judicial Council of California Civil Jury Instruction (CACI) 533 (2020).
174. Judicial Council of California Civil Jury Instruction (CACI) 3709 (2020).
175. Juodzbalys G, Wang HL, Sabalys G. *Anatomy of mandibular vital structures. Part I: mandibular canal and inferior alveolar neurovascular bundle in relation with dental implantology*, J Oral Maxillofac Res 2010;1: 1.
176. Juodzbalys G, Wang HL, Sabalys G. *Anatomy of mandibular vital structures. Part II: mandibular incisive canal, mental foramen and associated neurovascular bundles in relation with dental implantology*, J Oral Maxillofac Res 2010;1: 1.
177. Kakoli P, Nandakumar R, Romberg E, et al: *The effect of age on bacterial penetration of radicular dentin*, J Endod

Telegram: @dental_k

www.konkur.in

2009;35: 78.
178. Kane B, Sands D. *Guidelines for clinical use of electronic mail with patients*, J Am Med Inform Assoc 1998;5: 104.
179. Kasegawa TK, Matthews M Jr. *Knowing when you don't know: the ethics of competency*, Prosthodont Insights 1998;10: 5.
180. Katakami K, Mishima A, Shiozaki K, et al: *Characteristics of accessory mental foramina observed on limited cone-beam computed tomography images*, J Endod 2008;34: 1441.
181. Katz J, Finnerup NB, Dworkin RH. *Clinical trial outcome in neuropathic pain*, Neurology 2008;70: 263.
182. Keeva S. *Law and sympathy*, ABA J 2004;90: 74.
183. Kersten DD, Mines P, Sweet M. *Use of the microscope in endodontics: results of a questionnaire*, J Endod 2008;34: 804.
184. Kessler D. *Introducing MED Watch. A new approach to reporting medication, devices, adverse effects and product problems*, JAMA 1993;269: 2765.
185. Klasser GD, Epstein JB. *Oral burning and burning mouth syndrome*, J Am Dent Assoc 2012;143: 1317.
186. Kleier DJ, Averbach RE, Mehdipour O. *The sodium hypochlorite accident: experience of diplomates of the American Board of Endodontics*, J Endod 2008;34: 1346.
187. Kohn LT, Corrigan JM, Donaldson MS. To err is human: building a safer health system 2000; National Academy Press Washington, DC.
188. Kohn WG, Collins AS, Cleveland JL, et al: *Centers for Disease Control and Prevention (CDC). Guidelines for infection control in dental health-care settings—2003*, MMWR Recomm Rep 2003;52: 1.
189. Krasny A, Krasny N, Rescher A. *Study of inferior dental*

Telegram: @dental_k

www.konkur.in

canal and its contents using high-resolution magnetic resonance imaging, Surg Radiol Anat 2012;34: 687.
190. Kuehn B. *Drug safety*, JAMA 2008;300: 2359.
191. Kuehn BM. *New e-prescribing guide unveiled*, JAMA 2008;300: 2235.
192. Kunisada M, Adachi A, Asano H, Horikawa T. *Anaphylaxis due to formaldehyde release from root-canal disinfectant*, Contact Dermatitis 2002;47: 215.
193. Langton L, Cohen T: Civil Bench and Jury Trials in State Courts, 2005. Bur Justice Stat (Oct. 2008).
194. Le Bell Y, Jamsa T, Korri S, et al: *Effect of artificial occlusal interferences depends on previous experience of temporomandibular disorders*, Acta Odontol Scand 2002;60: 219.
195. Lee v. Deubre, 362 S.W.2d 900 (Tex. 1962).
196. Lee K, Bacchetti P, Sim I. *Publication of clinical trials supporting successful new drug applications: a literature analysis*, PloS Med 2008;5: e191.
197. Lee M, Winkler J, Hartwell G, et al: *Current trends in endodontic practice: emergency treatments and technological armamentarium*, J Endod 2009;35: 35.
198. Lewis MH, Gohagan J, Merenstein D. *The locality rule and the physician's dilemma*, JAMA 2007;297: 2633.
199. Lewis R. *Patient-centered clinical consultations show mixed results*, Cochran Library 2012; Dec 21.
200. Loescher AR, Robinson PP. *The effect of surgical medications on peripheral nerve function*, Br J Maxillofac Surg 1998;36: 327.
201. Lofthag-Hansen S, Huumonen S, Grondahl K, et al: *Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 114.

Telegram: @dental_k

www.konkur.in

202. Lopez FU, Fachin EV, Camargo Fontanella VR, et al: *Apical transportation: a comparative evaluation of three root canal instrumentation techniques with three different apical diameters*, J Endod 2008;34: 1545.
203. Loubele M, Jacobs R, Maes F, et al: *Image quality vs radiation dose of four cone beam computed tomography scanners*, Dentomaxillofac Radiol 2008;37: 309.
204. Low K, Dula K, Burgin W, et al: *Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery*, J Endod 2008;34: 557.
205. Lowe J. *Government must do more to regulate dietary supplements*, San Francisco Daily Journal 2003; April 1.
206. Lurie A. *Panoramic imaging*, White S Pharoah MJ Oral radiology, principles and interpretation 2008; Mosby St. Louis 185.
207. Pogrel. *Broken Local Anesthetic Needles*, JADA 12, 2009;140: 1517-1522.
208. Masters B. *GlaxoSmith Kline agrees to post clinical data in settlement (Paxil safety and efficacy)*, Washington Post 2004; Aug 27.
209. Mathews SC, Pronovost PJ. *Physician autonomy and informed decision making: finding the balance for patient safety and quality*, JAMA 2008;300: 2913.
210. Mazor KM. *Patients' views on disclosure of medical errors*, Ann Intern Med 2004;40: 6.
211. McFall WT, Bader JD, Rozier RG, et al: *Presence of periodontal data in patient records of general practices*, J Periodontol 1988;59: 444.
212. McNamara v. Emmons, 36 Cal.App.2d 199, 97 P.2d 503 (1939).
213. Meeks v. Marx, 15 Wash.App. 571, 550 P.2d 1158

Telegram: @dental_k

www.konkur.in

(1976).
214. Mehdipour O, Kleier DJ, Averbach RE. *Anatomy of sodium hypochlorite accidents*, Compend Contin Educ Dent 2007;28: 544.
215. Mehra P, Clancy C, Wu J. *Formation of a facial hematoma during endodontic therapy*, J Am Dent Assoc 2000;131: 67.
216. Melpolder A, Widman A, Doroshaw J. The bitterest pill: how drug companies fail to protect women and how lawsuits save their lives 2008; Center for Justice and Democracy New York centerjd.org/archives/studies/BitterestPill(f).pdf. Accessed September 8, 2009.
217. Merriam Webster's collegiate dictionary ed 11 2003; PSI Associates Miami.
218. Michalowicz BS, Hodges JS, Pihlstrom BL. *Is change in probing depth a reliable predictor of change in clinical attachment loss?*, J Am Dent Assoc 2013;144: 171.
219. Michaud RA, Burgess J, Barfield RD, et al: *Volumetric expansion of gutta-percha in contact with eugenol*, J Endod 2008;34: 1528.
220. Miller CH. *Cleaning, sterilization and disinfection: basics of microbial killing for infection control*, J Am Dent Assoc 1993;124: 48.
221. Miller FH. *Health care information technology and informed consent: computer and doctor-patient relationship*, Indiana Law Rev 1998;31: 1019.
222. Mills S. *The dental unit waterline controversy: defusing the myths, defining the solutions*, J Am Dent Assoc 2000;131: 1427.
223. Mission Insurance Co. v. Worker's Comp. Appeals Board, 123 Cal.App.3d 211, 176 Ca. Rptr. 439 (1981).

Telegram: @dental_k

www.konkur.in

224. Mitka M. *Chelation therapy trials halted*, JAMA 2008;300: 2236.
225. Molander A, Warfvinge J, Reit C, et al: *Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical periodontitis: a randomized clinical trial*, J Endod 2007;33: 1145.
226. Moore PA. *Adverse drug interactions in dental practice: professional and educational implications*, J Am Dent Assoc 1999;130: 47.
227. Morris WO. Dental litigation ed 2 1977; Michie Charlottesville, VA.
228. Mounce R. *The biologic objectives of root canal therapy: meeting the standard*, Compendium 2004;25: 576.
229. Murray v. U.S, 329 F.2d 270 (4th Cir. 1964).
230. Nase JB. *The clinical operating microscope advantage in fixed prosthodontics*, Compendium 2004;25: 486.
231. National Center for State Courts: *Examining the work of state courts, 2007: a national perspective from the court statistics project*, Williamsburg, VA. http://contentdm.ncsconline.org/cdm4/item_viewer.php?CISOROOT5/ctadmin&CISOPTR51278&REC510. Accessed September 8, 2009.
232. Neade v. Portes, 710 N.E. 2d 418 (Ill. Ct. App. 1999).
233. Nealy v. U.S. Healthcare HMO, 711 N.E.2d 621 (N.Y. 1999).
234. Neaverth E. *Disabling complications following inadvertent overextension of a root canal filling material*, J Endod 1989;15: 135.
235. Nebeker JR, Barch P, Smore MH. *Clarifying adverse drug events: a clinician's guide to terminology, documentation and reporting*, Ann Intern Med 2004;140:

Telegram: @dental_k

www.konkur.in

795.
236. Nelson v. Knight, DDS. Supreme Court of Iowa. 2012. No. 11-1857.
237. Nixdorf DR, Moana-Filho EJ, Law AS, et al: *Frequency of nonodontogenic pain after endodontic therapy: a systematic review and meta-analysis*, J Endod 2010;36: 1494.
238. Nixdorf DR, Moana-Filho EJ, Law AS, et al: *Frequency of persistent tooth pain following root canal therapy: a systematic review and meta-analysis*, J Endod 2010;36: 224.
239. Oxman AD, Thomson MA, Davis DA, Haynes RB. *No magic bullets: a systematic review of 102 trials of interventions to improve professional practice*, Can Med Assoc J 1995;153: 423.
240. Packer M, Miller AB. *A symposium: can physicians always explain the results of clinical trials? A case study of amlodipine in heart failure*, Am J Cardiol 1999;1L:.
241. Papapanou PN. *The prevalence of periodontitis in the US: forget what you were told*, J Dent Res 2012;91: 907.
242. Paredes-Vieyra J, Enriquez FJ. *Success rate of single- versus two-visit root canal treatment of teeth with apical periodontitis: a randomized controlled trial*, J Endod 2012;38: 1164.
243. Parirokh M, Torabinejad M. *Minero trioxide aggregate: a comprehensive literature review—part III: clinical applications, drawbacks and mechanism of action*, J Endod 2010;36: 400.
244. Patient Protection and Affordable Care Act of 2010, 42 CFT. §410.15(a). 2010.
245. Patient Protection and Affordable Care Act. Congress HR 3590. 2010:111-148. 2010.

Telegram: @dental_k

www.konkur.in

246. Patient Safety Organization Are Step 1; Data Sharing is Step 2, *Virtual Mentor* 11:642, 2011.
247. Pear R: Spending rise for health care and prescription drugs slows, *New York Times*, A17, January 5, 2009.
248. Pegram v. Herdrich, 530 U.S. 211 (2000).
249. Peika M, Petscheit A. *Permanent mimic musculature and nerve damage caused by sodium hypochlorite: a case report*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: e80.
250. Penesis VA, Fitzgerald PI, Fayad MI, et al: *Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: a randomized controlled trial with one-year evaluation*, J Endod 2008;34: 251.
251. Peters JR, Philip G. *Doctors & Juries. U of Missouri, Columbia School of Law Legal Studies Research Paper No. 2006-33*, Mich Law Rev 2007;105: 1453-http://ssrn.com/abstract5929474. Accessed September 8, 2009.
252. Pierce EJ. *The development of anesthesia guidelines and standards*, Qual Rev Bull 1990;16: 61.
253. Pihlstrom BL. *Reporting clinical trials results*, J Am Dent Assoc 2009;140: 12.
254. Pogrel A. *Damage to the inferior alveolar nerve as the result of root canal therapy*, J Am Dent Assoc 2007;138: 65.
255. Pomeranza HH, Eidelman DL, Goldberg MG. *Treatment considerations of the middle mesial canal of mandibular first and second molars*, J Endod 1981;7: 565.
256. Psaty BM, Furberg CD, Ray WA, Weiss NS. *Potential for conflict of interest in the evaluation of suspected adverse drug reactions*, JAMA 2004;92: 2622.
257. Qato D, Pharm D, Alexander G, et al: *Use of prescription*

Telegram: @dental_k

www.konkur.in

and over-the-counter medication and dietary supplements among older adults in the United States, JAMA 2008;300: 2867.

258. Randall T. *Antibodies to silicone detected in patients with severe inflammatory reactions*, JAMA 1992;268: 1821.
259. Rawson K: *Running late: FDA review timelines lengthen for new product applications*, The RPM Report, Sept 2008.
260. Reeh ES, Messer HH. *Long-term paresthesia following inadvertent forcing of sodium hypochlorite through perforation in maxillary incisor*, Endod Dent Traumatol 1989;5: 200.
261. Renton T. *Prevention of iatrogenic inferior alveolar nerve injuries in relation to dental procedures*, Dent Update 2010;350:.
262. Renton T, Dawood A, Shah A, et al: *Post-implant neuropathy of the trigeminal nerve: a case series*, Br Dent J 2012;212: E17.
263. Renton T, Yilmaz Z. *Managing iatrogenic trigeminal nerve injury: a case series and review of the literature*, Int J Oral Maxillofac Surg 2012;41: 629.
264. Restatement (Second) of Agency §§ 228-237.
265. Restatement (Second) Torts, §§ 282, 283, 289, 299A (comment e).
266. Restatement (Second) of Torts § 402A. (See Fig. 10-18.).
267. Reynolds J. *Accrual of the cause of action for medical malpractice: Penrod v. Hoskinson*, Mont Law Rev 1977;38: 399.
268. Ring KC, Murray PE, Namerow KN, et al: *The comparison of the effect of endodontic irrigation on cell adherence to root canal dentin*, J Endod 2008;34: 1474.
269. Rising K, Bacchetti P, Bero L. *Reporting bias in drug*

Telegram: @dental_k

www.konkur.in

trials submitted to the Food and Drug Administration: review of publication and presentation, PloS Med 2008;5: e217.

270. Robernieks A. *Study looks at in-office errors by family physicians*, AMA Med News April 12, 2004; www.ama-assn.org/amednews. Accessed September 8, 2009.
271. Rohlin M, Kullendorff B, Ahlqwist M, et al: *Observer performance in the assessment of periapical pathology: a comparison of panoramic with periapical radiography*, Dentomaxillofac Radiol 1991;20: 127.
272. Romano M. *Survey shows error rate among otolaryngologists*, Modern Physician 2004; Aug 3.
273. Roter DL. *Patient participation in the patient-provider interactions: the effects of patient question asking on the quality of interaction, satisfaction, and compliance*, Health Educ Monogr 1977;50: 281.
274. Rowbotham M. *The impact of selective publication on clinical research in pain*, Pain 2008;140: 401.
275. Ruskin JD, Norton D, Karayazgan B, Amir J. *Failed root replacement*, J Oral Maxillofac Surg 2005;63: 829.
276. Sabala C, Powell S. *Sodium hypochlorite injection into periapical tissues*, J Endod 1989;15: 490.
277. Santana RR, Lozada J, Kleinman A, et al: *Accuracy of cone beam computerized tomography and a three-dimensional stereolithographic model in identifying the anterior loop of the mental nerve: a study on cadavers*, J Oral Implantol 2012;38: 668.
278. Sargenti Opposition Society (SOS). www.sargentipaste.org. Accessed 2013.
279. Saupe W, Gluskin A, Radke R. *A comparative study of fracture resistance between morphologic dowel and cores and resin-reinforced dowel system in the intraradicular*

Telegram: @dental_k

www.konkur.in

restoration of structurally compromised roots, Quintessence Int 1996;27: 483.

280. Scannapieco FA, Bush RB, Paju S. *Associations between periodontal disease and risk for atherosclerosis, cardiovascular disease, and stroke: a systematic review*, Ann Periodontol 2003;8: 38.
281. Scarano v. Schnoor, 158 Cal.App.2d 618, 323 P.2d 178 (1958); see also 61 Am.Jur.2d (rev.).
282. Schleyer T, Song M, Gilbert GH, et al: *Electronic dental record use and clinical information management patterns among practitioner-investigators in the Dental Practice-Based Research Network*, J Am Dent Assoc 2013;144: 49.
283. Schoenbaum SC, Bovbjerg RR. *Malpractice reforms must include steps to prevent medical injury*, Ann Intern Med 2004;140: 51.
284. Schofferman J, Banja J. *Conflicts of interest in pain medicine: practice patterns and relationships with industry*, Pain 2008;139: 494.
285. Schwartz SI, Shires GT, Spencer FC. Principles of surgery ed 7 1999; McGraw-Hill New York.
286. Scully C, Boyle P. *Reliability of a self-administered questionnaire for screening for medical problems in dentistry*, Community Dent Oral Epidemiol 1983;11: 105.
287. Sedghizadeh PP, Stanley K, Caligiuri M, et al: *Oral bisphosphonate use and the prevalence of osteonecrosis of the jaw*, J Am Dent Assoc 2009;140: 61.
288. Selbst A. *Understanding informed consent and its relationship to the incidence of adverse treatment events in conventional endodontic therapy*, J Endod 1990;16: 387.
289. Serhal C, Jacobs R, Flygare L, et al: *Perioperative validation of localisation of the mental foramen*, Dentomaxillofac Radiol 2002;31: 39.

Telegram: @dental_k

www.konkur.in

290. Sheeley v. Memorial Hospital, 710 A.2d 161 (R.I. 1998).
291. Shelter v. Rochelle, 2 Ariz.App. 358, P.2d 74 (1965).
292. Sherman P. Use of Federal Statute in State Negligence Pending Action. 13 Whittier Law Review 831 (1992).
293. Shipper G, Trope M: In vitro microbial leakage of the roots obtained with new and standard techniques. Paper presented at the AAE annual meeting, Anaheim, California, 2003.
294. Simpson v. Davis, 219 Kan. 584, 549 P.2d.950 (1976).
295. Sismondo S. *Ghost Management: how much of the medical literature is shaped behind the scenes by the pharmaceutical industry?*, PloS Med 2007;4: e86-www.plosmedicine.org/article/info%3Adoi%2F10.1371%2 Accessed September 9, 2009.
296. Small v. Wegner, 267 S.W.2d 26 (Mo. 1954).
297. Spellberg B, Bartlett JG, Gilvert DN. *The future of antibiotics and resistance*, N Engl J Med 2013;268: 299.
298. Sritharan A. *Discuss that the coronal seal is more important than the apical seal for endodontic success*, Aust Endod J 2002;28: 112.
299. Staal JB, de Bie RA, de Vet HC, et al: *Injection therapy for subacute and chronic low back pain: an updated Cochrane review*, Spine 2009;34: 49.
300. Stephenson J. *Illegal Internet drug sales*, JAMA 2008;300: 2717.
301. St-Hilaire H, Montazem AH, Diamond J. *Pneumomediastinum in orthognathic surgery*, J Oral Maxillofac Surg 2004;62: 892.
302. Studdert D, Mello MM, Gawande AA, et al: *Claims, errors, and compensation payments in medical malpractice litigation*, N Engl J Med 2006; May 11.
303. Studdert DM, Brennan TA. *The problems with punitive*

Telegram: @dental_k

www.konkur.in

damages in lawsuits against managed-care organizations, N Engl J Med 2000;342: 280.

304. Sundar RP, Becker MW, Bello NM, Bix L. *Quantifying age-related differences in information processing behaviors when viewing prescription drug labels*, PLoS One 2012;7: e38819.
305. Tavares PBL, Bonte E, Boukpessi T, et al: *Prevalence of apical periodontitis in root canal-treated teeth from a urban French population: influence of the quality of root canal fillings and coronal restorations*, J Endod 2009;35: 810.
306. Taylor P. *Preventing medication errors*, JAMA 2007;297: 1717.
307. Taylor P. *Implementing an electronic medical record system: successes, failures, lessons*, JAMA 2008;300: 1594.
308. Texas & Pacific Railway Co. v. Behymer, 189 U.S. 468 (1903).
309. Thilander B, Odman J, Groteborg K, Friberg B. *Osseointegrated implant in adolescents: an alternative in replacing missing teeth?*, Eur J Orthod 1994;16: 84.
310. Thomas MV, Beagle JR. *Evidence-based decision-making: implants versus natural teeth*, Dent Clin North Am 2006;50: 451.
311. Trapani v. Holzer, 158 Cal.App.2d 1, 321 P.2d 803 (1958).
312. Trust me—I'm a drug salesman, *Financial Times*, p 6, Oct 24, 2000.
313. Tsang A, Sweet D, Wood RE. *Potential for fraudulent use of dental radiography*, J Am Dent Assoc 1999;130: 1325.
314. Tulsa dentist found guilty of mail fraud, conspiracy,

Telegram: @dental_k

www.konkur.in

Tulsa World, p 1, Jan 12, 1993.
315. Tunkl v. The Regents of University of California, 60 Cal.2d 92, 383 P.2d 441 (1963).
316. Turner EH, Mathews AM, Linardatos E, et al: *Selective publication of antidepressant trials and its influence on apparent efficacy*, N Engl J Med 2008;358: 252.
317. Tyndall DA, Price JB, Tetradis S, et al: *Position statement of the American Academy of Oral and Maxillofacial Radiology on selection criteria for the use of radiology in dental implantology with emphasis on CBCT*, Oral Maxillofac Radiol 2012;113: 817.
318. UCSF News Office: UCSF-Harvard team publishes major HIV therapy study over objection of sponsor: compound shows no effect on slowing disease progression, Oct 31, 2000. http://news.ucsf.edu/releases/ucsf-harvard-team-publishes-major-hiv-therapy-study-over-objections-of-spon/. Accessed September 9, 2009.
319. US Centers for Disease Control and Prevention National Center for Health: May 2012. *Health*, United States, 2011.
320. U.S. Courts Library: Statistical Reports, Federal Judicial Caseload Statistics, Table C-3, 2003 and 2007.
321. U.S. Dept. of Health and Human Services Office of Inspector General. FDA's review process for new drug application: a management review, OEI-01-01-00590 2003; U.S. Dept. of Health and Human Services Washington, DC March.
322. U.S. ex rel. Pogue v. American Health Corp, Inc, 914 F. Supp. 1507 (M.D. Tenn. 1996).
323. Uniform Electronic Transactions Act (1999) (UETA), 15 U.S.C. §§ 7001 et seq.
324. Valdez v. Worth, D.D.S. 2011. Orange County Superior

Telegram: @dental_k

www.konkur.in

Court. No: 30-2010-00348533.
325. Vandi v. Permanente Medical Group, 7 Cal.App.4th 1064, 9 Cal.Rptr.2d 464 (1992).
326. Veronese v. Lucas Film. *Cal.* Ct. App.—December 10, 2012.
327. Walton RE. *News: antibiotics not always necessary*, J Am Dent Assoc 1999;130: 782.
328. Warning on popular heartburn drug, FDA says Propulsid linked to 70 deaths, should be a last resort, *San Francisco Chronicle*, p A3, Jan 25, 2000.
329. Washington v. Blampin, 226 Cal.App.2d 604, 38 Cal.Rptr. 235 (1964).
330. Weisgram v. Marley, 528 U.S. 440 (2000).
331. When statute of limitations commences to run against malpractice action against physician, surgeon, *dentist*, or similar practitioner, 80 A.L.R.2d 368, § 7b (1961).
332. Wickline v. State of California, 192 Cal. App.3d 1630, 239 Cal.Rptr. 810 (1986).
333. Willard v. Hagemeister 121 Cal.App.3d 406, 175 Cal.Rptr. 365 (1981).
334. Wilson W, Taubert K, Geqitz M, et al: *Prevention of infective endocarditis: guidelines from the American Heart Association: A guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group*, J Am Dent Assoc 2008;139: 3.
335. Witkin, Summary of California Law Agency and Employment, § 2-42 (10th ed. 2005).

Telegram: @dental_k

www.konkur.in

336. Witton R, Henthorn K, Ethunandan M, et al: *Neurological complications following extrusion of sodium hypochlorite solution during root canal treatment*, Int Endod J 2005;38: 84.
337. Wollschlaeger B. *Hooked: Ethics, the medical profession, and the pharmaceutical industry*, JAMA 2008;300: 2675.
338. Wood AJ. *Progress and deficiencies in the registration of clinical trials*, N Engl J Med 2009;360: 824.
339. Wood AJ, Stein CM, Woosley R. *Making medicines safer: the need for an independent drug safety board*, N Engl J Med 1998;339: 1851.
340. Wynn RL. *Internet web sites for drug information*, Gen Dent 1998;46: 12.
341. Wysowski DK. *Reports of esophageal cancer with oral bisphosphonate use*, N Engl J Med 2009;360: 89.
342. Yagiela J, Dowd F, Mariotti A, et al: Pharmacology and therapeutics for dentistry ed 6 2001; Mosby St. Louis.
343. Yatsushiro JD, Baumgartner JC, Tinkle JS. *Longitudinal study of microleakage of two root end filling materials using a fluid conductive system*, J Endod 1998;24: 716.
344. Zeider S, Ruttimann U, Webber R. *Efficacy in the assessment of intraosseous lesions of the face and jaws in asymptomatic patients*, Radiology 1987;162: 691.
345. Zhang D, Yin P, Freemantle N, et al: *An assessment of the quality of randomized controlled trials conducted in China*, Trials 2008;9: 22.
346. Zinman E. *Informed consent to periodontal surgery: advise before you incise*, J West Soc Periodontol 1976;24: 101.
347. Zitter JM: *Annotation*, standard of care owed to patient by medical specialist as determined by local, “like community,” state, national, or other standards. 18 A.L.R.

Telegram: @dental_k

www.konkur.in

4th 603, 608-619, 1982.

348. Zweigoron RT, Binns HJ, Tanz RR. *Unfilled prescription in pediatric primary care*, Pediatrics 2012;130: 620.

Telegram: @dental_k

www.konkur.in

Suggested readings

U.S.C 31 U.S.C. §§ 3729 et seq. See also 18 U.S.C. § 287 (false claims), 18 U.S.C. § 1001 (false statements), and 42 U.S.C. §§ 1301 et seq. (Health Insurance Portability and Accountability Act of 1996, Public Law 104-191.).

ADA Caries Risk assessment forms. *Assertion additional resources*, J Am Dent Assoc Jan 12, 2009; update.

American Dental Association (ADA): Principles of Ethics and Code of Professional Conduct § 5-H and 5-I, 2005.

Aqrabawi J. *Sealing ability of amalgam, Super EBA cement & MTA when used as retrograde filling materials*, Br Dent J 2000;11: 266.

Axelrod K. *Even Bush administration data discredits civil justice myths*, Trials 2009; January 11.

Azar FM, Pickering RM. Campbell's operative orthopaedics ed 9 1998;vol 2: Mosby St Louis.

Beacham L. *Damages caps benefit big business, not the little people*, San Francisco Daily Journal 2004; October 1.

California Business and Professions Code, § 2056.

California Dental Association Newsletter 24:12, 2000.

California Dental Association: Principle of Ethics, §§ 8 and 9.

California Evidence Code, § 669.

Carmichael Carmichael v. Reitz, 17 Cal.App.3d 958, 1971; Judicial Council of California Civil Jury Instruction (CACI) 502.

Carroll R. Risk management handbook for healthcare organizations 1997; American Hospital Publishing Chicago.

Degnam D, Merryfield D, Hulgrens R. *Reaching out to*

Telegram: @dental_k

www.konkur.in

clinicians: implementation of a computerized alert system, J Healthcare Qual 2004;26: 6.

Dieppe PA, Ebrahim S, Juni P. *Lessons from the withdrawal of rofecoxib*, Br Med J 2004;329: 867.

Dorn S, Gartner A. *Retrograde filling materials: a retrospective study of amalgam, EBA, and IRM*, J Endod 1990;16: 391.

Edwards BJ, Hellstein JW, Jacobsen PL, et al: *American Dental Association Council on Scientific Affairs Expert Panel on Bisphosphonate-Associated Osteonecrosis of the Jaw: updated recommendations for managing the care of patients receiving oral bisphosphonate therapy: an advisory statement from the American Dental Association Council on Scientific Affairs*, J Am Dent Assoc 2008;139: 1674.

Epstein JB, Chong S, Le ND. *A survey of antibiotic use in dentistry*, J Am Dent Assoc 2000;131: 1600.

2004. FDA acts to strengthen the safety program for marketed drugs [press release] Nov 5, 2004; Food and Drug Administration Rockville, MD www.fda.gov/bbs/topics/news/2004/NEW01131.htm. Accessed November 11, 2004.

Fontana M, Zero D. *Assessing patients caries risk*, J Am Dent Assoc 2006;137: 1231.

Fontanarosa PB, Renne D. *DeAngelisco Postmarketing Surveillance—lack of vigilance, lack of trust*, JAMA 2004;92: 2647.

Forkner-Dunn DJ. *Commentary: to err is human—but not in health care*, Am J Med Qual 2000;15: 263.

Freeman R. *Barriers to accessing dental care: patient factors*, Br Dent J 1999;187: 141.

Graber JJ. *Am I obligated to treat a patient who is HIV*

Telegram: @dental_k

www.konkur.in

positive?, J Am Dent Assoc 2004;135: 1172.
Gutman Gutman v. Klein, 2008 U.S. Dist. LEXIS 92398 (E.D.N.Y. Oct. 15, 2008).
Hellwege J. *Deaths from hospital errors double 1999 estimates*, Trial 2004;40: 82.
Holmes Holmes S. *Texaco settlement could lead to more lawsuits*, San Francisco Examiner 1996;A-8: November 17.
Hughes Hughes v. Blue Cross of Northern California, 215 Cal.App.3d 832, 1998.
Hupp JR, Ellis E, Tucker MR. Contemporary oral and maxillofacial surgery ed 5 2008; Mosby St. Louis.
Illinois Dental Board v. Feldman 2008.
Johnson Johnson v. Kokemoor, 545 N.W.2d 495, 1996.
Keeling D. *Malpractice claim prevention*, J Calif Dent Assoc 1975;3: 55.
Kleirer D, Averbach R. *Painful dysesthesia on the inferior alveolar nerve following use of a paraformaldehyde-containing root canal sealer*, Endod Dent Traumatol 1988;4: 46.
Kumar P, Subramanian K. *Demystifying the mist: sources of microbial bioload in dental aerosols*, J Periodontol 2020; https://doi.org/10.1002/JPER.20-0395. Accessed July 19, 2020.
Kutsch V, Milicich G, Domb W, et al: *How to integrate CAMBRA into private practice*, J Calif Dent Assoc 11, 2007;35: 778.
Lazarou J, Pomeranz BH, Corey PN. *Incidence of adverse drug reactions in hospitalized patients: a meta-analysis of prospective studies*, JAMA 1998;279: 1200.
Liewehr FR. *Primum non nocere*, Pract Proced Aesthet Dent 2006;18: 416.

Telegram: @dental_k

www.konkur.in

Lyon TC. *Quaternary ammonia compounds: should they be used for disinfection in the dental office?*, J Dist Columbia Dent Soc 1973;48: 10.

Magh Magh T II: Small cancer risk reported with bone-strengthening drugs, *Los Angeles Times*, January 2009. www.atsdr.cdc.gov/MHMI/mmg184.html.

Mathews SC, Pronovost PJ. *Physician autonomy and informed decision making: finding the balance for patient safety and quality*, JAMA 2008;300: 2913.

McGee McGee v. Cessna Aircraft Co, 139 Cal.App.3d 179, 1983; Medtronic v. Lohr, 116 S.Ct. 2240, 1996.

McNeill C. *Occlusion: what it is and what it is not*, J Calif Dent Assoc 2000;28: 748.

Medical Management for Calcium Hypochlorite and Sodium Hypochlorite. ATSDR, 2009. www.atsdr.cdc.gov/MHMI/mmg184.html.

Mitka M. *FDA Preemption*, JAMA 2008;300: 2599.

Musikant Musikant B: The economic truths of endodontics, *Dental Economics* 12, December 19, 2008. www.dentaleconomics.com.

Oberli K, Bornstein MM, von Arx T. *Periapical surgery and the maxillary sinus: parameters for clinical outcome*, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103: 848.

Occupational Safety and Health Administration (OSHA). Occupational Safety and Health Administration directive 1999; Washington DC www.OSHA.90V.

PPG Industries, Inc. v. Transamerica Insurance Co, 20 Cal.4th 310, 1999.

Stone Stone v. The Regents of the University of California 77 Cal.App.4th 736, 2000.

Piorkowski JD. *Bayer's response to "Potential for conflict of*

Telegram: @dental_k

www.konkur.in

interest in the evaluation of suspected adverse drug reactions: use of cerivastatin and risk of rhabdomyolysis,", JAMA 2004;292: 2655.

Porter R. *Attorneys press discovery effort in litigation over cholesterol drug*, Trial 2003;39: 12.

Reigel Reigel v. Medtronics, 2008 128 S.Ct 999.

Report on the performance of drug and biologics firms in conducting postmarketing commitment studies—Department of Health and Human Services. *notice of availability*, Fed Regist 2004;69: 50.

Robinson PP, Loescher AR, Yates JR, Smith KG. *Current management of damage to the inferior alveolar and lingual nerves as a result of removal of third molars*, Br J Maxillofac Surg 2004;42: 285.

Rosenbaum S, Frankford DM, Moore B. *Who should determine medical care necessity?*, N Engl J Med 1999;340: 229.

Seneris Seneris v. Haas, 45 Cal.2d 811, 1955; see also 53 A.L.R.2d 124.

Serper A, Ucer O, Onur R, Etikan I. *Comparative neurotoxic effects of root canal filling material on rat sciatic nerve*, J Endod 1998;24: 592.

Shapiro DW. *Do medical errors really kill 98,000 Americans a year?*, Prof Liability Reporter 2003;33: 3.

Simone Simone v. Sabo, 37 Cal.2d 253, 1951.

Simonsen R. *Greed and gravy train: is this success?*, J Esthet Dent 1999;11: 287.

Strom B. *Potential for conflict of interest in the evaluation of suspected adverse drug reactions: a counterpoint*, JAMA 2004;292: 2643.

Terezhalmy G, Bottomley W. *General legal aspects of diagnostic dental radiography*, Oral Surg 1979;48: 486.

Telegram: @dental_k

www.konkur.in

U.S. General Accounting Office. FDA drug review: post approval risks 1876-85. Publication GAO/PEMD 90-15 1990; U.S. General Accounting Office Washington DC 24.

US US v. Eli Lilly, 03-943 (E.D. Pa.) 2008.

Wall Street Journal, November 20, 1996:A3.

Waxman HA. *The danger of promoting as yet unproven drugs*, Wall St J 2002;30:.

Weichman J. *Malpractice prevention and defense*, J Calif Dent Assoc 1975;3: 58.

Weisman Weisman G. *Direct restorations*, Dent Products Rep Jan 2000;21:.

Wensing M, Van der Weijden T, Grol R. *Implementing guidelines and innovations in general practice: which interventions are effective?*, Br J Gen Pract 1998;48: 991.

Worthington P, Evans JR. Controversies in oral and maxillofacial surgery 1994; Saunders Philadelphia 284.

Young D, Featherstone J, Roth J, et al: *Consensus statement caries management by risk assessment: implementation guidelines to support oral health*, J Calif Dental Assoc 2007;35: 799.

Zhang C, Miller MR. *Excess length of stay, charges, and mortality attributable to medical injuries during hospitalization*, JAMA 2003;290: 1868.

---

[a]California Code of Regulations, Title 16, section 1017(n), CDC Guidelines for Infection Prevention

[b]Role of dental foci of infection in specific types of body diseases. JADA June 1951; 42(6): 655–86; Easlick K: An Evaluation of Dental Foci of Infection on Health. JADA June 1951 42:615–656, 691–697.

Telegram: @dental_k

www.konkur.in

[c]Internet Movie Talking Points; Root Canal Safety AAE Fact Sheet.

[d]Colorado Board of Dental Examiners. Case No.: DE-95-04(1996), In the matter of the disciplinary proceedings regarding the license to practice dentistry in the State of Colorado of Hal Huggins, DDS, license no.: 3057.

[e]California Civil Jury Instruction number 506.

[f]Hems, R., Gulabivala Y., Ready D., Spratt D.A., An in vitro evaluation of the ability of ozone to kill a strain of Enterococcus faecalis. International Endodontic Journal, 2005, 38, 22–29.

[g]Centers for Disease Control and Prevention, Questions & Answers: Dental Unit Water Quality. (http://www.cdc.gov/oralhealth/infectioncontrol/questions/dental-unit-water-quality.html)

American Dental Association: Oral Health Topics—Dental Unit Waterlines.

(http://www.ada.org/en/member-center/oral-health-topics/dental-unit-waterlines).

[h]Centers for Disease Control and Prevention. Summary of Infection Prevention Practices in Dental Settings Basic Expectations for Safe Care. Atlanta, GA: Centers for Disease Control and Prevention, US Dept. of Health and Human Services; October 2016: p 16.

[i]Kohn WG, Collins AS, Cleveland JL, et al: Centers for Disease Control and Prevention (CDC). Guidelines for infection control in dental health-care settings—2003, MMWR Recomm Rep 2003, 52:1, p 45.

[j]California Business and Professions Code section 1601.6(a).

Telegram: @dental_k

www.konkur.in

[k]Peralta G., Tobin-D'Angelo M, Parham A, et al. Notes from the Field, *Mycobacterium abscessus* Infections Among Patients of a Pediatric Dentistry Practice—Georgia, 2015. MMWR Morb Mortal Wkly Rep 2016;65: 355–356.

[l]Children's Dental Group Cases, Orange County Superior Court Judicial Council Coordination No.: JCCP 4917 (2017).

[m]Centers for Disease Control and Prevention. Summary of Infection Prevention Practices in Dental Settings Basic Expectations for Safe Care. Atlanta, GA: Centers for Disease Control and Prevention, US Dept. of Health and Human Services; October 2016: p 15.

[n]Kattan S., Lee S., Hersh E., Karabucak B. Do buffered local anesthetics provide more successful anesthesia than nonbuffered solutions in patients with pulpally involved teeth requiring dental therapy? JADA March 2019, Vol. 150(3):165–177.

[o]National Institute on Drug Abuse. Opioid Overdose Crisis. January 2019.

[p]Daniels, S.E., et al. "A randomized, five-parallel-group, placebo-controlled trial comparing the efficacy and tolerability of analgesic combinations including a novel single-tablet combination of ibuprofen/paracetamol for postoperative dental pain." Pain, 2011; 152: 632–642.

[q]Dowell D, Haegerich T, Chou R. CDC Guideline for Prescribing Opioids for Chronic Pain—United States, 2016. JAMA, 2016;315(15): 1624–1645.

[r]Lin Y, Rothen M, Milgrom P. Pharmacokinetics of 38% topical silver diamine fluoride in healthy adult volunteers. JADA, March 2019, Vol. 150(3):186–192.

Telegram: @dental_k

www.konkur.in

[s]Slayton R, et al. Evidence-based clinical practice guideline on nonrestorative treatments for carious lesions. JADA, October 2018, Vol. 149(10): 837–849.

[t]Garisto, et al. Occurrence of paresthesia after dental local administration in the U.S. JADA 2010: 141:836–844; Hillerup S, Jensen RH, Ersboll BK. Trigeminal nerve injury associated with injection of local anesthetics: Needle lesion or Neurotoxicity. JADA 2011: 142:531–539.

[u]Torabinejad M, Corr R, Handysides R, Shababang S. Outcomes of Nonsurgical Retreatment and Endodontic Surgery: A Systemic Review. JOE Vol 35(7): 930–937 July 2009.

[v]Becker, DE, Reed KL. Local anesthetic: Review of Pharmacological considerations. Anesth Prog. 2012 Summer 59(2) 90–102.

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28: Key principles of endodontic practice management

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CHAPTER OUTLINE

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Operating a successful endodontic practice requires both excellent clinical *and* management skills (Fig. 28.1). Endodontists are extremely well trained in their clinical specialty, but most do not receive extensive business training prior to becoming a practice owner. This scenario is beginning to change as more in the dental profession recognize the value of practice management skills and the role they play in an endodontist's ability to have a successful and rewarding career.

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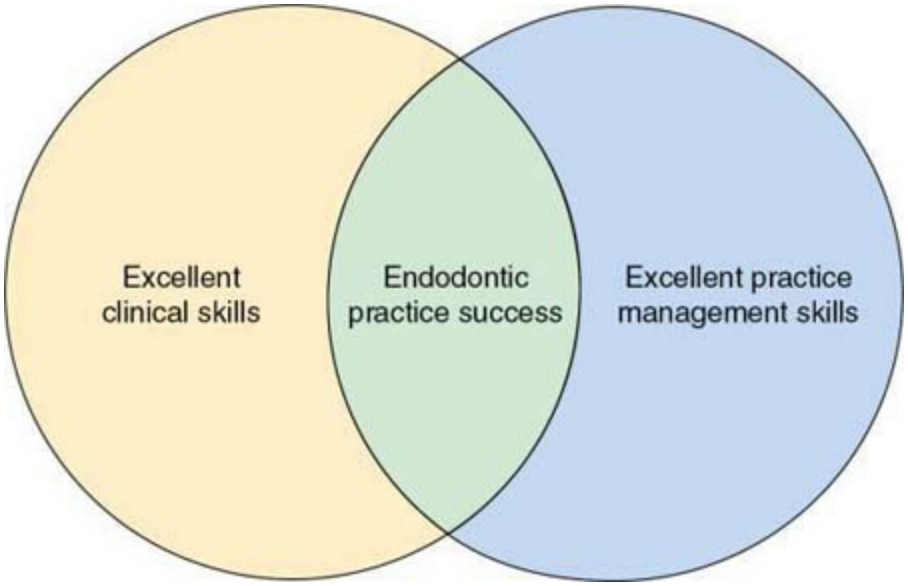

FIG. 28.1 The keys to achieving practice management success.

Venn diagram of practice management success shows two overlapping circles as excellent clinical skills (left) and excellent practice management skills (right), with common region labeled as endodontic practice success.

Endodontists who possess management skills are better positioned to handle the demands of operating an endodontic practice—a business that can generate significant revenue. Historically, there was a time when endodontists could enter practice without strong business skills and enjoy a long, prosperous career, but the business side of endodontics has become more complicated due to the difficult economy.

Young endodontists are leaving dental school and residency with far more debt than even a decade ago. The cost of purchasing or opening an endodontic practice continues to rise. In addition, endodontists face increased competition from other doctors who perform root canal treatments. The success of endodontic practices can also be affected by the development of new clinical techniques or services. For example, some doctors believe that dental implants represent a better treatment option than root canals, depending on the difficulty of saving the infected tooth. As dental implant procedures become easier and more common, this trend may continue to grow.

To meet these and other challenges, endodontists need to supplement their clinical expertise with excellent practice management skills. *Practice management* involves the use of documented systems to achieve superior

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business results. Effective practice management allows endodontists to provide optimal patient care while creating a highly productive, low-stress office environment. The design and implementation of effective practice management and marketing systems enables endodontists to operate their practices as successful businesses with steady growth and maximum efficiency. Endodontists who can combine excellent clinical skills with strong practice management skills will experience greater practice success (see Fig. 28.1).

Endodontic management systems

Written, documented systems are the basic foundation for all successful operations within an endodontic practice. Step-by-step, reproducible systems enable a practice to consistently operate and be successful as both a provider of patient care and a business enterprise. Documented systems reduce unnecessary stress and allow endodontists to remain chairside so they can focus on providing exceptional quality of care. As each system is established, every step in that system must be clearly documented and then implemented. Many endodontists fail to establish efficient systems in the early years of a new practice, and inefficient systems can hinder the practice's growth, increasing stress for the doctor and team.

Endodontists should begin their careers by developing and implementing effective practice systems. This way, when an endodontic practice begins to grow, the office has a solid management foundation in place to handle the growth with low stress as well as to reach more challenging goals. If the practice waits until it becomes more established, there is less time to design effective systems, and the endodontist often becomes less motivated to do so owing to an increasingly busy office and other responsibilities in life.

Systems design

New endodontic practice owners have an excellent opportunity to design systems because the office is nowhere near its capacity. This type of foresight sets the stage for practice growth in the years to come. However, systems should be evaluated and updated throughout an endodontist's career. It will be necessary to replace most major endodontic practice systems on a 5-year

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cycle to maintain growth and profitability. The reason is that practices regularly outgrow their systems as a result of changes in staff, equipment, technologies, referral relationships, and the economy.

Immersed in the day-to-day pressures of running the practice, evaluating systems can be difficult. Therefore endodontic practices are well advised to seek outside experts to help them determine the "state of the practice." An in-office practice analysis should include the following:

- First-hand observations of the team and practice operations
- Interviews with the doctor and key team members
- In-depth assessment of all key production indicators (KPIs)
- Thorough evaluation of all practice systems, physical layout, and patient flow

Through such careful analysis, it is possible to accurately determine the effectiveness of practice systems that are in place.

Systems should be designed based on the specific characteristics of each practice. Areas to be considered include the following:

- Days of operation
- Hours of operation
- Number of staff
- Number of patients
- Number of true emergencies
- Average daily patient flow
- Types of procedures offered
- Number of chairs

Effective management systems can make the difference between a successful endo practice and a *highly* successful endodontic practice. Many endodontists cannot fathom how they could see any more patients, produce any more revenue, or work any harder. Yet, there exist similar practices that have much higher production in the same number of days per week with the same number of staff. It is always interesting to reflect on why one endodontic practice can be so much more financially successful than another practice in similar circumstances.

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Assuming a sufficient patient flow, the answer always comes down to management systems. It is not about working harder or faster, but it is about creating systems that allow the practice to operate at a specific production and profitability level in a low-stress environment. Key systems and principles for effective endodontic management and marketing include these:

1. Vision
2. Mission
3. Goal setting
4. Target setting
5. Key production indicators
6. Staff training and development
7. Scheduling
8. Customer service

Management

Vision

The first question endodontists should answer before opening or purchasing an endodontic practice is, “What kind of endodontic practice do I want to have?” This question requires the endodontist to take some time and identify a vision, which is a description of where the doctor wants the practice to be in 3 years. Whereas some vision statements may reflect a longer period, this shorter time frame works well for most endodontists because it is difficult to predict changes in dentistry, the economy, and other events more than 3 years into the future. In addition, after 3 years, it is time for the doctor to review what has been accomplished and possibly establish a new vision for the practice.

To create a vision statement, the following questions should be answered:

- What location is most suitable for the practice?
- What size should the practice be in 3 years?
- Does the doctor want to be in solo practice or multidoctor practice?
- What working days and hours will be satisfactory for the practice?
- What services will the practice provide?

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- Is the intent to have only one practice or to have more than one location?

These are just examples of questions that can stimulate the design of a vision statement.

A vision statement clearly articulates where the practice is going. The length of a vision statement can vary between a short, single paragraph and several paragraphs. It is probably counterproductive to have a multipage vision statement; it will be difficult for both the endodontist and the staff to fully grasp such a lengthy and complex vision.

The vision guides the long-term development and growth of the endo practice. Once the vision statement has been created, it should be shared with the staff on a regular basis. Good leaders always communicate their vision to their staff. When team members understand where the practice is headed, they are empowered to help the endodontist achieve his or her vision. Sharing the vision inspires commitment and participation in achieving the vision. The following are two examples of vision statements.

Short vision statement

In 3 years, we will be the leading endodontic practice in the area. We will be known for exceptional quality of care and outstanding customer service. We will grow by 15% per year. Our staff is motivated and committed to continuously improving our management systems and to a lifelong learning process. We strive to meet or exceed our goals in a low-stress work environment.

Long vision statement

In 3 years, we will be known in the community for being the most comprehensive and skilled endodontic practice in the area, offering our patients an atmosphere of compassion and comfort.

Our excellent management skills create an environment that is low-stress and productive. We consistently meet our production goals without creating a rushed atmosphere, and our days are fulfilling.

We provide exceptional customer service that consistently generates spontaneous, positive comments. Team members have a clear understanding

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of their roles and responsibilities. We have developed practice systems that foster a sense of fulfillment and accomplishment for all. In year 3, we will engage an associate doctor.

Mission

Once the vision statement has been created, the next step is the development of a mission statement.

Whereas the vision statement is about where the practice will be in the future, the mission statement is focused on where the practice is today.

Once again, a strong leader has a clear practice mission and shares it with the team. Because of the similar services provided by endodontic practices, many mission statements resemble one another—they talk about quality, patient care, practice services, and customer satisfaction. It is not important to have a mission that differentiates one practice from another, but it *is* essential to have a mission that reflects the doctor's true vision. Here is an example of a mission statement:

> *Our endodontic practice is dedicated to delivering quality care to meet the endodontic needs of our patients and referring doctors. We provide exceptional clinical care and customer service in a comfortable, relaxed environment.*

One way to keep the mission statement on everyone's mind is to display it prominently in several places throughout the office, as well as on the practice's website. The most powerful reason for displaying the mission statement is to reinforce to the team that the mission is a living document that guides the practice. An added benefit of displaying the mission statement is the opportunity to let patients know that they are important to the practice.

The endodontist should refer to the mission statement on a regular basis. Reinforcing the mission during meetings helps staff members focus on why the practice exists and the difference the team is trying to make in the lives of patients.

Goal setting

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Goal setting represents one of the most important business activities for endodontists. It is critical to achieving success in a doctor's professional and personal life. Once a vision and mission are established, annual goals are the method to make the vision and mission realities. Goal setting is a step-by-step methodology that leads to the achievement of the overall vision. To be effective, a goal must be

1. Written
2. Deadline driven
3. Measurable

Goals that lack these three characteristics cannot be considered goals and are much less likely to be achieved. Be sure that all of the goals are aligned with the practice's vision and mission to achieve the desired results.

The pace of today's endodontic practice forces many doctors to focus on day-to-day operations. In doing so, they cannot see the big picture until many years have gone by. Goal setting and regular goal reviews can help endodontists manage their practice—instead of being managed by it.

Far more than just a motivational concept, goal setting acts as a timeline, benchmark, and directional process to achieve the vision and mission. A vision or mission without specific goals is merely an idea. Endodontists who set goals are much more likely to succeed.

Target setting

Targets measure whether you are accomplishing your short-term and long-term goals. Each year, endodontists need to set 12 to 15 specific targets to be achieved for the next year. This helps motivate the team to reach goals and drive practice growth. Targets should be reviewed on a weekly basis. For example, if the practice is only collecting 85% of its fees, then the office needs to implement more effective financial procedures to reach the 99% target (if that is the target that has been set).

Sample endodontic targets to achieve include the following:

- *Increase practice production by 15% in the next 12 months.*
- *Ensure that 98% of all nonemergency patients are scheduled at all*

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times.
- *Collect 99% of all money owed to the practice.*
- *Maintain a less than 1% no-show/last-minute cancellation rate.*

Key production indicators

Without data, an endodontic practice cannot be well managed. To obtain a clear picture of current and potential practice performance, practice goals must be monitored and measured. Key production indicators (KPIs) provide an accurate assessment of the practice's most critical functions, including the following:

- Production
- Collections
- Profit
- Overhead
- New patients
- Accounts receivable
- Number of re-treats versus initial root canal treatments
- Insurance percentages
- Production per hour

These KPIs offer endodontists a starting point for analyzing their practices. Once the information has been collected, weekly measurement and analysis are necessary. Although this may sound arduous, a weekly review should take only a few minutes. Such frequent analyses can bring substantial benefits to the practice.

Evaluating KPIs every week allows endodontists to rapidly identify trends or performance deviations that may be difficult to discern otherwise. With accurate practice data, endodontists are then able to take appropriate actions.

Staff training and development

Establishing practice systems will only be effective if team members understand their roles in making those systems work. Training the endodontic team begins with the development of job descriptions that list the skills, responsibilities, and accountabilities for each staff position.

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Bear in mind that the majority of staff will be hired without previous endodontic or dental experience and must be trained on the job. Training is specific to each position so that all team members can perform their duties as independently and effectively as possible. During training, it is critical that practices document training policies and procedures so they can be clearly communicated to new staff and regularly reinforced with established team members.

One frequent problem is that many staff members fail to fully understand their job responsibilities and are not held accountable for their performance. As a result, the endodontist will continue to handle numerous responsibilities that should be performed by other team members.

Although this type of behavior may be common in the early stages of an endo practice, endodontists who become effective leaders learn how to delegate appropriate tasks to the team. The goal for all endodontists should be to spend 98% of their workday involved in direct patient care or communication with referring doctors. To reach this goal, it is recommended that endodontists make a list of all activities they perform and which of those responsibilities can be assigned to the staff. This exercise will enable the endodontist to clearly identify which activities should be handled by the doctor only and which should be delegated to the team.

Delegation empowers the team to take ownership of their roles in contributing to practice success. The concept involves giving responsibilities to staff members so that endodontists can concentrate on what they do best. Without delegation, endodontists often carry out numerous responsibilities that can easily be performed by others. To spend more time in direct patient care, endodontists must delegate appropriate responsibilities to specific staff members. In addition, the team should be cross-trained on systems to cover for any illnesses, vacations, or turnover.

Once all team members have achieved a high level of expertise and independence, the endodontic practice can operate at peak performance. Efficiency, professional satisfaction, and quality of care steadily climb to maximum levels. At this stage, endodontists trust their teams to effectively perform their duties with minimal oversight. Best of all, doctors can focus on providing exceptional patient care without distractions and unnecessary stress.

In highly successful endodontic practices, the doctor focuses 98% of

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working hours strictly on patient care, interdisciplinary treatment, and referring-doctor communication. These practices will typically reach 90% to 100% of their production potential.

Scheduling

Making changes in the area of practice management should begin with a thorough evaluation of the schedule because of its impact on all other major systems. If the schedule is integrated properly with all other endodontic management systems, the practice has the best opportunity to achieve the endodontist's targets.

Schedules are created to achieve specific practice production in a low-stress environment. This means that an endodontic practice must establish a target for annual production and design the schedule accordingly. Each year, endodontic practices should calculate the desired level of growth and create a schedule that allows for that growth. The greatest challenge to increasing production is scheduling inefficiencies that make it all but impossible for the practice to grow.

The following principles are essential when designing the ideal schedule for any endo office.

Schedule for production

Design the schedule to achieve a specific production target. Endodontic practices are typically capable of growing between 5% and 15% annually. Less than 4% growth represents moving onto a plateau. There are endodontic practices that can grow by as much as 20% to 25% in a single year as optimal systems are implemented.

Scheduling is a quantitative and mathematical activity because time is quantitative and therefore measurable. The schedule should answer questions like these: *How many root canals will we need to perform this year? Will those numbers allow for the production goal to be achieved?* Benchmark these production goals against the hours and days per week the practice is available for patient care and adjust the schedule accordingly.

Set the daily production target

Setting the daily goal is a reflection of the annual production target. To

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determine the daily goal, simply take the annual production goal and divide it by the number of days the practice will operate that year. Then build the schedule around this key number. Front desk personnel should become aware that the top practice management priority is to achieve the daily goal. By implementing an excellent scheduling system, they will have a far greater chance of successfully accomplishing this task.

Perform procedural time studies

Many endodontic practices are unrealistic about how much time they actually need for procedures. One common example is for root canals to simply be scheduled for one set length of time, regardless of the level of complexity involved. Performing “procedural time studies” is recommended to determine the exact amount of time needed for each appointment.

During procedural time studies, each type of treatment performed by the office should be measured approximately 10 times to develop an average procedure time. Once these studies are performed and the schedule is updated, most endodontists will usually be able to increase their endodontic workload by using time more efficiently.

Young endodontists should perform procedural time studies once a year for the first 5 years they are in practice. After that, studies can be conducted at less frequent intervals (every 3 to 5 years) to evaluate whether the endodontist has become faster or more efficient in terms of experience, technique, or the use of new technologies in the practice. Procedural time studies play a critical role in building an effective and accurate schedule, which directly affects the success of the endodontic practice.

Create the ideal day

The schedule should be designed to perform larger and more complex appointments in the morning whenever possible. This can be extremely challenging, owing to the number of emergencies endodontists see during a normal day. By scheduling larger procedures in the morning, practices can take advantage of the high energy level for both the endodontist and the team earlier in the day. Of course, emergencies will continue to occur, but effectively managing other appointments will better position the practice to handle end-of-day emergencies. Other recommendations for creating the

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ideal day include the following:

- Handle less complex cases in the early afternoon.
- Perform consults and diagnoses in the midafternoon.
- Schedule all minor procedures, such as posttreatment evaluations, for the end of the day.

Basically, this means that the more difficult or larger cases will occur in the morning when the doctor and team are fresh. As the day goes forward, the doctor and staff have the opportunity to work on less complex cases, such as second visits and patient consultations, with all minor procedures taking place at the end of the day. A highly organized schedule will allow for increased productivity and higher profitability while decreasing stress for the endodontist and the team throughout the day.

Manage emergencies

No discussion of endodontic scheduling would be complete without focusing on emergency management. Obviously, emergency procedures are a frequent occurrence in endodontic practices. Managing emergencies well requires a combination of referring-doctor management, setting patient expectations, and effective scheduling. The result will be a highly productive, satisfying professional environment for any endodontic practice.

The first step in managing emergencies is to develop a schedule that allows some flexibility. It does not benefit the practice to create a schedule that is 100% maximized without accounting for emergencies. Instead, analyze how many genuine emergencies occur on an average day, and then leave time for that number of emergencies in the schedule. Many high-level endodontic practices will schedule only 80% of their day, leaving time slots for emergencies. The key is to not ignore the time slots and invite every emergency patient to visit the office immediately, but to manage each situation appropriately.

For example, a patient who is in mild discomfort calls at 10 a.m. The practice does not have an opening until noon. If the patient is allowed to come in immediately, the office feels a need to work the patient into the schedule, not only causing stress for the doctor and team but possibly forcing already-scheduled patients to wait longer than expected. If the practice

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instead schedules the patient in an emergency time slot, there is a much greater opportunity to maintain excellent flow for the day and create a positive customer service experience for every patient. The main point is to provide optimal care and exceptional customer service without disrupting the daily schedule.

The keys to effective emergency management are as follows:

- Evaluate each call from a patient or referring office to determine the level of emergency. Many calls are not true emergencies, and these patients do not need to be seen immediately. Scripting and training can help staff members elicit specific information from patients about the level of discomfort (mild tenderness versus extreme pain) while providing excellent customer service.
- If it is a true emergency, bring the patient in immediately, but still evaluate the schedule to facilitate the overall process. Highly systemized endodontic practices use trained staff to evaluate emergency situations as much as possible, including preliminary assessment and x-rays.
- If the emergency call does not constitute a true emergency, it is important to use effective scripting to move the patient into an emergency time slot that will prevent the appointment from disrupting the entire schedule. This approach can be used with either a referring office or a patient.
- If the practice notices ongoing disruptions from emergencies, it should reevaluate the emergency policy and scripting to create a more effective methodology. Many endodontic practices tell every emergency patient to come in immediately. The problem arises from mistaking high-level customer service with providing immediate treatment. It is much more effective to manage the emergency patient into a time slot reserved for emergencies, unless the patient is experiencing an endodontic problem that necessitates immediate treatment. Staff training is critical for effectively managing emergencies while providing quality customer service.

Scheduling is the single most important system because it integrates with all other systems. When an effective scheduling system has been established

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and properly implemented, the practice will immediately see an increase in productivity and a decrease in stress. Most stress in endodontic practices is based on poor scheduling methods that overwhelm the doctor and team and lower customer service. Even though the endodontist and team are actually working harder, profitability will be lower. An efficient schedule enables the endodontic practice to provide a high level of care and customer service to all patients.

Customer service

Customer service today means much more than just “being nice” to patients. It is a system focused on managing the patient experience from the initial referral to treatment to postoperative care. Outstanding customer service is critical to exceeding patient expectations and building strong relationships with referring doctors.

The majority of endodontic patients are referred to the practice by general dentists. The growth of the endodontic practice therefore depends on the satisfaction and confidence of referring doctors. General dentists want to know that their patients’ expectations, as well as their own, are being met. When patients receive excellent clinical treatment and superior customer service, they are likely to provide positive feedback when returning to their general dentist. Positive feedback from patients reinforces the dentist’s confidence in the endodontist and leads to continuing referrals.

Developing a customer service system is more complex than simply telling the staff to be nice to patients and referral sources. A customer service system requires that patients consistently receive the same level of responsiveness, caring, and concern from every member of the endodontic practice. Consistency and quality are at the core of a practice’s customer service system.

Although the patient’s time in the endodontic practice may be limited, the endodontist must realize that the patient will be returning to the referral source and sharing his or her experience with the referring doctor or the clinician’s staff. The patient will also share information about the experience with family, friends, and others. Negative word-of-mouth descriptions about the office will obviously hurt the endodontic practice’s reputation within the community.

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Patients want to believe they are the practice's *only* priority when they come in for their appointments. Endodontic practices with excellent customer service strive to meet this expectation. If a practice fails to return phone calls quickly, is running late when patients arrive, or does not seem prepared for appointments, patient confidence and trust disappears, as does the patient's perception of quality care.

Endodontic practices should manage expectations by making everything about the patient's treatment plan value based. Do patients fully understand the value of their treatment plans? Do they know exactly what the treatment entails, what the fees are, and why the treatment must be done? Does the patient know the projected outcome? All these questions must be satisfactorily answered before treatment starts.

Endodontic practices have an opportunity to exceed expectations when the following occurs:

- There is complete understanding about why, how, and when endodontic treatment will take place
- Patients have realistic expectations of outcomes
- Patients and referring doctors receive accurate and timely information about the treatment process
- The practice has timely follow-up posttreatment communication with patients

Marketing

Endodontic marketing

The science of endodontic marketing has evolved considerably. For many years, endodontic practices were in such high demand that referral marketing was relatively unimportant. In fact, endodontists felt that they couldn't handle any additional patients.

Today's supply and demand scenario has changed dramatically with the great expansion of different sources (read: competition) for endodontic procedures. Factors that have added to the competitive challenges of endodontists include:

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- *General dentists.* General dentists are performing more root canals than ever. The 2008 recession slowed general practice production leaving general dentists scrambling to make up for lost revenue. According to the Levin Group Data Center, the first specialty service that general dentists typically expand in their practices during a downturn is root canals. All indications are that this trend will continue.
- *Dental support organizations (DSOs).* Corporate dentistry also benefits from offering root canal treatment. One of today's largest DSOs has an in-house training program for the GPs in their practices to learn and perform endodontics.
- *Dental implants.* Dental implants have changed the referral pattern for endodontic procedures. Many implant procedures are no longer being referred out and are simply being treatment-planned for extraction and root canal.
- *Endodontic retreatment.* Retreatment by endodontists is on the decline. When general dentists perform root canals that fail years later, they do not necessarily refer treatment to an endodontist. More GPs are opting to perform implant procedures in-house.

All of these factors are changing the referral patterns of endodontics for the foreseeable future and causing practices to rethink their referral marketing strategy.

Endodontic referral marketing

Emerging competitive factors are having a profound effect on how endodontic practices approach referral marketing. While referral marketing (Fig. 28.2) may have been an afterthought in previous years, it's now a crucial part of endodontic practice success that can be described in two highly effective parts—quality and quantity.

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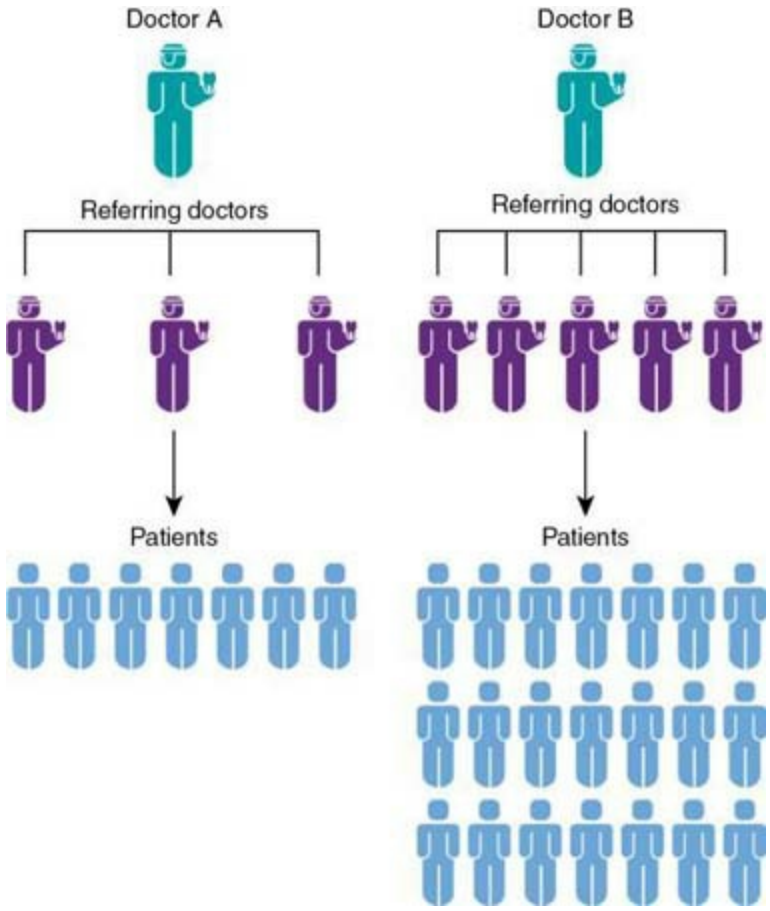

FIG. 28.2 The impact of referral marketing. *Doctor A* does not have a formal referral marketing program in place. *Doctor B* does have a formal referral marketing program in place. The results are dramatic. *Doctor B* has many more referring doctors, who refer more patients for treatment.

Doctor A leads to three referring doctors, which further leads to seven patients. Doctor B leads to five referring doctors, which further leads to twenty one patients.

Quality refers to the type of strategies that are employed in referral marketing such as education, events, branding, and relationship-building. The quantity of strategies is the part that is far less understood. Practices often employ two or three strategies, believing that will be enough. However, it has been clearly demonstrated that a minimum of 15 different referral marketing strategies targeted to referring doctors should be performed on an on-going basis. The key is to select the proper 15 (or more) that represents the quality and quantity necessary for the referral marketing program to be successful.

This outreach is best performed by a marketing coordinator who can carry

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out 95% of the referral marketing program activities, allowing the endodontist to continue to focus heavily on practice production. These activities can include hosting seminars, visiting referring offices, or creating fun contests and events for referring doctors and staff. A strong referral marketing program of this nature run by a focused marketing coordinator will increase referrals and maintain a strong referral base. Typically this will be a part-time position, typically 16 to 20 hours a week for a one- to two-doctor practice or 22 to 24 hours for a three-doctor practice. Practices with four more doctors will require a full-time marketing coordinator.

An endodontic referral marketing program should focus on four key areas —referring doctors, patients, social media, and the community. These areas work great together in order to create a synergistic affect where the whole is greater than the sum of the parts.

Social media

Social media has become a major marketing vehicle if not *the* major marketing vehicle in the world today. Unfortunately, the benefits of social media are fewer for endodontic practices as most endodontic patients are referred by other doctors and do not directly seek endodontic services on their own. However, social media can play a major role in an endodontic referral marketing program by helping practices connect to the community, build their brand, and create name recognition so that their practices will be top of mind with referring doctors that have patients who need endodontic services or individuals seeking endodontic treatment.

While it may be tempting, the social media component of an endodontist referral marketing program should not focus on constantly posting information, pictures, or videos. In fact, that may be counterproductive. Instead, having a small but steady flow of content related to endodontics and dentistry will effectively create name recognition and branding.

Transitions

In the past, endodontic practices typically reached a certain production level and remained at that level throughout the endodontist’s career. When the time came for the sale of the practice, the endodontist could count on a strong valuation for the sale.

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Today is a very different era. When endodontic practices reach a point of sale or transition, the owners are finding that their valuation is not as strong as they had hoped. This is typically due to having fewer referrals or lower production than in the past due—competitive factors outlined earlier in this chapter. Valuations are typically based on 3 years of information demonstrating whether or not the practice has had consistent growth and performance. For example, a practice that is strong in production but demonstrating a declining number of referrals will have a lower valuation.

One of the most important factors in preparing for a practice transition or sale is to keep the practice strong. As they edge toward retirement, many doctors tend to lose focus. And while it may feel good to cut back, they will be in for a lower practice valuation when the time comes for a sale. Keeping a practice strong in the last few years will allow for an excellent valuation and a higher sale price, which contributes to the endodontist's retirement funding. If the practice has demonstrated weaknesses or decline in the last 3 years, the endodontist will either have to accept a lower sale price or delay selling the practice if financially necessary.

A strong marketing program that aids in increasing and maintaining patient and referring doctor relationships will add to the value of an endodontic practice. Unlike other businesses that have ongoing customers when the business is sold, an endodontic practice is often dependent on the endodontist and his or her relationships with referring offices. When selling a practice, it is often prudent to offer to remain with the practice for 6 months or more to help ensure a smooth transition of referral sources to the new doctor. The more time the selling endodontist stays to endorse and help the buying doctor transition, the better. If possible, it makes sense to hire an associate who can buy in over a number of years and develop relationships with the referring doctors.

Leadership

Leaders possess many skills, qualities, and attributes that are the result of learning, experience, and self-motivation. Many people have great ideas or vision, but true leaders are able to implement those ideas or carry out the steps necessary to achieve predetermined goals. They have the ability to inspire others to do their best. The most successful endodontic practices are

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guided by strong leaders.

Becoming an excellent leader starts with the desire to have an outstanding practice. Most endodontists are highly motivated to be excellent clinicians. Endodontic continuing education is focused mainly on clinical topics, which is appropriate, given that doctors have an obligation to provide the best possible care to patients. This makes it even more important for endodontists to seek out opportunities to develop leadership, business management, and marketing skills.

The endodontic leader

As practice owners, endodontists are automatically viewed as leaders by team members. The more leadership skills endodontists master, the better leaders they become. Though not an exhaustive list, the following leadership skills are critical for endodontists.

Interpersonal communication

Interpersonal skills are the foundation of leadership. Endodontists work with a small group of people in a fairly limited space. Good interpersonal skills are essential for a smoothly functioning office environment. In business, there are many successful, “tough” bosses. Even though they may be disliked, they are respected. However, being tough or inflexible is not always the best strategy, especially in dentistry. Few endodontists can be tough leaders in a very small office environment and still expect to have happy team members and a successful practice.

The best practice leaders can motivate their staff, accomplish goals, and earn the respect of team members. Regular morning meetings and monthly staff meetings provide excellent opportunities to communicate with the team, increase awareness of practice challenges and goals, and build stronger interpersonal relationships with team members.

Decision making

As the CEOs of their practices, endodontists must make business decisions. There are many people who do not progress in business simply because they vacillate between two choices and fail to make a decision in a timely manner. No matter what the issue, excellent leaders make decisions and trust their

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instincts, based on analysis, education, and experience. Why? Strong leaders believe in themselves and their knowledge. They are confident that they have the ability to make decisions that are in their best interest and that of their teams.

Motivation

The ability to motivate is one of the most important leadership skills. By earning the trust and respect of the team, the endodontic leader is able to bring together the individuals to achieve shared goals and move toward the practice vision. Many people are not self-motivated. Instead, some external force motivates them. In this case, the external force is the practice leader.

Pursuit of knowledge

Practice leaders are usually in constant pursuit of knowledge. They want to excel in the management of the endodontic practice and will read, listen, and learn as much as possible about the subject of practice management. Leaders are typically lifelong learners who do not seek knowledge sporadically but take advantage of opportunities to learn new ideas and strategies on an ongoing basis.

Teamwork

An endodontic leader never works on his or her own. These leaders rely on their team to help achieve practice goals. However, many people mistake the concept of being a leader with being a “ruler.” This approach is not an effective one when working closely with a team whose performance is tied to the ultimate success of the practice. Therefore endodontic leaders must remember that they are part of a group dynamic. In fact, the practice leader is like the coach of a basketball or football team. He or she creates a game plan, guides the team, and facilitates teamwork.

Time management

Time is one of the leader’s most important assets. Endodontic leaders must not only manage their own schedule but their team’s as well. However, good leaders do not micromanage their team. They educate staff members, provide guidance, set expectations, and hold the team accountable for the results.

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Successful leaders effectively and efficiently use their time, creating positive outcomes for themselves, their teams, and their practices. Good leaders also eliminate extraneous items from their lives that take time away from achieving their goals.

Leading by example

The best way to earn the team's respect is to "talk the talk and walk the walk." The way the endodontist behaves will have a significant effect on how the team acts. The professionalism exhibited toward patients, the doctor's attitude and interactions with individual staff members—all of these behaviors send messages to the team. The team is looking to the endodontist for guidance, and the best strategy is to serve as an effective role model. If the team is expected to show up on time, the endodontist cannot stroll in 15 minutes late every day without causing resentment. Good leaders adhere to the rules others are expected to follow.

Leadership is a way of thinking and acting based on desire, skills, training, and specific behaviors. Endodontists who are willing to develop leadership skills will inevitably build better teams with lower turnover and higher efficiency.

Summary

Effective practice management involves management and referral marketing systems that enable an endodontic practice to provide outstanding patient care while operating as a successful business enterprise (Fig. 28.3). The number one source of practice stress is the lack of efficient systems. When an endo practice operates year after year without strong systems, the endodontist experiences increased stress and frustration. On the other hand, endodontists who implement documented, high-performance systems enjoy greater satisfaction (and profitability) throughout their careers.

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FIG. 28.3 Key principles of endodontic practice management. *KPIs,* Key production indicators.

The information in triangular structure is marked top to bottom as follows:

• Leadership.

• Management and referral marketing.

• Scheduling, customer service, and staff training.

• Vision, mission, goals, and KPIs.

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Index

Page numbers followed by *f*, *t*, or *b* indicate figures, tables, or boxes, respectively; *e* indicates online-only content.

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